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To all of our colleagues, fellows, and sonographers with whom we have had the honor to be associated for the past 30 years. Know how much your support, dedication, and expertise has meant, not only to us, but to the entire field of echocardiography. Without these relationships, this book would not have been possible.
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Preface

“Hopefully, the book is short enough and the text is sufficiently readable that it should appeal to physicians who do not want to become personally involved in doing the examination, but who merely want a greater knowledge of the technique’s capabilities and limitations. Such knowledge is necessary if one is to use this diagnostic tool in the most efficient and advantageous manner to help manage cardiac patients.”

Harvey Feigenbaum 1972
From the Preface to the First Edition of Echocardiography

The First Edition of Echocardiography was published in 1972. It was 239 pages long and dealt exclusively with the M-mode technique. With each subsequent edition, Echocardiography has evolved to include new technologies and applications. New developments have broadened the role of echocardiography to evaluate patients in increasingly detailed and sophisticated manners. To keep pace with these changes, the text has grown to over 800 pages with over 2,000 illustrations and access to an eBook with associated video clips where this icon ![video](http://example.com) appears in the print book.

As with previous editions, the Eighth Edition of Feigenbaum’s Echocardiography is focused on proven uses of echocardiography. It is intended as a resource for those engaged in, or learning the practice of clinical echocardiography. As was the case with the First Edition, we hope that this latest edition will serve as a valuable resource to the practicing clinician by demonstrating the pivotal role of echocardiography in patient management. We have tried to emphasize how newer methods supplement and improve upon older approaches and how other imaging modalities can be complimentary and additive to echocardiography for clinical decision
making. To the degree possible, we have avoided platform-specific techniques or methodologies that are of research interest only and instead focused on clinically relevant applications that are widely available.

Utilization rates of imaging have come under intense scrutiny and it is incumbent on the clinician and echocardiographer to be responsible stewards of medical resources. To this end, we have provided evidence-based guidance on usage, including when and how often an echocardiogram should be performed. Whenever applicable we have included a table for the relevant Appropriate Use Criteria for the utilization of echocardiography. Recognizing the nearly universal and instantaneous access to the medical literature, including clinical guidelines, position papers, and other expert opinion documents, we have minimized references to the medical literature and limited the reproduction of tables of normative data that are readily available to the reader. We have concentrated our efforts on providing information that will be durable and relevant for the lifespan of this edition.

As with the first and all subsequent editions we have tried to approach the echocardiographic diagnosis from the perspective of the clinician, rather than solely that of the imager. We believe that it is most helpful to cover the art and science of echocardiography by presenting the information in clinical context. This is because we are also clinicians and consultants and are committed to the concept that echocardiography is an essential tool that should be integrated into the diagnostic evaluation of virtually all forms of cardiovascular disease. We hope the Eighth Edition serves to help train a new generation of echocardiographers, fellows, and sonographers and serves as a valuable reference for those who share our passion for the field.

William F. Armstrong
Thomas Ryan
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Chapter 1
History of Echocardiography
Harvey Feigenbaum

Many histories of diagnostic ultrasound, and cardiac ultrasound in particular, have been written. They all seem to address this field from a different perspective. One can begin the history in the 20th century, Roman times, or any of the centuries in between. It is stated that a Roman architect, Vitruvius, first coined the word echo. A Franciscan friar, Marin Mersenne (1588–1648), is frequently called the “father of acoustics” because he first measured the velocity of sound. Another early physicist, Robert Boyle (1627–1691), recognized that a medium was necessary for the propagation of sound. Abbe Lazzaro Spallanzani (1727–1799) is frequently referred to as the “father of ultrasound.” He demonstrated that bats were blind and in fact navigated by means of echo reflection using inaudible sound. In 1842, Christian Johann Doppler (1803–1853) noted that the pitch of a sound wave varied if the source of the sound was moving. He worked out the mathematical relationship between the pitch and the relative motion of the source and the observer. The ability to create ultrasonic waves came in 1880 with the discovery of piezoelectricity by Curie and Curie. They noted that if certain crystalline materials are compressed, an electric charge is produced between the opposite surfaces. They then noted that the reverse was also true. If an electrical potential is applied to a crystal, it is compressed and decompressed depending on the polarity of the electric charge, and thus very high-frequency sound can be produced. In 1912, a British engineer, L. F. Richardson, suggested that an echo technique could be used to detect underwater objects. Later during World War I, Paul Langevin was given the duty of detecting enemy submarines using sound, which culminated in the
development of sonar. Sokolov described a method for using reflected sound to detect metal flaws in 1929. In 1942, Floyd Firestone, an American engineer, began to apply this technique and received a patent. It is this flaw detection technique that ultimately was used in medicine.

An Austrian, Karl Dussik, was probably the first to apply ultrasound for medical diagnosis in 1941. He initially attempted to outline the ventricles of the brain. His approach used transmission ultrasound rather than reflected ultrasound. After World War II, many of the technologies developed during that war, including sonar, were applied for peaceful and medical uses. In 1950, W. D. Keidel, a German investigator, used ultrasound to examine the heart. His technique was to transmit ultrasonic waves through the heart and record the effect of ultrasound on the other side of the chest. The purpose of his work was to try to determine cardiac volumes. The first effort to use pulse-reflected ultrasound, as described by Firestone, to examine the heart was initiated by Dr. Helmut Hertz of Sweden. He was familiar with Firestone’s observations and in 1953 obtained a commercial ultrasonoscope, which was being used for nondestructive testing. He then collaborated with Dr. Inge Edler, who was a practicing cardiologist in Lund, Sweden. The two of them began to use this commercial ultrasonoscope to examine the heart. This collaboration is commonly accepted as the beginning of clinical echocardiography as we know it today.

The original instrument (Fig. 1.1) was quite insensitive. The only cardiac structures that they could record initially were from the back wall of the heart. In retrospect, these echoes probably came from the posterior left ventricular wall. With some modification of their instrument, they were able to record an echo from the anterior leaflet of the mitral valve. However, they did not recognize the source of this echo for several years and originally attributed the signal to the anterior left atrial wall. Only after some autopsy investigations did they recognize the echo’s true origin. Edler went on to perform a number of ultrasonic studies of the heart. Many of the cardiac echoes currently used were first described by him. However, the principal clinical application of echocardiography developed by Edler was the detection of mitral stenosis. He noted that there was a difference between the pattern of motion of the anterior mitral leaflet in patients who did or did not have mitral stenosis. Thus, the early studies published in the mid-1950s and early 1960s primarily dealt with the detection of this disorder.
The work being done in Sweden was duplicated by a group in Germany headed by Dr. Sven Effert.\textsuperscript{19,20} Their publications began to appear in the late 1950s and were primarily duplications of Edler’s work describing mitral stenosis. One notable observation made by Effert and Domanig\textsuperscript{20} was the detection of left atrial masses. Schmidt and Braun\textsuperscript{21} in Germany also began working with ultrasound cardiography and published their work in 1958, again repeating what Edler and Effert had been doing. Edler et al.\textsuperscript{22} developed a scientific film that was shown at the Third European Congress of Cardiology in Rome in 1960. Edler et al.\textsuperscript{23} also wrote a large review of cardiac ultrasound as a supplement to \textit{Acta Medica Scandinavica}, which was published in 1961, and remained the most comprehensive review of this field for more than 10 years. In the movie and the review, Edler and his coinvestigators described the ultrasonic techniques for the detection of mitral stenosis, left atrial tumors, aortic stenosis, and anterior pericardial effusion.

\textbf{FIGURE 1.1.} Ultrasonoscope initially used by Edler and Hertz for recording their early echocardiograms. (From Edler I. Ultrasound cardiography. \textit{Acta Med Scand Suppl} 370 1961;170:39.)
Despite their initial efforts at using ultrasound to examine the heart, neither Edler nor Hertz really anticipated that this technique would flourish. Helmut Hertz was primarily interested in being able to record the ultrasonic signals. In the process, he developed ink-jet technology and spent only a few years in the field of cardiac ultrasound. He devoted most of the rest of his career to ink-jet technology, for which he held many important patents. He also advised Siemens Corporation, which provided its first ultrasonic instrument, that it should not enter the field of cardiac ultrasound because he personally did not feel that there was a great future in this area (Effert, personal communication, 1996). Edler too did not develop any further techniques in cardiac ultrasound. He retired in 1976 and until then was primarily concerned with the application of echocardiography for mitral stenosis and, to a lesser extent, mitral regurgitation. He never became involved with any of the newer techniques for pericardial effusion or ventricular function.

China was another country where cardiac ultrasound was used in the early years. In the early 1960s, investigators both in Shanghai and Wuhan were using ultrasonic devices to examine the heart. They began initially with an A-mode ultrasound device and then developed an M-mode recorder. The investigators duplicated the findings of Edler and Effert with regard to mitral stenosis. Unique contributions of the Chinese investigators included fetal echocardiography and contrast echocardiography, using hydrogen peroxide and then carbon dioxide.
In the United States, echocardiography was introduced by John J. Wild, H. D. Crawford, and John Reid,\textsuperscript{29} who examined the excised heart. They were able to identify a myocardial infarction and published their findings in 1957 in the *American Heart Journal*. Neither Wild nor Reid was a physician. Reid was an engineer who subsequently went to the University of Pennsylvania for his doctorate degree. While there, he wanted to continue his interest in examining the heart ultrasonically. He joined forces with Claude Joyner, who was a practicing cardiologist in Philadelphia. Reid proceeded to build an ultrasonoscope, and Joyner and he began duplicating the work on mitral stenosis that was described by Edler and Effert. This work was published in *Circulation* in 1963 and represents the first American clinical effort, using pulsed reflected ultrasound to examine the heart.\textsuperscript{30}

I became interested in echocardiography in the latter part of 1963. While operating a hemodynamic laboratory and becoming frustrated with the limitations of cardiac catheterization and angiography, I saw an advertisement from a now defunct company that was claiming that it had an instrument that could measure cardiac volumes with ultrasound. This claim ultimately proved to have no basis. However, when I first saw the ultrasound instrument displayed at the *American Heart Association* meeting in Los Angeles in 1963, I placed the transducer on my chest and saw a moving echo, which had to be coming from the posterior wall of my heart. This signal undoubtedly was the same echo that Hertz and Edler had noted approximately 10 years earlier. I had the people from the company explain the principles by which such a signal might be generated. I asked them whether fluid in back of the heart would give a different type of a signal, and they said that fluid would be echo free. When I returned to Indiana, I found that the neurologists had an ultrasonoscope that they used for detecting the midline of the brain. Fortunately for me, the instrument was rarely being used and I was able to borrow it. I proceeded to examine more individuals, and again I was able to record an echo from the back wall of the left ventricle. I looked for a patient with pericardial effusion. As predicted, there were now two echoes separated by an echo-free space. The more posterior echo no longer moved, whereas the more anterior echo moved with cardiac motion. We went to the animal laboratory to confirm these findings and thus began my personal career in cardiac ultrasound. This initial paper on pericardial effusion was published in *JAMA* in 1965.\textsuperscript{31}
Although this phase of the history of echocardiography is commonly considered the origins of the early practice of echocardiography, it should be mentioned that Japanese investigators were working simultaneously using ultrasound to examine the heart. In the mid-1950s, several Japanese investigators such as Satomura, Yoshida, and Nimura at Osaka University were using Doppler technology to examine the heart. They began publishing their work in the mid-1950s. These efforts laid the basis for much of what we do today with Doppler ultrasound.

The field of cardiac ultrasound has evolved with the efforts of numerous individuals over the past 50 years. This development is an outstanding example of collaboration among physicists, engineers, and clinicians. Each of the cardiac ultrasonic techniques has its own individual history. Even the name echocardiography has a story of its own. Edler and Hertz first called this technique ultrasound cardiography with the abbreviation being UCG. Ultrasound cardiography was a somewhat cumbersome name. The most common use of medical diagnostic ultrasound in the late 1950s and early 1960s was an A-mode technique to detect the midline of the brain. This midline echo would shift if there were an intracranial mass. The technique was known as echoencephalography, and the instrument was an echoencephalograph. It was such an instrument that I borrowed from the neurologists. If the ultrasonic examination of the brain is echoencephalography, then an examination of the heart should be echocardiography. Unfortunately, the abbreviation for an echocardiogram would be ECG, which was already preempted by electrocardiography. We could not use the abbreviation “echo” because it did not differentiate from an echoencephalogram. The reason the term echocardiography finally caught on was because echoencephalography disappeared. No other diagnostic ultrasound technique used the term echo except for the examination of the heart. So, the abbreviation “echo” now stands only for echocardiography and is not confused with any other ultrasonic examination.

**DEVELOPMENT OF VARIOUS ECHOCARDIOGRAPHIC TECHNOLOGIES**

The story of echocardiography involves the evolution and development of its
many modalities such as A-mode, M-mode, contrast, two-dimensional, Doppler, transesophageal, and intravascular applications. The Doppler story is truly lengthy and international. The Japanese began working with Doppler ultrasound in the mid-1950s.\textsuperscript{32,33} American workers, such as Robert Rushmer in Seattle, were early investigators using Doppler techniques.\textsuperscript{34} Dr. Rushmer was a recognized expert in cardiac physiology. John Reid later moved to Seattle and joined Rushmer and his group in developing Doppler technology. One of the engineers, Donald Baker, was in that group and developed one of the first pulsed Doppler instruments.\textsuperscript{35} Eugene Strandness was a vascular surgeon in Seattle using Doppler for peripheral arterial disease.\textsuperscript{36} European investigators were also very active in using Doppler technology. Several early French workers, namely, Peronneau\textsuperscript{37} and later Kalmanson,\textsuperscript{38} wrote extensively on the use of Doppler ultrasound to examine the cardiovascular system. A major development in Doppler ultrasound came when Holen\textsuperscript{39} and then Hatle\textsuperscript{40} demonstrated that one could derive hemodynamic information from Doppler ultrasound. They noted that one could use a modified version of the Bernoulli equation to detect gradients across stenotic valves. The report that the pressure gradient of aortic stenosis could be determined with Doppler ultrasound was probably the development that established Doppler echocardiography as a clinically important technique.

The field of contrast echocardiography began with an unexpected observation by Gramiak et al.\textsuperscript{41} at the University of Rochester. They apparently were doing an ultrasonic examination on a patient undergoing an indicator dilution test using indocyanine green dye. Much to their surprise, they noticed a cloud of echoes introduced into the cardiovascular system with the injection of dye. Apparently, Joyner had noticed a similar observation with the injection of saline but did not report the finding. I heard Gramiak present his group’s work at a meeting and promptly used that technique to help establish the echocardiographic identity of the left ventricular cavity.\textsuperscript{42} Workers at the Mayo Clinic headed by Jamil Tajik and Jim Seward went on to use this contrast technique in a very eloquent way to identify right-to-left shunts.\textsuperscript{43} Contrast agents have evolved to the current commercial products, which are manufactured. The tiny echo-producing bubbles are small enough to pass through capillaries so that a peripheral injection can be seen on the left side of the heart.\textsuperscript{44}
Two-dimensional echocardiography has a lengthy and fascinating history. As with almost every aspect of cardiac ultrasound, there is an international flavor to this story. Two-dimensional ultrasonic scanning dates back to early workers such as Douglass Howry when he began using compound scanning for various parts of the body. One of his early compound scanners used a transducer that was mounted on a ring from a B29 gun turret. The Japanese introduced a variety of ultrasonic devices to create two-dimensional recordings of the heart. They used elaborate water baths and scanning techniques (Fig. 1.2). Gramiak et al. at the University of Rochester used reconstructive two-dimensional M-mode techniques to create ultrasonic “cinematography” (Fig. 1.3). Donald King in New York developed a stop-action type of technique for creating a reconstructed two-dimensional image of the heart (Fig. 1.4).
A major breakthrough occurred when an engineer, Nicholas Bom, in Rotterdam, developed a linear scanner (Fig. 1.5). By using multiple crystals, he could create a rectangular image of the heart in real time.
Although this technique ultimately never proved to be useful in examining the heart, partially because of the rib shadows, this technique did show the virtue of real-time imaging. It ultimately proved to be a leading form of two-dimensional imaging in other parts of the body but not the heart. Real-time two-dimensional echocardiography became practical by using a sector scan rather than a linear scan. Initially, the scan devices were mechanical. Griffith and Henry\textsuperscript{50} at the National Institutes of Health developed a mechanical device that rocked the transducer back and forth. The device was handheld; however, the ability to manipulate the transducer was very limited. Reggie Eggleton, who originally worked at the University of Illinois with Robert, Frank, and Elizabeth Frye, moved to Indiana and developed a mechanical two-dimensional scanner (Fig. 1.6). Interestingly enough, his first prototype was actually a modified sunbeam electric toothbrush. This early mechanical scanner was the first commercially successful real-time two-dimensional device.\textsuperscript{51} Eventually, mechanical sector scanners were replaced by phased-array technology, which was initially developed by Fritz Thurstone and Olaf vonRamm\textsuperscript{52} at Duke University.
Color flow Doppler or two-dimensional Doppler ultrasound dates back to the late 1970s. A group headed by Brandestini working at the University of Washington in Seattle showed how one could use an M-mode recording of a multigated Doppler signal (Fig. 1.7).\textsuperscript{53} They encoded the Doppler signal with color to indicate the direction of flow. This principle was later more fully developed by Japanese workers including Kasai et al.\textsuperscript{54} The key to the development of their two-dimensional color display was the autocorrelation detection of the Doppler velocities. They were now able to provide an excellent real-time two-dimensional display of color flow. Omoto, a Japanese cardiovascular surgeon, and coworkers\textsuperscript{55} helped popularize the clinical value of two-dimensional color Doppler imaging.
FIGURE 1.7. Combined M-mode and Doppler recording whereby the Doppler signal is superimposed on the M-mode tracing. The direction and velocity of the Doppler signal are displayed in varying colors. This particular recording shows the right ventricular outflow tract and aorta. (From Brandestini MA, Eyer MK, Stevenson JG. M/Q: M/Q-mode echocardiography. The synthesis of conventional echo with digital multigate Doppler. In: Lancee CT, Erasmus Universiteit Rotterdam, eds. *Echocardiology*. The Hague, Netherlands: Martinus Nijhoff, 1979. Reproduced with permission of M. Nijhoff in the format Book via Copyright Clearance Center.)

The origin of transesophageal echocardiography also dates back to the 1970s. Lee Frazin, a cardiologist in Chicago, placed an M-mode transducer at the tip of a transesophageal probe and demonstrated how one could obtain an M-mode recording of the heart via the esophagus. This technique never became clinically popular. However, both Japanese and European investigators began working with this technology. They all attempted to obtain two-dimensional images with a transesophageal probe. Initially, the devices were mechanical and later became electronic. Hisanaga et al. were among the Japanese engineers, and Jacques Souquet was a European engineer who made a major contribution to transesophageal electronic probes in 1982. Most of the early clinicians who demonstrated the utility of transesophageal echocardiography were Europeans.

The versatility of ultrasound is exemplified by the fact that one can devise ultrasonic imaging techniques, using very large or very small transducers. An
exquisite ultrasonic imaging device used to examine the entire body was
developed by an Australian engineer, George Kossoff. He developed an
instrument called an Octoson. It consisted of eight very large transducers that
rotated around the body. The instrument produced images that were of
excellent resolution and clarity. The other extreme is the ability to put a tiny
transducer on the tip of a catheter that can be inserted in the cardiovascular
system. Reggie Eggleton devised a catheter-based imaging system in the
1960s as did Ciezynski in Europe and Omoto in Japan. In the early 1970s,
Nicholas Bom et al. described a real-time intracardiac scanner using a
circular array of 32 elements at the tip of a catheter. This technology
developed further to the point that catheter-tipped transducers could be placed
on an intracoronary device. Such instruments have been used clinically and
for investigational purposes for many years now. Possibly, the clinician who
used intracoronary ultrasound to its greatest extent is Steven Nissen, who
currently is at the Cleveland Clinic. He has used this technique to
revolutionize our understanding of coronary atherosclerosis.

There has been interest in three-dimensional echocardiography for many
years. Numerous efforts at using compound two-dimensional scans to
produce three-dimensional imaging have been demonstrated. Some of
these compound three-dimensional devices have been used clinically. Among
the early leaders in three-dimensional echocardiography was Olaf vonRamm
and his group.
FIGURE 1.8. Early M-mode echocardiograph using a Polaroid camera to record an echocardiogram.
Handheld echocardiographs date back to 1978. This early device did not have sufficient image quality to be useful. However, now several such instruments are available and increasing in popularity.
Along with developing instruments to create images and physiologic information of the heart, there has been a simultaneous history of developing techniques for recording this information. From the very beginning, Helmut Hertz was primarily interested in recording rather than creating ultrasonic images. In so doing, he developed ink-jet technology, which proved to be extremely important. When I first began using ultrasound in the early 1960s, a Polaroid camera was the principal recording technique for A-mode and M-mode echocardiograms (Figs. 1.8 and 1.9). This approach was extremely limited and had many problems. Some investigators, such as Gramiak, used 35-mm film to record their M-mode echocardiograms. Much of my early efforts were to get commercial companies to provide strip chart recorders for our M-mode echocardiograms. The variety of strip chart recorders that became available has its own history. With the advent of two-dimensional echocardiography, we had to work out a scheme for recording these real-time two-dimensional images. At our own institution, we first used super 8 movie film as our recording medium. We would direct a movie camera at the oscilloscope and generate movies. The use of the movie film was short lived and we soon went to videotape. Initially, we used reel-to-reel tape recorders. Then a variety of recorders with cassettes became available. A popular tape recorder in the early years was produced by Sanyo. Unfortunately, analyzing a study frame by frame was very tedious. One had to turn a small button-like control and could not view images backward. Finally, Panasonic developed a tape recorder that permitted easy forward and backward viewing as well as frame-by-frame analysis.

Because of the dominance of two-dimensional echocardiography in the clinical use of echocardiography, videotape became the standard means of recording echocardiogram for decades. Unfortunately, videotape also has major limitations. Looking at serial studies with videotape is problematic. The accessibility of videotape is inconvenient. One cannot make measurements from videotaped images. Copies of videotaped images are always degraded. Digital recording of echocardiograms began in the early 1980s. Interest in using digital techniques has been accelerating ever since. There are numerous advantages to using a digital recording. Side-by-side comparisons are facilitated. One can make measurements easily, and the
images are more accessible. Initially, the digital images were generated by
grabbing the video signal either from the instrument or by digitizing the
videotape. In recent years, a direct digital output from ultrasonic instruments
has become available. Digital recording standards using DICOM (Digital
Imaging and COmmunication in Medicine) have facilitated the use of digital
imaging and have become a major factor in the general utility of this
approach.

CARDIAC SONOGRAPHERS

Early in my experience with cardiac ultrasound, it became apparent that the
technique would become fairly popular. Performing the echocardiograms
myself became a fairly time-consuming activity. Being a clinical cardiologist
with responsibilities for patient care, including cardiac catheterization, I
clearly felt that I could not continue to be the principal person to obtain
echocardiograms. We also did not have sufficient physicians interested in the
technique to provide a complement of physicians to do the echocardiograms
throughout the day. As a result, I believed that it would be possible to train a
nonphysician to do an echocardiogram. There was considerable skepticism
among the few physicians active in the field of ultrasound at the time as to
whether this approach was feasible. The first nonphysician hired to perform
echocardiograms was Charles Haine.
The American College of Cardiology

INDIANA UNIVERSITY
SCHOOL OF MEDICINE
and
KRANNERT INSTITUTE
OF CARDIOLOGY
announce

ULTRASOUND:
Diagnostic Use
In Cardiovascular Disease

January 11 and 12, 1968

To Be Presented at:
Fesler Hall
INDIANA UNIVERSITY
MEDICAL CENTER
Indianapolis, Indiana
FIGURE 1.10. The program for the first course devoted to diagnostic ultrasound and cardiovascular disease held in Indianapolis in January 1968.

Our second cardiac sonographer was Sonia Chang. Her skills in obtaining an M-mode echocardiogram were so outstanding that with my encouragement she eventually published a book on the M-mode echocardiographic examination. It was a major publication from which many of the early users of M-mode echocardiography learned their technical skills. Most of the visitors who came to Indiana in the early days learned how to do echocardiograms from Sonia. Sonia left Indiana just after the introduction of two-dimensional echocardiography. She went to Emory University in Atlanta to work with Dr. Willis Hurst, who was the chairman of cardiology at the time.

Virtually, every echocardiographic laboratory in the United States has a sonographer who excels in the ability to obtain an echocardiogram. Cardiac sonographers have been a major factor in making echocardiography a cost-effective examination. Using a nonphysician to create echocardiograms is not a worldwide concept. In most countries, echocardiograms are still obtained by physicians. One exception is England, where there is a somewhat different situation. Their cardiac sonographers are probably more highly trained individuals than our sonographers. They come closer to being a physician’s assistant and have a greater formal education in cardiac physiology and anatomy. They also perform interpretations with a higher degree of frequency than do sonographers in the United States.

ECHOCARDIOGRAPHIC EDUCATION AND ORGANIZATIONS

The first meeting dedicated solely to cardiac ultrasound was in Indianapolis in January 1968 (Fig. 1.10). Among the faculty were Drs. Edler, Joyner, Reid, and Strandness (Fig. 1.11). There were approximately 50 people who attended that course, one of whom was Raymond Gramiak. At that meeting, Dr. Edler showed the movie that he had created for the 1960 European Congress of Cardiology Meeting in Rome. Another member of the faculty was Richard Popp, who was a cardiology fellow at Indiana at the time.
Bernard Ostrum, who was a radiologist at Albert Einstein Medical Center, presented data on abdominal aortas. Chuck Haine was an integral part of the program and demonstrated some of our ultrasonic techniques at Indiana.

The American Society of Echocardiography was also created in Indianapolis in 1975. The decision to create the society was made at a postgraduate meeting in Indianapolis. The *Journal of the American Society of Echocardiography* began in 1988 and the first annual American Society of Echocardiography scientific meeting was held in Washington, DC, in 1990. There are now several worldwide echocardiography organizations, publications, and meetings.

![FIGURE 1.11. Photograph of Drs. Edler and Feigenbaum demonstrating an M-mode echocardiograph at the 1968 meeting of cardiac ultrasound in Indianapolis.](image)

Echocardiography has come a long way since its beginnings in the mid-1950s. Although there are many new, highly sophisticated imaging technologies being developed, there is every reason to believe that the clinical utility and popularity of echocardiography will continue to grow. This diagnostic tool is amazingly versatile. It is still very cost effective compared
with competing technologies and has many new possibilities as to how this examination can be improved and provide more and better information. Thus, the future of echocardiography should be as productive and exciting as have been the previous five decades.

References


Cardiac ultrasound, or echocardiography, relies on the generation, propagation, reflection, and reception of generated sound waves. Understanding the basic physics of sound wave behavior is essential for understanding image generation. Sound is a mechanical vibration transmitted through an elastic medium. When it propagates through the air at the appropriate frequency, sound may produce the sensation of hearing. Ultrasound includes that portion of the sound spectrum having a frequency greater than 20,000 cycles/s (20 KHz), which is considerably above the audible range. The use of ultrasound to study the structure and function of the heart and great vessels defines the field of echocardiography. The production of ultrasound for diagnostic purposes involves complex physical principles and sophisticated instrumentation. As technology has evolved, a thorough understanding of these principles mandates an extensive background in physics and engineering. Fortunately, the use of echocardiography for clinical purposes does not require a complete mastery of the physics and instrumentation involved in the creation of the ultrasound image. However, a basic understanding of these facts is necessary to take full advantage of the technique and to appreciate the strengths and limitations of the technology.

This book is intended principally as a clinical guide to the broad field of echocardiography, to be used by clinicians, students, and sonographers concerned more about the practical application of the technology than the underlying physics. For this reason, an extensive description of the physics and engineering of ultrasound is beyond the scope of this book. Instead, this chapter focuses on those aspects of physics and instrumentation that are relevant to the understanding of ultrasound and its practical application to patient care. In addition, many of the newer technical advances in ultrasound
instrumentation are presented briefly, primarily to provide the reader a sense of the changing and ever-improving nature of echocardiography.

**PHYSICAL PRINCIPLES**

Ultrasound (in contrast to lower, i.e., audible frequency sound) has several characteristics that contribute to its diagnostic utility. First, ultrasound can be directed as a beam and focused. Second, as ultrasound passes through a medium, it obeys the laws of reflection and refraction. Finally, targets of relatively small size reflect ultrasound and can therefore be detected and characterized. A major disadvantage of ultrasound is that it is poorly transmitted through a gaseous medium and attenuation occurs rapidly, especially at higher frequencies. As a wave of ultrasound propagates through a medium, the particles of the medium vibrate parallel to the line of propagation, producing *longitudinal waves*. Thus, a sound wave is characterized by areas of more densely packed particles within the medium (an area of compression) alternating with regions of less densely packed particles (an area of rarefaction). The amount of reflection, refraction, and attenuation depends on the acoustic properties of the various media through which an ultrasound beam passes. Tissues composed of solid material interfaced with gas (such as the lung) will reflect most of the ultrasound energy, resulting in poor penetration. Very dense media also reflect a high percentage of the ultrasound energy. Soft tissues and blood allow relatively more ultrasound energy to be propagated, thereby increasing penetration and improving diagnostic utility. Bone also reflects most ultrasound energy, not because it is dense but because it contains so many interfaces.

The ultrasound wave is often graphically depicted as a sine wave in which the peaks and troughs represent the areas of compression and rarefaction, respectively (Fig. 2.1). Small pressure changes occur within the medium, corresponding to these areas, and result in tiny oscillations of particles, although no actual particle motion occurs. Depicting ultrasound in the form of a sine wave has some limitations but allows the demonstration of several fundamental principles. The sum of one compression and one rarefaction represents *one cycle*, and the distance between two similar points along the wave corresponds to *wavelength* (see Table 2.1 for definitions of commonly
used terms). Over the range of diagnostic ultrasound, wavelength varies from approximately 0.15 to 1.5 mm in soft tissue. The frequency of the sound wave is the number of wavelengths per unit of time. Thus, wavelength and frequency are inversely related and their product represents the velocity of the sound wave:

\[ v = f \times \lambda \]  

[Eq. 2.1]

where \( v \) is velocity, \( f \) is frequency (in cycles per second or hertz), and \( \lambda \) is wavelength. Velocity through a given medium depends on the density and elastic properties or stiffness of that medium. Velocity is directly related to stiffness and inversely related to density. Ultrasound travels faster through a stiff medium, such as bone. Velocity also varies with temperature, but because body temperature is maintained within a relatively narrow range, this is of little significance in medical imaging. Table 2.2 provides a comparison of average velocity values in various types of tissues. Within soft tissue, velocity of sound is fairly constant at approximately 1,540 m/s (or 1.54 m/ms, or 1.54 mm/μs). Thus, to find the wavelength of a 3-MHz transducer, the solution would be given by:

\[ \lambda = v \div f \]

\[ \lambda = 1,540 \text{ m/s} \div 3,000,000 \text{ cycles/s} \]
FIGURE 2.1. This schematic illustrates how sound can be depicted as a sine wave whose peaks and troughs correspond to areas of compression and rarefaction, respectively. As sound energy propagates through tissue, the wave has a fixed wavelength that is determined by the frequency and amplitude that is a measure of the magnitude of pressure changes. See text for further details.

### Table 2.1
DEFINITIONS OF BASIC TERMS

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absorption</td>
<td>The transfer of ultrasound energy to the tissue during propagation</td>
</tr>
<tr>
<td>Acoustic</td>
<td>The product of the density of the medium and the velocity of sound;</td>
</tr>
<tr>
<td>impedance</td>
<td>differences in acoustic impedance between two media determine the ratio</td>
</tr>
<tr>
<td></td>
<td>of transmitted versus reflected sound at the interface</td>
</tr>
<tr>
<td>Amplitude</td>
<td>The magnitude of the pressure changes along the wave; also, the strength</td>
</tr>
<tr>
<td></td>
<td>of the wave (in decibels)</td>
</tr>
<tr>
<td>Attenuation</td>
<td>The net loss of ultrasound energy as a wave propagates through a medium</td>
</tr>
<tr>
<td>Cycle</td>
<td>The combination or sum of one compression and one rarefaction of a</td>
</tr>
<tr>
<td></td>
<td>propagating wave</td>
</tr>
<tr>
<td>Dead time</td>
<td>The time in between pulses that the echograph is not emitting ultrasound</td>
</tr>
<tr>
<td>Decibel</td>
<td>A logarithmic measure of the intensity of sound, expressed as a ratio to</td>
</tr>
<tr>
<td></td>
<td>a reference value (dB)</td>
</tr>
<tr>
<td>Duty factor</td>
<td>The fraction of time that the transducer is emitting ultrasound, a unitless</td>
</tr>
<tr>
<td></td>
<td>number between 0 and 1</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
</tr>
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<td>-------------------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Far field</td>
<td>The diverging conical portion of the beam beyond the near field</td>
</tr>
<tr>
<td>Frequency</td>
<td>The number of cycles per second, measured in Hertz (Hz)</td>
</tr>
<tr>
<td>Gain</td>
<td>The degree, or percentage, of amplification of the returning ultrasound signal</td>
</tr>
<tr>
<td>Half-layer value</td>
<td>The distance an ultrasound beam penetrates into a medium before its intensity has attenuated to one-half the original value</td>
</tr>
<tr>
<td>Intensity</td>
<td>The concentration or distribution of power within an area, often the cross-sectional area of the ultrasound beam, analogous to loudness</td>
</tr>
<tr>
<td>Longitudinal wave</td>
<td>A cyclic disturbance in which the energy propagation is parallel to the direction of particle motion</td>
</tr>
<tr>
<td>Near field</td>
<td>The proximal cylindrical-shaped portion of the ultrasound beam before divergence begins to occur</td>
</tr>
<tr>
<td>Period</td>
<td>The time required to complete one cycle, usually expressed in microseconds (μsec)</td>
</tr>
<tr>
<td>Piezoelectricity</td>
<td>The phenomenon of changing shape in response to an applied electric current, resulting in vibration and the production of sound waves; the ability to produce an electric impulse in response to a mechanical deformation; thus, the interconversion of electrical and sound energy</td>
</tr>
<tr>
<td>Power</td>
<td>The rate of transfer over time of the acoustic energy from the propagating wave to the medium, measured in Watts</td>
</tr>
<tr>
<td>Pulse</td>
<td>A burst or packet of emitted ultrasound of finite duration, containing a fixed number of cycles traveling together</td>
</tr>
<tr>
<td>Pulse length</td>
<td>The physical length or distance that a pulse occupies in space, usually expressed in millimeters (mm)</td>
</tr>
<tr>
<td>Pulse repetition frequency</td>
<td>The rate at which pulses are emitted from the transducer, i.e., the number of pulses emitted within a period of time, usually 1 sec</td>
</tr>
<tr>
<td>Resolution</td>
<td>The smallest distance between two points that allows the points to be distinguished as separate</td>
</tr>
<tr>
<td>Sensitivity</td>
<td>The ability of the system to image small targets at a given depth</td>
</tr>
<tr>
<td>Ultrasound</td>
<td>A mechanical vibration in a physical medium, characterized by a frequency &gt;20,000 Hz</td>
</tr>
<tr>
<td>Velocity</td>
<td>The speed at which sound moves through a given medium</td>
</tr>
<tr>
<td>Wavelength</td>
<td>The length of a single cycle of the ultrasound wave; a measure of distance, not time</td>
</tr>
</tbody>
</table>

### Table 2.2

**VELOCITY OF SOUND IN AIR AND VARIOUS TYPES OF TISSUES**

<table>
<thead>
<tr>
<th>Medium</th>
<th>Velocity (m/s)</th>
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</tbody>
</table>
A simpler version of this equation is given by $\lambda$ (in millimeters) = $\frac{1.54}{f}$, where $f$ is the transducer frequency (in megahertz). This converts 1,540 m/s to 1.54 mm/μs, expresses frequency in megahertz, and yields wavelength in millimeters. Thus,

$$\lambda = \frac{v}{f}$$

$$\lambda = \frac{1.54}{3.0} \approx 0.51 \text{ mm}$$

If an ultrasound wave encounters an area of higher elasticity or stiffness, for example, velocity will increase. Because frequency does not change, wavelength will also increase. As is discussed later, wavelength is a determinant of resolution: the shorter the wavelength is, the smaller the target that is able to reflect the ultrasound wave and thus the greater the resolution.

Another fundamental property of sound is *amplitude*, which is a measure of the strength of the sound wave (Fig. 2.1). It is defined as the difference between the peak pressure within the medium and the average value, depicted as the height of the sine wave above and below the baseline. Amplitude is measured in *decibels*, a logarithmic unit that relates acoustic pressure to some reference value. The primary advantage of using a logarithmic scale to display amplitude is that a very wide range of values can be accommodated and weak signals can be displayed alongside much stronger signals. Of practical use, an increase of 6 dB is equal to a doubling of signal amplitude, and 60 dB represents a 1,000-fold change in amplitude or loudness. A parameter closely related to amplitude is power, which is defined as the rate of energy transfer to the medium, measured in watts. For clinical purposes, power is usually represented over a given area (often the beam area) and referred to as *intensity* (watts per centimeter squared or W/cm$^2$). This is
analogous to loudness. Intensity diminishes rapidly with propagation distance and has important implications with respect to the biologic effects of ultrasound, which are discussed later.

INTERACTION BETWEEN ULTRASOUND AND TISSUE

These basic characteristics of ultrasound have practical implications for the interaction between ultrasound and tissue. For example, the higher the frequency of the ultrasound wave (and the shorter the wavelength), the smaller the structures that can be accurately resolved. Because precise identification of small structures is a goal of imaging, the use of high frequencies would seem desirable. However, higher-frequency ultrasound has less penetration compared with lower-frequency ultrasound. The loss of ultrasound as it propagates through a medium is referred to as attenuation. This is a measure of the rate at which the intensity of the ultrasound beam diminishes as it penetrates the tissue. Attenuation has three components: absorption, scattering, and reflection. Attenuation always increases with depth and is also affected by the frequency of the transmitted beam and the type of tissue through which the ultrasound passes. The higher the frequency is, the more rapidly it will attenuate. Attenuation may be expressed as the “half-value layer” or the “half-power distance,” which is a measure of the distance that ultrasound travels before its amplitude is attenuated to one-half its original value. Representative half-power distances are listed in Table 2.3. As a rule of thumb, the attenuation of ultrasound in tissue is between 0.5 and 1.0 dB/cm/MHz. This approximation describes the expected loss of energy (in decibels) that would occur over the round-trip distance that a beam would travel after being emitted by a given transducer. For example, if a 3-MHz transducer is used to image an object at a depth of 12 cm (24-cm round trip), the returning signal could be attenuated as much as 72 dB (or nearly 4,000-fold). As expected, attenuation is greater in soft tissue compared with blood and is even greater in muscle, lung, and bone.

Table 2.3 REPRESENTATIVE HALF-POWER DISTANCES RELEVANT TO
### ECHOCARDIOGRAPHY

<table>
<thead>
<tr>
<th>Material</th>
<th>Half-power Distance (cm)</th>
<th>Attenuation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>380</td>
<td>Less</td>
</tr>
<tr>
<td>Blood</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Soft tissue (except muscle)</td>
<td>1–5</td>
<td></td>
</tr>
<tr>
<td>Muscle</td>
<td>0.6–1</td>
<td></td>
</tr>
<tr>
<td>Bone</td>
<td>0.2–0.7</td>
<td></td>
</tr>
<tr>
<td>Air</td>
<td>0.08</td>
<td></td>
</tr>
<tr>
<td>Lung</td>
<td>0.05</td>
<td>More</td>
</tr>
</tbody>
</table>

The velocity and direction of the ultrasound beam as it passes through a medium are a function of the acoustic impedance of that medium. Acoustic impedance (Z, measured in rayls) is simply the product of velocity (in meters per second) and physical density (in kilograms per cubic meter). Within a homogeneous structure, the density and stiffness of the medium primarily determine the behavior of a transmitted ultrasound beam. In such a structure, sound would travel in a straight line at a constant velocity, depending on the density and stiffness. Variations in impedance create an acoustic mismatch between regions. The greater the acoustic mismatch, the more the energy is reflected rather than transmitted. Within the body, the tissues through which an ultrasound beam passes have different acoustic impedances. When the beam crosses a boundary between two tissues, a portion of the energy is reflected, a portion is refracted, and a portion continues in a relatively straight line (Fig. 2.2A).

These interactions between the ultrasound beam with acoustic interfaces form the basis for ultrasound imaging. The phenomena of reflection and refraction obey the laws of optics and depend on the angle of incidence between the transmitted beam and the acoustic interface as well as the acoustic mismatch, that is, the magnitude of the difference in acoustic impedance. Small differences in velocity also determine refraction. These properties explain the importance of using an acoustic coupling gel during transthoracic imaging. Without the gel, the air–tissue interface at the skin surface results in more than 99% of the ultrasonic energy being reflected at this level. This is primarily due to the very low acoustic impedance of air.
The use of gel between the transducer and the skin surface greatly increases the percentage of energy that is transmitted into and out of the body, thereby allowing imaging to occur.

**FIGURE 2.2.** A: A transmitted wave interacts with an acoustic interface in a predictable way. Some of the ultrasound energy is reflected at the interface and some is transmitted through the interface. The transmitted portion of the energy is refracted, or bent, depending on the angle of incidence and differences in impedance between the tissues. B: The interaction between an ultrasound wave and its target depends on several factors. A specular reflection occurs when ultrasound encounters a target that is large relative to the transmitted wavelength. The amount of ultrasound energy that is reflected to the transducer by a specular target depends on the angle and the impedance of the tissue. Targets that are small relative to the transmitted wavelength produce a scattering of ultrasound energy, resulting in a small portion of energy being returned to the transducer. This type of interaction results in “speckle” that produces the texture within tissues.

**FIGURE 2.3.** This schematic demonstrates how speckle tracking is performed. In
In this simplified example, a single region of interest in the posterior LV wall is tracked based on its unique speckle signature. In the drawing, a small region in the mid-myocardium moves over time from point 1 to point 2.

As the ultrasound beam is transmitted through tissue, it encounters a complex array of large and small interfaces and targets, each of which affect the transmission of the ultrasound energy. These interactions can be broadly categorized as specular echoes and scattered echoes (Fig. 2.2B). Specular echoes are produced by reflectors that are large relative to ultrasound wavelength, such as the endocardial surface of the left ventricle. Such targets reflect a relatively greater proportion of the ultrasound energy in an angle-dependent fashion. The spatial orientation and the shape of the reflector determine the angles of specular echoes. Examples of specular reflectors include endocardial and epicardial surfaces, valves, and pericardium.

Targets that are small relative to the wavelength of the transmitted ultrasound produce scattering, and such objects are sometimes referred to as Rayleigh scatterers. The resultant echoes are diffracted or bent and scattered in all directions. Because the percentage of energy returning to the transducer from scattered echoes is considerably less than that resulting from specular interactions, the amplitude of the signals produced by scattered echoes is very low (Fig. 2.2B). Despite this fact, scattering has important clinical significance (for both echocardiography and Doppler imaging). Scattered echoes contribute to the visualization of surfaces that are parallel to the ultrasonic beam and also provide the substrate for visualizing the texture of gray-scale images. The term “speckle” is used to describe the tissue-ultrasound interactions that result from a large number of small reflectors within a resolution cell. Without the ability to record scattered echoes, the left ventricular wall, for example, would appear as two bright linear structures, the endocardial and the epicardial surfaces, with nothing in between.

Because the distribution of speckle within a small region of interest is random but fairly constant, if such regions could be uniquely identified, they could be tracked over time and space. By exploiting this phenomenon, a region within the myocardium can be followed throughout the cardiac cycle, a technique referred to as speckle tracking. This method can be used to track the motion of a single region of interest, such as the mitral annulus, or to simultaneously track multiple regions of the left ventricular myocardium, to
generate strain maps (Fig. 2.3). And because this is not a Doppler technique, it is not angle dependent.

From the above discussion, it is evident that the interaction between an ultrasound beam and a reflector depends on the relative size of the targets and the wavelength of the beam. If a solid object is submerged in water, for example, whether reflection of ultrasound occurs depends on the size of the object with respect to the wavelength of the transmitted ultrasound. Specifically, the thickness or profile of the object relative to the ultrasound beam must be at least one-fourth the wavelength of the ultrasound. Thus, as the size of the target decreases, the wavelength of the ultrasound must decrease proportionately to produce a reflection and permit the object to be recorded. This explains why higher-frequency ultrasound allows smaller objects to be visualized. In clinical practice, echocardiography typically employs ultrasound with a range of 2,000,000 to 8,000,000 cycles/s (2 to 8 MHz). At a frequency of 2 MHz, it is generally possible to record distinct echoes from interfaces separated by approximately 1 mm. However, because high-frequency ultrasound is reflected by many small interfaces within tissue, resulting in scattering, much of the ultrasonic energy becomes attenuated and less energy is available to penetrate deeper into the body. Thus, penetration is reduced as frequency increases. Similarly, as the medium becomes less homogeneous, the degree of reflection and refraction increases, resulting in less penetration of the ultrasound energy.

THE TRANSDUCER

The use of ultrasound for imaging became practical with the development of piezoelectric transducers. The principles of piezoelectricity are illustrated in Figure 2.4. Piezoelectric substances or crystals rapidly change shape or vibrate when an alternating electric current is applied. It is the rapidly alternating expansion and contraction of the crystal material that produces the sound waves. Equally important is the fact that a piezoelectric crystal will produce an electric impulse when it is deformed by reflected sound energy. The creation of an ultrasound pulse thus requires that an alternating electric current be applied to a piezoelectric element. This results in the emission of sound energy from the transducer, followed by a period of quiescence during
which the transducer “listens” for some of the transmitted ultrasound energy to be reflected back (known as “dead time”). The amount of acoustic energy that returns to the transducer is a measure of the strength and depth of the reflector. The time required for the ultrasound pulse to make the round-trip from transducer to target and back again allows calculation of the distance between the transducer and reflector.

FIGURE 2.4. The principles of piezoelectricity are illustrated in this schematic. A piezoelectric crystal will vibrate when an electric current is applied, resulting in the generation and transmission of ultrasound energy. Conversely, when reflected energy encounters a piezoelectric crystal, the crystal will change shape in response to this interaction and produce an electrical impulse. See text for further details.

An ultrasound transducer consists of many small, carefully arranged piezoelectric elements that are interconnected electronically. The frequency of the transducer is determined by the thickness of these elements. Each element is coupled to electrodes, which transmit current to the crystals, and then record the voltage generated by the returning signals. An important component of transducer design is the dampening (or backing) material,
which shortens the ringing response of the piezoelectric material after the brief excitation pulse. An excessive ringing response (or “ringdown”) lengthens the ultrasonic pulse and decreases range resolution. Thus, the dampening material both shortens the ringdown and provides absorption of backward and laterally transmitted acoustic energy. At the surface of the transducer, matching layers are applied to provide acoustic impedance matching between the piezoelectric elements and the body. This increases the efficiency of transmitted energy by minimizing the reflection of the ultrasonic wave as it exits the transducer surface.

Transducer design is critically important to optimal image creation. An important feature of ultrasound is the ability to direct or focus the beam as it leaves the transducer. This results in a parallel and cylindrically shaped beam. Eventually, however, the beam diverges and becomes cone shaped (Fig. 2.5). The proximal or cylindrical portion of the beam is referred to as the near field or Fresnel zone. When it begins to diverge, it is called the far field or Fraunhofer zone. For a variety of reasons, imaging is optimal within the near field. Thus, maximizing the length of the near field is an important goal of echocardiography.

![Diagram of ultrasound beam divergence](image)

**FIGURE 2.5.** When ultrasound is emitted from a transducer, the shape of the beam behaves in a predictable manner. If the transducer face is round, the transmitted beam will remain cylindrical for a distance, defined as the near field. After propagating for a certain distance, the beam will begin to diverge and become cone shaped. This region of the beam is referred to as the far field. Within this portion of the beam, a decrease in intensity occurs. The length of the near field is determined by the radius of the transducer face and the wavelength or frequency of the transmitted energy. See text for details.

The length of the near field \((l)\) is described by the formula:

\[
l = \frac{r^2}{\lambda}
\]
\[ l = \frac{r^2}{\lambda} \quad \text{[Eq. 2.2]} \]

where \( r \) is the radius of the transducer and \( \lambda \) is the wavelength of the emitted ultrasound. Either decreasing the wavelength (increasing the frequency) or increasing the size of the transducer will lengthen the near field. These relationships are illustrated in Figure 2.6. From the above formula, one might conclude that optimal ultrasound imaging would always employ a large-diameter, high-frequency transducer to maximize the length of the near field. Several factors prevent this approach from being practical. First, the transducer size is predominantly limited by the size of the intercostal spaces. A transducer that is too large will not be able to image between the ribs. Second, although higher frequency does lengthen the near field, it also results in greater attenuation and lower penetration of the ultrasound energy, thereby limiting its usefulness. These tradeoffs must be balanced to maximize imaging performance. Even when the near-field length is maximized, most targets will still lie in the far field. To improve imaging in this area, the rate of beam divergence must be minimized. To decrease the amount of divergence in the far field, a large-diameter, high-frequency transducer is optimal. As discussed previously, focusing of the transmitted beam tends to improve imaging in the near field but will increase the rate or angle of divergence in the far field (Fig. 2.7). Focusing is accomplished through the use of an acoustic lens placed on the surface of the transducer or by constructing the piezoelectric crystal in a concave shape. Thus, transducer frequency, size, and focusing all interact to affect image quality in the near and far fields. Tradeoffs exist that must be taken into account to create optimal images. Figure 2.8 is an example of the effects of varying transducer frequencies on image quality and appearance. On the left, a short-axis view is recorded using a 3-MHz transducer. On the right, a similar image is captured using a 5-MHz probe. Note how the higher frequency results in improved resolution and detail, especially within the myocardium.
FIGURE 2.6. The length of the near field depends on transducer frequency and transducer size, as illustrated in these four examples. On the left, a transducer with a 10-mm diameter emits ultrasound at 2 MHz. This determines both the length of the near field and the rate of divergence in the far field. If the same size transducer emits energy at 4 MHz, the length of the near field increases and the rate of dispersion is less. A transducer half that size (5 mm) transmitting at 4 MHz will have a shorter near field. Finally, a 5-mm transducer that transmits at 2 MHz will have the shortest near field and the greatest rate of dispersion in the far field.
FIGURE 2.7. The ultrasound beam emitted by a transducer can be either unfocused (top) or can be focused by use of an acoustic lens (bottom). Focusing results in a narrower beam but does not change the length of the near field. An undesirable effect of focusing is that the rate of dispersion in the far field is greater.

FIGURE 2.8. The effects of different transducer frequencies on image quality and
MANIPULATING THE ULTRASOUND BEAM

For most clinical applications, the ultrasound beam is both focused and steered electronically. Although beam manipulation can be done mechanically, with modern equipment, it is achieved through the use of phased-array transducers, which consist of a series of small piezoelectric elements interconnected electronically (Fig. 2.9). In such transducers, the wave front of the beam consists of the sum of the individual wavelets produced by each element. By manipulating the timing of excitation of individual elements, both focusing and steering are possible. If all elements are excited simultaneously, each one will produce a circular wavelet that combines to generate a longitudinal wave front that is parallel to the face of the transducer and propagates in a direction perpendicular to that face. By adjusting the timing of excitation, as shown in Figure 2.10A, the beam can be steered. Further adjustments in the timing allow the beam to be steered through a sector arc, resulting in a two-dimensional image. Using a similar approach, electronic transmit focusing of the beam is also possible (Fig. 2.10B). For example, by exciting the outside elements first and then...
progressively activating the more central elements, the individual wavelets form a curved front that allow focusing at a particular distance within the near field. This can either be fixed or adjustable, and the process is referred to as **dynamic transmit focusing**.

**FIGURE 2.10.** A: Phased-array technology permits steering of the ultrasound beam. By adjusting the timing of excitation of the individual piezoelectric crystals, the wave front of ultrasound energy can be directed, as shown. Beam steering is a fundamental feature of how two-dimensional images are created. B: By adjusting the timing of excitation of the individual crystals within a phased-array transducer, the beam can be focused. In this example, the outer elements are fired first, followed sequentially by the more central elements. Because the speed of sound is fixed, this manipulation in the timing of excitation results in a wave front that is curved and focused. This is called transmit focusing.

It should be recognized that the ultrasound beam is a three-dimensional structure that, in the case of a phased-array transducer, is roughly rectangular in cross section (**Fig. 2.11**). The dimensions of the beam are referred to as axial (along the axis of wave propagation) and lateral (parallel to the face of the transducer, sometimes called azimuthal). The lateral dimension is further divided into a vertical and horizontal component. Acoustic focusing through a lens will change the shape in the vertical and horizontal dimensions equally. Electronic focusing will narrow the beam in one of these two dimensions, resulting in a “thinner” sector slice. Transducers that employ annular phased-array technology have the capacity to focus in both dimensions, resulting in a compact, high-intensity beam profile.

Another type of transducer uses a **linear array** of elements. Such transducers have a rectangular face with crystals aligned parallel to one another along the length of the transducer face. Unlike phased-array transducers, the elements are excited simultaneously so the individual scan lines are directed perpendicular to the face and remain parallel to each other. This results in a rectangle-shaped beam that is unfocused. Linear-array technology is often used for abdominal, vascular, or obstetric applications. Alternatively, the face of a linear transducer can be curved to create a sector
scan. This innovative design is now being used in some handheld ultrasound devices.

FIGURE 2.11. The ultrasound beam can be represented as a three-dimensional structure. A single-crystal transducer (top) will emit a cylindrically shaped beam. If the transducer face is rectangular shaped (bottom), the beam will also have a rectangular shape. The various beam axes are labeled in the two drawings.

To perform real-time three-dimensional echocardiography, a more complex transducer design is needed. This requires the arrangement of the piezoelectric elements into a two-dimensional matrix. Each element represents a scan line that is used to construct the three-dimensional dataset. For example, if the matrix consists of 64 by 64 elements, 4,096 scan lines can be generated. Through careful manipulation of the timing of excitation, a
pyramidal-shaped volume (rather than a tomographic slice) of ultrasound data can be collected. By interrogating the volumetric shape several (>20) times per second, real-time imaging in three dimensions is possible (Fig. 2.12). This topic is covered in greater detail in Chapter 4.

Focusing has the effect of concentrating the acoustic energy into a smaller area, resulting in increased intensity at the point of focus. Intensity also varies across the lateral dimensions of the beam, being greatest at the center and decreasing in intensity toward the edges. When the shape of the ultrasonic beam is diagrammed, it is conventional to draw the edge of the beam to the half-value limit of the beam plot. An example of a transaxial beam plot is illustrated in Figure 2.13. This diagram illustrates the important relationship between intensity and beam width. At its peak intensity, the beam may be as narrow as 1 mm. At its weakest intensity, however, beam width may be as great as 12 mm. For purposes of comparison, it is customary to measure the beam width at its half amplitude or intensity. In the example shown, the beam width would be reported as 6.2 mm. Finally, it should be remembered that gain setting will affect these values in a predictable manner. At high-gain settings, the weaker portion of the ultrasound beam is recorded and beam width is greater. Conversely, at low-gain settings, the beam width would be narrower.

As is apparent from the previous discussion, focusing of the ultrasonic beam is generally desirable. By increasing beam intensity within the near field, the strength of returning signals is enhanced. An undesirable effect of focusing is its effect on beam divergence in the far field. Because focusing results in a beam with a smaller radius, the angle of divergence in the far field is increased. However, because beam divergence begins from a small cross-sectional area of a focused beam, the net effect is variable. The result of these relationships is a tradeoff between resolution at the point of focus and depth of field. Divergence also contributes to the formation of important imaging artifacts such as side lobes (discussed later).

**Resolution**

Resolution is the ability to distinguish between two objects in close proximity. Because echocardiography involves imaging small structures to
provide detailed anatomic information, resolution is one of its most important variables. Furthermore, because echocardiography is a dynamic imaging technique, resolution has at least two components: spatial and temporal. Spatial resolution is defined as the smallest distance that two targets must be separated by for the system to distinguish between them. It, too, has two components: Axial resolution refers to the ability to differentiate two structures lying along the axis of the ultrasound beam (i.e., one behind the other) and lateral resolution refers to the ability to distinguish two reflectors that lie side by side relative to the beam (Fig. 2.14).

![Figure 2.12](image)

**FIGURE 2.12.** The relationship between two-dimensional and three-dimensional imaging is demonstrated in this schematic. In (A), the piezoelectric elements are arranged linearly, allowing the ultrasound beam to sweep through a sector arc to record a two-dimensional tomographic image of the left ventricle (B and C). With volumetric scanning (D), the piezoelectric crystals are arranged in a rectangular matrix, rather than linearly. The ultrasound beam covers a pyramid-shaped region containing most or all cardiac structures (E). By removing a portion of the pyramid, internal structures such as the mitral valve, can be visualized in real time (F).

The primary determinants of axial resolution are the frequency of the transmitted wave and, more importantly, its effect on pulse length. Higher frequency is associated with shorter wavelength, and the size of the wave
relative to the size of the object determines resolution. In addition to frequency, pulse length or duration also affects axial resolution. The shorter the train of cycles is, the greater the likelihood that two closely positioned targets can be resolved. Because a higher-frequency and/or broad bandwidth transducer delivers a shorter pulse, it is also associated with higher resolution.

![Graph showing normalized intensity vs position in mm]

**FIGURE 2.13.** A transaxial beam plot is demonstrated. The beam width or lateral resolution is a function of the intensity of the ultrasonic beam. The beam width is commonly measured at the half-intensity level, and, in this case, the beam width would be reported as 6.2 mm.

Lateral resolution varies throughout the field of imaging and is affected by several factors. The width or thickness of the interrogating beam, at a given depth, is the most important determinant. Ideally, the ultrasonic beam should be very narrow to provide a thin “slice” of the heart. Recall that the beam has finite width, even in the near field, and tends to diverge as it propagates. The importance of beam width stems from the fact that the system will display all targets within the path of the beam along a single line represented by the central axis of the beam. In other words, the echograph displays structures
within the image as if the beam were infinitely narrow. Thus, lateral resolution diminishes as beam width (and depth) increases. The distribution of intensity across the beam profile will also affect lateral resolution. As illustrated in Figure 2.15, both strong and weak reflectors can be resolved within the central portion of the beam, where intensity is greatest. At the edge of the beam, however, only relatively strong reflectors may produce a signal. Furthermore, the true size and position of such objects may be distorted by the width of the beam, resulting in significant beam width artifacts. This is illustrated in Figure 2.15. This observation also explains the importance of overall system gain and its effect on lateral resolution. Gain is the amplitude, or the degree of amplification, of the received signal. When gain is low, weaker echoes from the edge of the beam may not be recorded and the beam appears relatively narrow. If system gain is increased, weaker and more peripheral targets are recorded and beam width appears greater. Thus, to enhance lateral resolution, a minimal amount of system gain should be employed. Figure 2.16 illustrates how changes in gain setting can drastically alter lateral resolution and anatomic information.

FIGURE 2.14. The different types of resolution are demonstrated in this schematic. See text for details.
A third component of resolution is called *contrast resolution*. Contrast resolution refers to the ability to distinguish and to display different shades of gray within the image. This is important both for the accurate identification of borders and for the ability to display texture or detail within the tissues. To convert the returning radio frequency (RF) information into a gray-scale image, pre- and postprocessing of the data are performed. These steps in image formation rely heavily on contrast resolution. From a practical standpoint, contrast resolution is necessary to differentiate tissue signals from background noise. Contrast resolution is also dependent on target size. A
higher degree of contrast is needed to detect small structures compared with larger targets.

*Temporal resolution*, or frame rate, refers to the ability of the system to accurately track moving targets over time. It is dependent on the amount of time required to complete a scan, which in turn is related to the speed of ultrasound and the depth of the image as well as the number of lines of information within the image. In general, the greater the number of frames per unit of time, the smoother and more aesthetically pleasing the real-time image. Factors that reduce frame rate, such as increasing depth of field, will diminish temporal resolution. This is particularly important for structures with relatively high velocity, such as valves. Temporal resolution is the main reason that M-mode echocardiography is still a useful clinical tool. With sampling rates of 1,000 to 2,000 images/s, temporal resolution of this modality is much higher than that of two-dimensional imaging.

**FIGURE 2.16.** Parasternal long-axis images demonstrate the effect of gain on the appearance on the echocardiographic image. **A:** Gain is adjusted appropriately to allow recording of all relevant information. **B:** Too much gain is used, distorting the image, reducing resolution, and increasing noise.
CREATING THE IMAGE

The instrument used to create an ultrasound image is called an echograph. It contains the electronics and circuitry needed to transmit, receive, amplify, filter, process, and display the ultrasound information. The essential components of the system are illustrated in Figure 2.17. As a first step, the returning energy is converted from sound waves to voltage signals. These are very low-amplitude, high-frequency signals that must be amplified and, because they arrive slightly out of phase, realigned in time. In modern instrumentation, this realignment is accomplished using a digital beam former to allow proper summation and phasing of all the channels. Because the signals are still very high frequency at this point, the scan lines are referred to as RF data. The complexity of the information at this stage is in part due to the wide range of amplitudes and the inclusion of background noise. Logarithmic compression and filtering are performed to render the RF
data more suitable for processing.

The polar scan line data at this point consist of sinusoidal waves, and each ultrasound target is represented as a group of these high-frequency spikes. Each group of high-frequency RF data is consolidated into a single envelope through a curve-fitting process called envelope detection. The resulting signal is then referred to as the polar video signal. This is sometimes called R-theta, indicating that each point in a polar map can be defined by its distance \((R)\) and angle \((\theta)\) from a reference point. The next very important step involves digital scan conversion and refers to the complex task of converting polar video data into a cartesian or rectangular format. The image formed at this stage can be either stored in digital format or converted to analog data for videotape storage and display.

**Figure 2.18** displays these different forms of imaging data as energy is received and processed by the echograph. The energy created by excitation of the piezoelectric elements is an RF signal (**Fig. 2.18A**). As discussed in the previous section, for the signal to be in a form that can be displayed visually, it must be converted to a video signal. This is accomplished by outlining (envelope detection) the outer edge of the upper portion, or positive deflection, of the RF signal (**Fig. 2.18B**). Differentiation of the video signal effectively accentuates the leading edge of the echo (**Fig. 2.18C**), providing a brighter signal and improving the ability to differentiate closely spaced targets. This is sometimes referred to as A-mode, for amplitude, imaging. Finally, intensity modulation converts the height or amplitude of the signal to a corresponding brightness level for video display (**Fig. 2.18D**). This is often called B-mode, for brightness, imaging and forms the basis of both M-mode and two-dimensional imaging display. How these various signal formats are used to create a visual display is covered in greater detail in a later section.

**TRANSMITTING ULTRASOUND ENERGY**

For most clinical applications, ultrasound is emitted from the transducer as a brief pulse of energy. A fundamental control feature is power output, which is simply the amount of ultrasound energy within each emitted pulse. For any given reflector, the higher the power output, the higher the amplitude of the returning signal. The pulse, which is a collection of cycles traveling together,
is emitted at fixed intervals (Fig. 2.19). The time between pulsing is referred to as the dead time and is largely a function of depth. During the dead time, the transducer is “listening” for returning signals. The duration of the ultrasound pulse is sometimes referred to as pulse length, and the pulse repetition period represents the total of one pulse length plus one dead time. To image at a greater depth, the dead time is lengthened, allowing the ultrasound system to listen for reflections arising from greater depths before returning to the transducer. Duty factor, or the percentage of time that the transducer is pulsing, is simply the pulse duration divided by the pulse repetition period. This is a very small number, in the range of 0.1%, indicating that the system is “on” for a brief time and “off,” or listening, for the majority of time. Each pulse of ultrasound energy results in the reception of a single line of ultrasound data.

FIGURE 2.18. Some of the key steps in image creation are illustrated graphically. See text for details.
FIGURE 2.19. Ultrasound energy is usually emitted from the transducer in a series of pulses, each one representing a collection of cycles. Each pulse has a duration and is separated from the next pulse by the dead time. The diagram is not drawn to scale. In reality, dead time is much greater than pulse duration. See text for details.

Pulsing in ultrasound is necessary to obtain range resolution, that is, to localize reflectors accurately along the axis of the beam. In theory, an emitted pulse must travel to the target and be reflected back to the transducer before a second pulse can be emitted to prevent interference and range ambiguity. Pulses are typically quite short, usually less than 5 ms. Unlike continuous-wave ultrasound, pulsed ultrasound results in a relatively broad frequency spectrum. The shorter the pulse duration is, the broader the frequency spectrum is (Fig. 2.20). This means that the distribution of frequencies occurs over a predictable range that are centered around a central frequency. This is referred to as **bandwidth**, and such a transducer is said to deliver a band of frequencies. Bandwidth has important effects on the texture of the image and the resolution. Transducers that deliver a wider bandwidth will provide higher axial resolution, primarily because the pulse length is shorter.
As pulse length increases, the frequency spectrum narrows.

\[ \therefore \text{Longer pulse length} \Rightarrow \text{narrower bandwidth} \Rightarrow \text{lower resolution} \]

**FIGURE 2.20.** The diagram demonstrates the relationship between pulse duration, or length, and bandwidth. With increasing pulse length, the bandwidth becomes narrower, thereby reducing resolution. Therefore, to improve resolution, a short pulse length should be employed.

To obtain an image, ultrasound must be transmitted, reflected, and received. A brief current of electricity intermittently excites the piezoelectric elements. This results in a pulse or burst of ultrasound that travels into the body while the transducer waits for the returning signal. Commercial echographs have repetition rates between 200 and 5,000/s. To perform M-mode examinations, pulse repetition rates of between 1,000 and 2,000/s are used. For two-dimensional imaging, repetition rates of 3,000 to 5,000/s are necessary to create the 90-degree sector scan. This does not mean, however, that temporal resolution is higher for two-dimensional imaging. In fact, the opposite is true. Although the pulse repetition rate is lower for M-mode, because all the pulses are devoted to a single raster line, the temporal resolution is actually much higher for M-mode compared with two-dimensional echocardiography. Diagnostic echographs are extremely sensitive receivers and can detect a signal that is greatly attenuated, which is necessary because less than 1% of the emitted ultrasound energy is typically reflected back to the transducer.

**Figure 2.21** demonstrates how one can use ultrasound to obtain an image
of an object. In this illustration, a transducer placed on the side of a beaker of water sends out short pulses of ultrasound. These pulses travel through the homogeneous water and are reflected at the interface between the water and the opposite beaker wall (part A). The pulse retraces its original path and strikes the transducer, which, functioning as a receiver, converts the mechanical vibration of the impact into an electric signal that is registered on the oscilloscope of the echograph. Because the velocity of the sound wave traveling through the water is known, the time it takes for the echo to leave the transducer and return to excite the crystal, sometimes called time of flight, can be measured and used to calculate the distance between the transducer and the opposite wall of the beaker. Although the echograph is actually measuring a “time” variable, the value can automatically be converted to “distance.” The various options for displaying this information, including A-, B-, and M-modes, are illustrated in Figure 2.21.

If an object, such as a rod, is placed in the center of the beaker, the same ultrasound beam would now first strike the rod, which is closer to the transducer than the far side of the beaker (Fig. 2.21B). In this case, some of the acoustic energy is reflected back from the rod, while a portion of the beam continues on to the far beaker wall before returning to the transducer. Both returning echoes would be recorded on the oscilloscope, indicating the position of the two targets relative to the transducer. Finally, if the rod is moved slowly within the beaker in a direction parallel to the sound beam, the distance between it and the transducer is constantly changing (Fig. 2.21C). Each pulse of ultrasonic energy will strike the rod at a different position relative to the transducer, and its motion can be graphed over time. How well the motion is visualized depends in part on the repetition rate of the ultrasound pulse, also known as the sampling rate or pulse repetition frequency (PRF) of the echograph. The higher the repetition rate, the more precisely the motion of the rod is tracked. Some of the important implications of PRF are discussed in greater detail in the next section.
In the previous section, the concept of signal processing of the returning ultrasound energy is discussed. The raw RF energy is sequentially converted to various forms, including an amplitude signal and a brightness form (Fig. 2.18). Returning to Figure 2.21, if the motion of the rod is visualized on the oscilloscope, it would appear as a bright signal moving back and forth on the
scope. This motion could be recorded by filming the oscilloscopic image. The motion could also be displayed using the technique of intensity modulation. This technique converts the amplitude of the echo (displayed as a spike) to intensity (displayed as a bright dot). In the amplitude mode (also known as A-mode), the height of the spike corresponds to the amplitude of the returning echo. In the brightness mode (known as B-mode), the intensity of the signal corresponds to the brightness of the dot.

Because the heart is a moving object, one can record that motion by introducing time as the second dimension. For example, if the tracing is swept from bottom to top, as is shown in Figure 2.21 (bottom panels), a wavy line is inscribed to indicate the motion of the rod. This is how an M-mode recording is created. In this case, M stands for motion and allows a single dimension of anatomy to be graphed against time. The intensity of any given echo within that display is represented as the density or thickness of the line, as is shown in the figure. By definition, the M-mode presentation depicts anatomy along a single dimension corresponding to the ultrasound beam creating what has been called the “ice-pick” view of the heart. The relationship among these display formats, as they relate to cardiac imaging, is illustrated in Figure 2.22.

Figure 2.23 shows how the echocardiographic system can record an M-mode tracing of the heart. In this example, the beam is directed toward the left ventricle. The ultrasonic beam also intersects a small portion of the right ventricular cavity. In the illustration, the M-mode recording was created using a strip chart recorder. The beam first passes through the chest wall structures, which are stable and unmoving. They appear as a series of straight lines. The echoes reflected by the anterior right ventricular wall are poorly visualized and recorded as a fuzzy band of reflections that are thicker during systole and thinner in diastole. The relatively echo-free space between the right ventricular wall and right side of the interventricular septum is a portion of the right ventricular cavity. The band of echoes running through the middle of the tracing represents the interventricular septum (right and left sides). Note that the left side of the interventricular septum moves downward in systole and upward in diastole. Next, echoes are seen originating from the posterior left ventricular wall with the endocardial echo having higher amplitude during systole than the epicardial echo. The less echogenic space between the endocardial and epicardial reflectors is the myocardium. The
echo-free space between the septum and the posterior left ventricular wall is the cavity of the left ventricle. Within this space, echoes from the mitral valve apparatus are intermittently recorded.

FIGURE 2.22. Echocardiography provides several display options. **Left:** A transducer is applied to the chest wall, and an ultrasound beam is directed through the heart at the level of the mitral valve. The returning ultrasound information can be displayed in amplitude mode (A-mode) in which the amplitude of the spikes corresponds to the strength of the returning signal. Amplitude can be converted to brightness (B-mode), in which the strength of the echoes at various depths is depicted as relative brightness. Motion can be introduced by plotting the B-mode display against time. This is the basis of M-mode echocardiography.
FIGURE 2.23. M-mode echocardiography is often described as an “ice-pick” view of the heart. The diagram shows the relationship of the transducer to the structures of the chest wall and heart. The corresponding M-mode echocardiogram provides relative anatomic information along a single line of information. ARV, anterior right ventricular wall; RS, right septum; LS, left septum; EN, posterior left ventricular endocardium; EP, posterior left ventricular epicardium.

In the early years of clinical echocardiography, M-mode scanning formed the backbone of clinical echocardiography. By positioning the transducer over different acoustic “windows” of the chest wall, single-dimensional images of cardiac structures could be recorded and inferences about structure, dimensions, and function could be made. Conversely, the B-mode display when held stationary to represent a one-dimensional format offered little in the way of useful diagnostic information. It was soon recognized, however, that a B-mode scan swept through a sector arc could provide a cross-sectional image that depicted structure and function in real time. This technique was originally called cross-sectional echocardiography and is now widely
referred to as *two-dimensional echocardiography*.

**Figure 2.24** compares the M-mode imaging examination with a two-dimensional sector scan and a three-dimensional volumetric scan. The object being recorded is a sphere moving as a pendulum within a beaker of fluid. Using the M-mode technique, the oscilloscope display shows a series of wavy lines that primarily depict the leading and trailing edges of the sphere as it moves relative to the transducer within the beaker (Fig 2.24A). Because the one-dimensional beam actually has a finite width or thickness, multiple secondary, less intense echoes are also recorded. Thus, the M-mode image provides an assessment of the dimensions of the object and its motion relative to the ultrasound beam. No information about motion in the orthogonal direction is provided and a complete recording of the object’s shape is lacking.

**FIGURE 2.24.** The relationship between M-mode and two-dimensional echocardiography is demonstrated. In (A), a circular object swings through a beaker of water on a string. The motion of the ball is recorded using M-mode echocardiography, as shown below. Only motion in a single dimension, relative to the transducer, is recorded. In (B), the same motion is visualized using two-dimensional imaging. In this case, motion in two dimensions is recorded as displayed in the lower panel. In (C), if the ball is moved through three dimensions, volumetric (3D) imaging would be required to completely track its motion.
If the same recording is created using two-dimensional imaging, more structural information is provided (Fig. 2.24B). Still, however, complete knowledge of the object is impossible because only two of the three spatial dimensions are included. In addition, two-dimensional imaging provides a more precise understanding of the true motion pattern compared with the M-mode recording. In the example, the simplistic M-mode recording suggests that the object is moving back and forth, whereas the two-dimensional recording confirms that the object is moving in an arc. Motion outside the plane of the scan is still not recorded, even with two-dimensional imaging. A key assumption in the discussion is that the rate of scanning through the sector arc (the PRF of the system) is sufficiently high relative to the movement of the object to record the motion accurately.

The next step in complexity is real-time three-dimensional imaging. By scanning through three dimensions, rather than two, a pyramid-shaped image is recorded (Fig. 2.24C). The challenge is to acquire the entire dataset quickly enough to allow accurate recording of cardiac motion. In the simple example shown (Fig. 2.24C), the object being recorded is clearly demonstrated to be a sphere, rather than a circle. Had there been motion out of the imaging plane, this also would be recorded with three-dimensional imaging.

Using a sophisticated two-dimensional array of elements and applying parallel computer processing techniques, acquisition rates of more than 60 volumes/s are currently possible. This is sufficiently robust to allow cardiac motion to be recorded and displayed. Using this approach, a more complete analysis of both shape and motion is accomplished. This technology is discussed in detail in Chapter 4.

**TRADEOFFS IN IMAGE CREATION**

Imaging a moving object, such as the heart, in real time creates a series of challenges. Not only must each “snapshot” be acquired rapidly enough to avoid blurring and distortion, but each successive snapshot must be captured at a sufficient rate to record the nuances and subtlety of motion smoothly and accurately. Then, each individual picture can be assembled into a motion picture that is the essence of real-time imaging. Because ultrasound travels at a fixed and relatively slow velocity through tissue, the ultimate rate at which
imaging information can be acquired and assembled is limited. Thus, tradeoffs and constraints exist that must be recognized.

The variables to consider include the desired depth of examination, the line density, the PRF, the sweep angle, and the frame rate. Constructing a complex, real-time image begins with emission of an ultrasound pulse that penetrates the body and returns information from varying depths. Because the velocity of sound in the body is essentially fixed, the time required to send and receive information is a function of depth of view. The rate at which individual pulses are transmitted is referred to as the PRF. Each pulse allows a single line of ultrasonic data to be recorded. To go from a single line of ultrasonic data to a two-dimensional image, the beam must be swept through an angle that typically varies from 30 to 90 degrees. The larger the angle is, the more lines are needed to fill the sector with data. Because line density is an important determinant of image quality, it is desirable to acquire as many ultrasonic lines as possible. The term *line density* refers to the number of lines per degree of sweep. A line density of approximately two lines per degree is necessary to construct a high-quality image.

Another important factor in image quality is frame rate. Depending on the speed of motion of the structure of interest, a higher or lower frame rate will be necessary to construct an accurate and aesthetically pleasing “movie” of target motion. For example, the aortic valve can move from the closed to the open position in less than 40 ms. At an imaging rate of 30 frames/s, it is likely that the valve will appear closed in one frame and open in the next, with no appearance of motion because intermediate positions were not captured. If one wished to record the aortic valve in an intermediate position, a very high frame rate must be employed. However, to increase the frame rate, additional compromises must be accepted. Specifically, increasing the frame rate generally results in a decrease in line density and degradation in image quality.

It should be appreciated that modern echocardiographic instruments use scan converters and forms of digital manipulation to convert the image into an aesthetically pleasing display format. Individual raster lines are thereby eliminated so that the appearance of individual lines radiating like spokes from the apex of the scan are no longer present. Instead, images are displayed on a television screen using the concept of *fields* and *frames*. A field is the total ultrasonic data recorded during one complete sweep of the beam. A
frame is the total sum of all imaging data recorded and generally implies that new information is superimposed on previously recorded data. With television technology, two fields are interlaced (to improve line density) to produce one frame. Using this approach, the frame rate would be half of the corresponding sweep rate.

**SIGNAL PROCESSING**

When the transducer acts as a receiver, the piezoelectric elements convert the returning ultrasonic energy to an electric impulse in the form of RF data. As previously discussed, the RF data are processed and converted to a video signal in which signal strength corresponds to brightness. Because of attenuation, signals returning from the most distant reflectors (i.e., structures at greater depth) will be the weakest or least bright echoes. By selectively amplifying echoes from greater depths, using a method referred to as time gain compensation, images of uniform brightness are created. This process allows returning signals from different depths to be selectively suppressed or amplified to provide relatively uniform signal strength (Fig. 2.25). Some control of depth compensation is provided on virtually all commercially available echocardiographic instruments. Although this is one of the most useful and important image control features, it is also a source of distortion and misuse. If one remembers that the purpose of this device is to compensate for the loss of ultrasonic energy (i.e., attenuation) as the beam propagates through the body, then one better understands how the controls should be used. The primary purpose is to enhance the far echoes and suppress near echoes, without creating distortion or artifact.

A late and very important stage in image creation involves the use of gray scale to display anatomic data. The challenge here results from discordance between the wide range of signal strength of the returning echoes and the limitations of the human eye to perceive differences in gray scale. The range of voltages generated during data acquisition extends over several log units, whereas the human eye is able to distinguish only approximately 30 shades of gray. The ultrasound instrument, using an operation called preprocessing, must reduce the range of the voltage signals to a more manageable number. Dynamic range is the extent of useful ultrasonic signals that can be processed
It is expressed in decibels and is defined as the ratio of the largest to smallest signals measured at the point of input to the display. At the low end, noise and undesired weak echoes exist that can be eliminated using a reject control. At the high end, signal saturation occurs and these echoes are also suppressed. In between, it is desirable to preserve as large a dynamic range as possible to ensure that all clinically important returning signals are included in the image. For example, scattered echoes are by definition much weaker than specular echoes, yet both are important in image construction. A mechanism to accommodate both is necessary, and this is accomplished through the use of a proper dynamic range. Through the technique of nonlinear compression, a wide dynamic range can be handled for processing by the scan converter.

The second challenge is how to convert the wide range of input signals into a manageable range of gray scales. With the exception of color flow imaging, echocardiography is essentially a black and white medium. An image is constructed of very small pixels that are assigned a gray level ranging from absolute white to absolute black. This is accomplished using a digital approach in which the range of brightness is divided into either 128 or 256 levels of gray (Fig. 2.27). The process of remapping the digital output of the scan converter to the range of gray scale values used in the video display is called post processing. This step permits manipulation of the imaging data to enhance the visual quality of the display.
FIGURE 2.25. The amplitude of returning signals is plotted against distance, or depth, from the transducer. Time gain compensation can be used to enhance the amplitude of the weaker signals returning from targets at greater depth and permits similar targets at different depths to be displayed accurately. On the right, the time gain compensation controls from an ultrasound machine are shown.

FIGURE 2.26. The schematic illustrates the concept of dynamic range. See text for details.

TISSUE HARMONIC IMAGING

As the ultrasound wave propagates through space, the transmitted, or fundamental, frequency of the signal may be altered due to nonlinear interactions with the tissue. The net effect of such interactions is the generation of frequencies not present in the original signal. These new frequencies are integer multiples of the original frequency and are referred to as *harmonics*. The returning signal contains both fundamental and harmonic frequencies. By suppressing or eliminating the fundamental component, an image is created primarily from the reflected harmonic energy. Unlike the harmonic technique that is so important to contrast echocardiography, in which the interaction of the ultrasound energy and the microbubbles produces vibrations that occur at multiple (harmonic) frequencies, tissue harmonics are generated during propagation by gradual conversion of energy from the transmitted frequency to one of its multiples. The development of tissue harmonics can be compared with an ocean wave that changes shape and speed as it approaches the beach. Similarly, the strength of the harmonic frequency actually increases as the wave penetrates the body. This is profoundly different from the fate of the fundamental frequency wave that attenuates constantly during propagation (Fig. 2.28A). This difference in behavior has important and practical implications for imaging. Close to the
chest wall, where many of the troublesome imaging artifacts are generated, there is very little harmonic signal. For this reason, imaging that exploits the harmonic frequency avoids many of the near field artifacts that affect fundamental imaging. At depths of 4 to 8 cm, the relative strength of the harmonic signal is near its maximum, whereas the fundamental frequency has diminished considerably. Thus, the harmonic signal is strongest at distances that are most relevant to transthoracic imaging.

A second feature of tissue harmonic imaging, again the result of nonlinear interactions, relies on the fact that strong fundamental signals produce intense harmonics and weak fundamental signals produce almost no harmonic energy. This phenomenon further reduces the artifact generation during harmonic imaging because most such artifacts result from weak fundamental signals. By producing images from the harmonic frequency reflections, the weak signals that cause many artifacts are disproportionately suppressed. The net result is that harmonic imaging reduces near-field clutter and many of the other sources of imaging artifact that plague fundamental frequency imaging. The signal-to-noise ratio is improved and image quality is enhanced, especially in patients with poor fundamental frequency images. A consistent finding in most studies has been improved endocardial border definition. However, an important side effect of tissue harmonic imaging is that strong specular echoes, such as those arising from valves, appear “thicker” than they would on fundamental imaging. This is particularly true in the far field and can lead to false-positive interpretations. To avoid such pitfalls but to take advantage of the benefits of harmonic imaging, most clinical studies should include both fundamental and harmonic imaging in the course of the examination.
Gray scale is a key concept in the creation of a two-dimensional image. The gray scale refers to the number of shades that can possibly be displayed between the two extremes of white and black. In the example, 256 shades are depicted. Each pixel is assigned one of these shades. In a digital system in which imaging data are stored as a binary code, eight bits are required to encode one of the 256 shades of gray.
FIGURE 2.28. A: Unlike fundamental frequencies, harmonic frequencies increase in strength as the wave penetrates the body. At the chest wall, where many artifacts are generated, very little harmonic signal is present. At useful imaging depths (4 to 8 cm), the relative strength of the harmonic signal is at its maximal. See text for details. B: The concept of pulse inversion technology is demonstrated. See text for details.

One application of harmonic imaging involves pulse inversion technology. Unlike tissue harmonic imaging, in which the fundamental signal is filtered, pulse inversion harmonic imaging takes a different approach to eliminating the fundamental frequency. In the pulse inversion mode, the transducer sequentially emits two pulses with similar amplitude but with inverted phase (Fig. 2.28B). When backscattered from a linear reflector such as tissue, and
then summed, these pulses cancel each other, resulting in almost complete elimination of the fundamental frequency signal, called *destructive interference*. The remaining harmonic energy can then be selectively amplified, producing a relatively pure harmonic frequency spectrum. The result is an image with many of the potential advantages previously attributed to tissue harmonic imaging. How much additional benefit can be ascribed to pulse inversion technology remains to be determined.

**ARTIFACTS**

The complexity of image creation using phased-array transducer technology is evident. Therefore, it should not be surprising that a variety of artifacts can occur that have a significant impact on image quality and diagnostic potential. One of the most important of these artifacts involves the generation of side lobes. Side lobes occur because not all the energy produced by the transducer remains within the single, central beam. Instead, a portion of the energy will concentrate off to the side of the central beam and propagate radially, a phenomenon known as edge effect. A side lobe may form where the propagation distance of waves generated from opposite sides of a crystal differs by exactly one wavelength. Side lobes are three-dimensional artifacts, and their intensity diminishes with increasing angle. The artifact created by side lobes occurs because all returning signals are interpreted as if they originated from the center beam. Hence, a weak-intensity echo originating from a laterally positioned target (but recorded via the off-axis side lobe) will be displayed as if it were located along the central axis of the main beam. It should be emphasized that side lobes are considerably weaker than the main beam so the returning echoes produced by a side lobe are also weaker. Side lobe reflections usually become evident when they do not conflict with real echoes. A prerequisite for a dominant side lobe artifact is that the source of the artifact must be a fairly strong reflecting target. The atrioventricular groove and the fibrous skeleton of the heart are examples of good sources of side lobe echoes ([Fig. 2.29](#)). When strong, these artifactual echoes can lead to significant problems in interpretation. Lesser degrees of side lobe artifact merely increase the general noise level of the system.

A second important source of artifacts in echocardiography is
reverberations. To understand how these occur, it is helpful to return to the example of a transducer held against a beaker of water (Fig. 2.21). In this case, the strongest reflector of the beam is the opposite beaker wall. As the reflected ultrasound returns to the transducer, it is likely that a portion of the returning signal undergoes a second reflection at the near beaker wall interface. This portion of the acoustic energy again reflects off the far wall and is finally returned to the transducer. With each step, the signal becomes weaker but may still be within the range of detection by the transducer. Most of the signal correctly identifies the far beaker wall as the primary reflector. That portion of the signal that makes two round-trips to the far wall also registers a signal. In this case, the pulse traveled twice as far and took twice as long before being detected, and was therefore incorrectly placed twice the distance from the transducer. This secondary echo represents a reverberation and occurs because of secondary reflection at the near beaker wall or at the surface of the transducer. In the clinical situation, such artifacts not only result from the beam reflecting from the transducer but also may originate from other strong echo-producing structures within the heart or chest (Fig. 2.30). Typically, a reverberation artifact that originates from a fixed reflector will not move with the motion of the heart. It appears as one or more echo targets directly behind the reflector, often at distances that represent multiples of the true distance. On the other hand, a mobile target may produce a reverberation that has twice the amplitude of motion as the original structure. In some cases, the source of reverberations is not apparent. These are particularly troublesome and frequently result in misinterpretation of the image.
FIGURE 2.29. Two examples of side lobes are shown (arrows). A: The strong echoes produced by the posterior mitral annulus and atroventricular groove produce a side lobe artifact that appears as a mass within the left atrium. B: Bright echoes within the pericardium produce a linear artifact that appears within the descending aorta and left atrium.
FIGURE 2.30. Reverberation artifacts are demonstrated. A: The source of the artifact is the posterior pericardium, which is a very strong reflector. This creates the illusion of a second structure behind the heart. In this case, the second line of echoes (far arrows) is twice the distance from the transducer as the actual pericardial echoes. B: A second lumen appears just distal to the descending aorta (DA) in this subcostal view. The illusion of a second vessel was apparent with two-dimensional imaging (*, B) and color Doppler imaging (C).
Another potential artifact is shadowing. Its appearance, in some ways, is the opposite of a reverberation. That is, instead of a series of echoes behind the source of the artifact, shadowing results in the absence of echoes directly behind the target (Fig. 2.31). Shadowing occurs when one attempts to visualize structures beyond a region of unusually high attenuation, such as a strong reflector. Because only a very small portion of the ultrasound beam can propagate beyond such a reflector, an acoustic shadow is created from which no reflections are produced. Perhaps the most relevant example of shadowing occurs in the setting of prosthetic valves. Such mechanical devices create strong reflectors behind which imaging is quite limited. Native structures that become heavily calcified are additional sources of shadowing. In this case, the presence of shadowing can be useful to identify the existence of strong reflectors, such as calcium. Contrast containing blood also produces shadowing, which when used improperly, limits its utility.

One additional source of artifact is termed near-field clutter. This problem, also referred to as “ringdown artifact,” arises from high-amplitude oscillations of the piezoelectric elements. This only involves the near field...
and has been greatly reduced in modern day systems. The artifact is troublesome when trying to identify structures that are particularly close to the transducer, such as the right ventricular free wall or left ventricular apex. This artifact is illustrated in Figure 2.32.

**DOPPLER ECHOCARDIOGRAPHY**

Doppler imaging is an integral and indispensable part of the echocardiographic examination. Knowledge of basic Doppler imaging principles is essential to fully understand the value and limitations of these techniques. Although Doppler imaging can be regarded as being complementary to two-dimensional imaging, the principles and instrumentation underlying this technique are substantially different. Used primarily to examine the flow of blood, Doppler imaging is concerned with the direction, velocity, and then pattern of blood flow through the heart and great vessels. The differences between B-mode or imaging echocardiography and Doppler imaging are fundamental (Table 2.4). The primary targets of the anatomic echocardiographic examination are the myocardium and valves of the heart. For Doppler imaging, the primary target is the red blood cells. Whereas echocardiography provides information on structure, Doppler imaging provides information on function. Thus, echocardiography can be regarded as an imaging technique that focuses on anatomy, whereas Doppler imaging focuses on physiology and hemodynamics. Finally, whereas echocardiography functions optimally when the beam and the target are at right angles, the Doppler equations rely on a more parallel alignment between the beam and the flow of blood. Thus, echocardiography and Doppler imaging provide diagnostic data that are largely complementary.
FIGURE 2.32. This apical two-chamber view demonstrates an artifact called near-field clutter (arrows). This is the result of high-amplitude oscillations emitted by the transducer and is a common source of misinterpretation.

| Table 2.4 A COMPARISON OF TWO-DIMENSIONAL ECHOCARDIOGRAPHY AND DOPPLER |
|--------------------------------------------------|-----------------|-----------------|
| Ultrasound target | Two-dimensional Echocardiography | Doppler |
| Tissue | Blood |
| Goal of diagnosis | Anatomy | Physiology |
Principles of Doppler Ultrasound

The Doppler principle is based on the work of the Austrian physicist Christian Doppler, first published in 1842. He studied the phenomenon that the apparent pitch of sound was affected by motion either toward or away from the listener. If the source of sound were stationary, then the pitch or frequency of that sound was constant. If, however, the source of sound moved toward the listener, the frequency increased and the pitch appeared to rise. Conversely, if the sound source was moving away from the listener, the frequency of the sound decreased relative to the listener and the pitch appeared lower.

The application of this phenomenon to blood flow measurement is illustrated in Figure 2.33. In this example, ultrasound is emitted from a transducer and reflected from a moving target such as a red blood cell. If that target is stationary, the frequency and wavelength of the emitted and reflected ultrasound are identical. If the target is moving toward the transducer, the reflected frequency is “shifted” upward proportional to the velocity of the target relative to the transducer. Conversely, movement of the target away from the transducer results in the reflected ultrasound having a lower frequency than the emitted ultrasound, a downward shift in frequency. The increase or decrease in frequency due to relative motion between the transducer and the target is referred to as the Doppler shift.
FIGURE 2.33. The basic principles of the Doppler phenomenon are illustrated. **Top:** A stationary source of sound produces a given pitch or frequency. If the sound is moving toward a recorder, the pitch appears increased and if the sound is moving away from a recorder, the pitch appears decreased. **Bottom:** This same concept is applied to blood flow. If the red blood cells are moving toward the transducer at a given velocity ($v$), the reflected frequency ($f_r$) will be higher than the emitted frequency ($f_0$). If the red blood cells are moving away from the transducer, the opposite will occur.
FIGURE 2.34. Calculation of the Doppler shift requires knowledge of the transmitted frequency \( f_0 \), the reflected frequency \( f_r \), the angle of incidence \( \theta \), and the speed of sound. See text for details.

In addition to the qualitative observation of the frequency shift, Christian Doppler also described the mathematical relationship between the magnitude of the frequency shift and the velocity of the target relative to the source. As can be seen in Figure 2.34, the Doppler shift \( \Delta f \) depends on the transmitted frequency of the ultrasound, the speed of sound, the intercept angle between the interrogating beam and the flow, and, finally, the velocity of the target.

\[
\Delta f = \frac{2f_0 \nu}{c} \cos \theta
\]  

[Eq. 2.3]

Based on Eq. 2.3, it can be seen that the actual Doppler shift is quite small. For example, using a 3-MHz transducer to sample blood flowing toward the transducer at 1.0 m/s, the received frequency is increased up by only 4 KHz, from 3.0 MHz to 3.004 MHz. It is further apparent from the equation that the Doppler shift depends not only on blood velocity, but also on the angle of incidence, \( \theta \).

\[
\Delta f \propto \nu \cos \theta
\]  

[Eq. 2.4]

Thus, the velocity of blood flow (the unknown variable) is directly related to the Doppler shift (what is actually measured by the instrument) corrected for the angle \( \theta \). This angle correction actually depends on the cosine of \( \theta \), which has a predictable and critically important effect on the calculation of velocity. Because the cosine of 0 degrees = 1, this correction (i.e., multiplying by 1) has no net effect on the calculation of the Doppler shift.
Thus, the derived blood flow velocity is the true velocity. As the angle between the beam and the blood flow direction increases from 0 to 90 degrees, the cosine θ decreases from 1 to 0. The relationship between θ and cosine θ is shown in Figure 2.35A. For any angle other than 0, multiplying by the cosine θ results in a decrease in calculated velocity. Consequently, misalignment of the interrogating beam will lead to underestimation but never overestimation of true velocity. For practical purposes, this only becomes significant beyond approximately 20 degrees. As can be seen in the graph, if θ equals 10 degrees, cosine θ equals approximately 0.98 and the degree of underestimation is trivial. As θ increases to 30 degrees, cosine θ becomes 0.83 and the true velocity is underestimated by 17%. As the angle increases further, the rate of underestimation increases rapidly. The effect of angle θ on the accuracy of the Doppler gradient calculation is illustrated in Figure 2.35B. For example, if a jet with a peak velocity of 5 m/s is properly aligned, an accurate pressure gradient of 100 mm Hg will be measured. If the same jet is recorded at an incident angle (θ) of 30 degrees, the calculated gradient will be approximately 75 mm Hg, a significant underestimation.

FIGURE 2.35. A: The effect of intercept angle on the Doppler equation is shown. See text for details. B: The intercept angle has an important effect on the accuracy of velocity measurement. This effect is magnified at higher velocity and becomes increasingly important as the intercept angle increases from 0 to 40 degrees, as shown by the different curves. See text for details.

Another important component of the Doppler equation is the transducer or carrier frequency, which is a primary determinant of the maximal blood flow velocity that can be resolved. The relationship between the Doppler shift and blood flow velocity at four different transmitted frequencies is illustrated in Figure 2.36. A high-flow velocity such as 5 m/s is more readily recorded

\[ \Delta f = \frac{2 \Delta V \cos \theta}{c} \]

\[ \text{Parallel to flow} \]

\[ \text{Perpendicular to flow} \]

\[ \text{Increasing angle (θ) between Doppler beam and blood flow direction} \]

\[ \text{Jet velocity (V_j) in m/s} \]

\[ \text{Calculated peak gradient (mm Hg)} \]
using a low-carrier frequency such as 1 MHz compared with a high-
transducer frequency such as 5 or 10 MHz because of the corresponding
Doppler shift. In this respect, Doppler imaging is the opposite of
echocardiographic imaging. With echocardiography, a higher-transducer
frequency is desirable because it is associated with higher resolution. With
Doppler imaging, a lower frequency is advantageous because it allows high-
flow velocity to be recorded.

The primary job of the Doppler instrument is to measure the Doppler shift,
and from this measurement, velocity can be calculated. The Doppler shift is
defined as the difference in frequency between the transmitted and received
or backscattered signal. In cardiac imaging, values are generally in the 5 to 20
KHz range, well within the audible range of human hearing. The process of
determining the Doppler shift is a complex one, referred to as *spectral
analysis*. This involves a comparison of the actual waveforms of the
transmitted and received frequencies using a method called fast Fourier
transform analysis. The net result of this analysis is a spectral display of the
entire range of velocities.

**Doppler Formats**

For cardiovascular applications, there are five clinically relevant types of
Doppler techniques: continuous-wave Doppler, pulsed-wave Doppler, color
flow imaging, tissue Doppler, and duplex scanning. Pulsed-wave Doppler
transmits and receives energy in a similar fashion to anatomic (two- and
three-dimensional) imaging. Short, intermittent bursts of ultrasound are
transmitted into the body. Although targets at multiple points along the beam
may reflect the transmitted ultrasound, the pulsed Doppler instrument only
“listens” at a fixed and very brief time interval after transmission of the pulse
(*Fig. 2.37*). This permits returning signals from one specific distance from the
transducer to be selectively received and analyzed, a process called *range
resolution*. By adjusting the timing between transmission and reception,
different ranges or depths can be evaluated. This effectively creates a sample
volume at a specified point along the transmitted beam that can be positioned
within the field of view to permit blood flow velocity information to be
sampled. Using a superimposed two-dimensional image for purposes of
localization, pulsed-wave Doppler imaging interrogates the distribution of
blood flow values within a relatively limited region.

![Graph showing the relationship between Doppler shift and blood flow velocity for four different transducers.](image)

**FIGURE 2.36.** The relationship between the Doppler shift and blood flow velocity for four different transducers is shown. The graph demonstrates that lower-frequency transducers are capable of resolving higher-velocity flow. See text for details.

An important limitation of pulsed Doppler imaging is the maximal velocity that can be accurately resolved. This occurs because of the phenomenon referred to as *aliasing*. The number of pulses transmitted from a Doppler transducer each second is called the PRF. Sampling rate is an important determinant of how accurately the system resolves frequency information. To accurately represent a given frequency, it must be sampled at least twice, that is:

\[
PRF = 2 \times f_{DOP} \quad \text{[Eq. 2.5]}
\]

This formula establishes the limit (Nyquist limit) below which the sampling rate is insufficient to characterize the Doppler frequency. This key concept is demonstrated in **Figure 2.38**. In **Figure 2.38A**, a sine wave of fixed wavelength is tracked at three different sampling rates. In the top panel, the sampling rate is sufficiently high relative to the wavelength (17 times in four wavelengths or 4.25 per cycle) that the frequency can be reasonably estimated. This is indicated by how well the dashed line (sampling rate) tracks the solid line (the ultrasound wave). In the middle panel, a lower sampling rate (11 times every four wavelengths) results in a less precise
tracking of the true frequency. In the bottom panel, by sampling only seven times over the four cycles, it is impossible to accurately characterize the frequency of the wave. The relevance of this phenomenon to pulsed-wave Doppler imaging is shown in Figure 2.38B. In each panel, a constant sampling rate, or PRF (11 times over time t, indicated by the vertical arrows), is maintained. This results in a Nyquist limit of 5.5. In the top panel, this sampling rate is adequate to characterize the relatively low-frequency wave (a frequency of three cycles per time t). As the frequency increases, the sampling rate will eventually become too slow to follow the frequency. For example, in the middle panel, the frequency has increased to five cycles per time t. This frequency is still below the Nyquist limit, so aliasing does not occur and the true frequency is accurately resolved. In the bottom panel, at a frequency of eight per time t, the Nyquist limit of 5.5 has now been exceeded and aliasing occurs. Practically speaking, aliasing is the inability of a pulsed-wave Doppler system to detect the higher-frequency Doppler shifts. The upper limit of frequency that can be detected by a given pulsed system is the Nyquist limit, which is defined as one-half the PRF.

FIGURE 2.37. The differences between pulsed- and continuous-wave Doppler imaging are illustrated. See text for details.
Figure 2.38 demonstrates the concept of aliasing graphically in this schematic. See text for details.

Figure 2.39 shows a sample volume at the level of the mitral valve in a patient with mitral regurgitation. High-velocity flow occurs in systole and is directed away from the transducer. Because this velocity exceeds the Nyquist limit, the Doppler signal aliases and appears to wrap around the baseline. Aliasing creates confusion as to the direction of flow and prevents an accurate measure of maximal velocity. Figure 2.40 illustrates the relationship between sample volume depth, or range, and the maximal velocity that can be resolved. Note that the relationship again depends on the transducer frequency. As the depth increases, the maximal velocity that can be accurately detected decreases. However, for any given depth, a lower-frequency transducer permits higher velocities to be resolved compared with a higher-frequency transducer.

Continuous-wave Doppler imaging differs fundamentally from pulsed Doppler and anatomic echocardiography. Rather than sending out intermittent pulses of information, continuous-wave Doppler imaging simultaneously transmits and receives ultrasound signals continuously. This can be accomplished in one of two ways. One type of transducer employs two distinct elements: one to transmit and the other to receive (Fig. 2.41).
Alternatively, with phased-array technology, one crystal within the array is dedicated to transmitting while another is simultaneously receiving. Because the transmitted signal is not pulsed, range resolution is impossible and the reflected signals all along the ultrasound beam are sampled simultaneously. Thus, it is impossible to know where along the sample beam that any recorded velocity signal arises. Using a variety of amplification and signal-processing techniques, however, both the direction and the velocity spectrum of blood flow are recorded. A major advantage of continuous-wave Doppler imaging is that aliasing does not occur and very high velocities can be accurately resolved. The combination of pulsed- and continuous-wave Doppler imaging forms a powerful tool for clinical applications.

High-PRF Doppler imaging is a technique that combines features of both pulsed- and continuous-wave Doppler imaging. Using pulsed-wave Doppler imaging, velocity within a single sample volume is determined by receiving signals only at the point in time that corresponds to that depth. However, the listening window will also capture returning signals from twice the depth that were emitted by the previous ultrasound pulse. Using this approach, velocity information from the primary sample volume as well as integer multiples of that depth can all be analyzed during a single listening event. If the sample volume is placed at one-half of the actual depth of interest, velocity information from both sites can be recorded over two consecutive pulses. Because the use of the shallower sample volume depth is associated with a higher PRF, higher velocities can also be analyzed without aliasing. Although some degree of range ambiguity is inherent, this has limited practical effects. By positioning multiple sample gates along the beam, a significant increase in PRF is achieved, allowing relatively high velocities to be resolved with a modest loss of range resolution.
FIGURE 2.39. An example of aliasing is provided. Using pulsed-wave Doppler imaging, the sample volume is placed in the left atrium, just beyond the mitral valve. In systole, mitral regurgitation produces a high-velocity jet that cannot be resolved with the pulsed-wave Doppler technique. Aliasing of the jet is the result.
This graph demonstrates the relationship between range, or depth, and the maximal velocity that can be resolved, using two different transducer frequencies. The relationship is given by the equation. In both cases, as depth increases, the maximal velocity that can be recorded decreases. However, for any given depth, the lower-frequency transducer is capable of resolving higher velocities compared with the higher-frequency transducer. \( V_{\text{max}} \), maximal velocity; \( c \), velocity of ultrasound; \( f_0 \), transducer frequency; \( R \), range.


Because Doppler imaging provides information on direction and velocity of flow, it is useful to display this information graphically by plotting instantaneous flow velocity against time. By convention, velocity is displayed on the vertical axis with flow toward the transducer above the baseline and flow away from the transducer below the baseline (Fig. 2.42). In the illustration, aortic flow accelerates toward the transducer in systole, with very little flow occurring during diastole. A relatively thin envelope of the Doppler signal indicates that the flow is essentially laminar. Under physiologic conditions, most examples of blood flow in the cardiovascular system are laminar, meaning that individual blood cells are traveling at approximately
the same speed in approximately the same direction parallel to the walls of the chamber or vessel. Of course, some range of velocities naturally occurs. For example, velocity tends to be higher in the center of a vessel and lower near the vessel wall, as predicted by basic hydraulic principles (Fig. 2.43). As shown in the schematic, a flat, laminar profile is characteristic of large straight vessels. Flow tends to become more parabolic (i.e., less flat) as vessel size decreases. Flow through a curved vessel is characterized by higher velocities along the outside wall and lower velocities nearer the inside. Flow through a bifurcation produces eddy currents along the inner side of the branches, but relatively laminar flow along the outer walls. Blood flow through a U-shaped vessel, such as the aortic arch, is complex, depending on the profile of flow entering the arch, the angle of curvature, and the centrifugal forces acting on the blood. Even within the heart itself, flow remains generally laminar and occurs over a relatively narrow range of velocities. In pathologic situations, such as valve abnormalities or congenital defects, flow tends to become turbulent, often with abnormally high velocity.

**FIGURE 2.41.** A nonimaging, or Pedoff, continuous-wave Doppler transducer is shown. The transducer contains two elements: one for transmitting and one for receiving.
Doppler instrumentation depends on an ability to record and display the range of velocities and directions within a region of interest. By digitizing a snapshot of Doppler shift information and then applying a complex mathematical technique called fast Fourier transform, the instantaneous flow velocity spectrum can be displayed. At each instant, the range of velocities determines the width of the Doppler signal and the frequency distribution of each individual velocity is represented by the gray scale. In the cardiovascular system, most flow is pulsatile. Purely laminar flow has a narrow envelope of velocities, indicating that most of the blood cells travel over a narrow range of velocity. With increasing turbulence, both the direction and the range of velocities increase, and this leads to a widening of the spectral pattern as shown in Figure 2.44. Thus, a narrow spectral envelope indicates the presence of laminar flow, whereas spectral broadening is
consistent with turbulence. It should be emphasized that this distinction is only possible when using pulsed-wave Doppler imaging. Because continuous-wave Doppler imaging samples at multiple sites along the beam, a narrow spectral envelope almost never occurs.

FIGURE 2.43. Various types of flow patterns are shown. See text for details.
FIGURE 2.44.  

A: A laminar flow profile occurs when most of the red blood cells are traveling in approximately the same direction at approximately the same velocity. In a pulsatile system, this results in a Doppler signal that has a narrow envelope, as shown on the right. This would be typical of systolic flow through the aortic valve. 

B: The changes seen in the setting of turbulent flow as might occur with aortic stenosis are shown. In this case, blood accelerates through a narrow orifice and becomes disturbed distal to the site of obstruction. This has two primary effects on the Doppler signal: velocity increases (as flow accelerates) and spectral broadening occurs.

COLOR FLOW IMAGING

Color flow imaging is a form of pulsed-wave Doppler imaging that uses multiple sample volumes along multiple raster lines to record the Doppler shift, based on principles described earlier for pulsed-wave and high-PRF Doppler imaging. By overlaying this information on a two-dimensional or M-mode template, the color flow image is created. Constructing the color flow image is complex. Each pixel represents a region of interest in which the flow characteristics must be measured. Rather than analyzing the entire velocity spectrum within one of these small regions (which would require several seconds for each image if a complete Fourier transform were performed), some compromises are necessary and only mean frequencies and frequency spreads (variance) are calculated.
As a first step, for each pixel, the strength of the returning echo is determined. If it is above a predetermined threshold, it is painted a shade of gray and displayed as a two-dimensional echocardiographic data point. If it is below the threshold, it is analyzed as Doppler information. By repetitive sampling, an average value for mean velocity and variance is determined with greater accuracy. The flow velocity, direction, and a measure of variance are then integrated and displayed as a color value (Fig. 2.45). By performing such operations extremely rapidly over the entire range of the Doppler overlay, a color pattern is created that provides information on flow characteristics. By using a color reject threshold, only flow above a given velocity level is displayed as color. This limits the potential for “information overload” and allows the observer to integrate the Doppler and gray scale image information in a meaningful way. A color algorithm can be constructed to display these multiparametric data. For example, the direction of flow can be displayed using red (toward) and blue (away). The brightness of these primary colors encodes the magnitude of the mean velocity. High variance, or turbulence, is coded green, which, when mixed with red or blue, yields yellow or cyan, respectively, often with a mosaic appearance. Color Doppler information is most commonly displayed in conjunction with real-time two-dimensional gray scale imaging. It can also be displayed volumetrically, in three dimensions, and this is being done with increasing frequency. Frame rates for 3D color remain suboptimal and the format to display such information is challenging. Both of these limitations should improve with ongoing updates in computer technology. The ability to visualize regurgitant jets in three dimensions has potential advantages. Thus, three-dimensional color applications will continue to expand over the coming years.
Technical Limitations of Color Doppler Imaging

By “visualizing” the velocity of flow in a two-dimensional format, color Doppler imaging has been used extensively to assess abnormal flow patterns such as valvular regurgitation. Although routinely used for clinical decision making, the technical limitations of this technique are considerable. As described previously, the instrumentation needed to construct a color flow map is complex and involves several compromises and manipulations. Although color Doppler imaging is a very sensitive technique for detecting regurgitation, the relationship between jet size and regurgitation severity is complex. For example, remember that jets are three-dimensional entities that can never be completely captured in a two-dimensional format. For this reason, jets should always be recorded in multiple views to qualitatively assess their three-dimensional size.

It is worth exploring the various factors that affect the relationship between color Doppler jet area and regurgitation severity. It is well established that the severity of regurgitation is best described by the flow rate \( Q \), which is the product of jet velocity \( v \) and effective regurgitant orifice area \( A \). However, color jet size is more closely related to jet momentum \( M \), which is given by

\[
M = Q \times v \quad [\text{Eq. 2.6}]
\]
Since $Q = v \times A$, then

$$M = A \times v^2 \quad [\text{Eq. 2.7}]$$

This suggests that, for a given ROA, jet size will vary with the square of velocity. The clinical implications of this relationship are profound. For example, a patient with mitral regurgitation typically demonstrates a regurgitant jet velocity between 4 and 6 m/s. If the patient’s blood pressure increases, the regurgitant jet velocity might rise from 4 m/s to 5 m/s. This will increase the apparent jet area by as much as 40%, a much greater increase than the actual change in regurgitation severity. Thus, color jet appearance is very blood pressure dependent. This means that regurgitant jets with high velocity (e.g., a mitral regurgitation jet in the presence of aortic stenosis) will appear substantially larger than a tricuspid regurgitation jet, which has a much lower velocity. If color Doppler imaging is performed when blood pressure is either very high or very low, this clinical information should be noted and taken into account when the study is interpreted.

Chamber constraint is another factor that determines jet size. As a jet propagates beyond the orifice, velocity decreases with distance. This rate of velocity decline will be greater for eccentric, or constrained, jets than for central jets. For this reason, an eccentric (wall hugging) jet will appear relatively smaller than a central jet of similar severity. For the same reason, chamber size can also influence the apparent area of a color flow jet, with smaller chambers being associated with smaller appearing color Doppler jets.

Among the most important determinants of jet size are instrument settings (see Table 2.5). For example, by adjusting the color scale, PRF is altered and jet size can change dramatically. By lowering the scale (or Nyquist limit), the lower-velocity blood at the periphery of the jet becomes encoded and displayed, making the jet appear larger. In general, the color scale should be set as high as possible for a given depth. Increasing the wall filter will have the opposite effect; this will reduce the jet size by excluding velocities at the periphery. Power and instrument gain will also alter jet size. Increasing these settings will increase jet area. To optimize the settings, color gain should be increased until color pixels appear within the tissues, then the gain should be reduced slightly. Finally, transducer frequency has a complex effect on color jet area. The jet size will tend to increase with high-carrier frequency because
of the relationship between velocity and the Doppler shift. On the other hand, greater attenuation at higher frequency will make jets appear smaller. For all these reasons, instrument settings can profoundly affect the clinical utility of color flow imaging. It is recommended that most settings related to color imaging should be optimized when the machine is first set up and then left unchanged, to the degree possible, to maximize consistency.

<table>
<thead>
<tr>
<th>Table 2.5</th>
<th>FACTORS THAT AFFECT COLOR DOPPLER JET AREA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Factor</strong></td>
<td><strong>Impact on Jet Area</strong></td>
</tr>
<tr>
<td>Physiologic</td>
<td>Increase in blood pressure</td>
</tr>
<tr>
<td></td>
<td>Increased jet velocity</td>
</tr>
<tr>
<td></td>
<td>Eccentric jet (wall hugging)</td>
</tr>
<tr>
<td></td>
<td>Slit-like orifice</td>
</tr>
<tr>
<td></td>
<td>Multiple orifices</td>
</tr>
<tr>
<td>Technical</td>
<td>Higher pulse repetition frequency (PRF)</td>
</tr>
<tr>
<td></td>
<td>Decrease in Nyquist limit</td>
</tr>
<tr>
<td></td>
<td>Increase in color Doppler gain</td>
</tr>
</tbody>
</table>
FIGURE 2.46. A: This is a schematic depiction of mitral regurgitation, with the triangles representing blood within the left ventricle, and the circles indicating left atrial blood. B: Mitral regurgitation is demonstrated by some of the triangles moving through the orifice into the left atrium. The effect of those cells on the left atrial blood (circles) is shown. Because of the increase in velocity, some of the triangles and some circles are encoded and displayed by the color Doppler signal (filled triangles and circles). See text for details.

From the above discussion, it should be clear that color Doppler imaging and angiography are fundamentally different. If contrast is injected into the left ventricle of a patient with mitral regurgitation, any contrast that appears in the left atrium must come through the mitral valve as regurgitation. The amount of contrast that is visualized in the atrium, although impossible to quantify, correlates with the regurgitant flow volume. Doppler imaging, however, records velocity rather than flow. Thus, the color jet that is seen in the left atrium includes not only red blood cells that regurgitate through the
mitral valve but also blood that was already in the atrium and is being displaced by the incoming jet. This has been called the “billiard ball effect” and is illustrated in Figure 2.46. In the upper diagram, the blood in the left ventricle is depicted by the triangles and the left atrial blood by the circles. In the lower panel, some left ventricular blood has entered the left atrium through the incompetent mitral valve (filled triangles). This blood displaces the left atrial blood, transferring some of its energy and forcing the atrial blood to accelerate away from the regurgitant orifice (filled circles). If the velocity of this left atrial blood is sufficiently high, it will be detected by color Doppler imaging, just as the blood that accelerated through the regurgitant orifice is similarly detected. Thus, Doppler imaging records velocity, not flow. It cannot distinguish whether the moving left atrial blood originated in the ventricle (the filled triangles) or atrium (the filled circles), simply that it has sufficient velocity to be detected. Unlike angiography, the Doppler jet consists of both atrial and ventricular blood, all of which is moving faster than a predetermined velocity.

The important difference between velocity and flow is further illustrated in Figure 2.47. This schematic demonstrates yet another limitation of using color Doppler imaging for regurgitant flow quantification. The ROA is a fundamental measure of regurgitation severity. In this example, three different sizes of ROA are shown, along with their corresponding jet areas. As the ROA increases, flow rate increases and more blood enters the chamber and is detected by the Doppler method (middle panel). However, because velocity is inversely related to orifice area, as the ROA increases, the velocity of the regurgitant jet may decrease (if the pressure gradient is less). Because Doppler imaging records velocity, this larger (but lower velocity) flow may be recorded as a smaller color jet (lower panel). For this reason, a tricuspid regurgitation (lower gradient and velocity) jet area corresponds to a greater regurgitant volume than a mitral regurgitation (higher gradient and velocity) jet area of similar size.
FIGURE 2.47. This diagram demonstrates how turbulent flow through a regurgitant orifice relates to color Doppler signal. See text for details. ROA, regurgitant orifice area.

Despite these limitations, color flow imaging can provide a semiquantitative approach to regurgitation severity. When viewed in real time, with proper instrument settings, jet area and regurgitant volume are correlated. Analyzing such images, however, can be confusing. Color Doppler imaging aliases at a low velocity, so jets change color frequently, in part because of changes in velocity and in part because of changes in location relative to the transducer (Fig. 2.45). Because of the low frame rate of color Doppler imaging, rapidly moving structures, such as valves, can produce color artifacts. Due to the large number of operations that must be rapidly
performed to construct each color image, any one frame can contain artifacts or ghosts. For this reason, using stop-frame techniques to measure jet dimensions should be done very cautiously. Real-time viewing tends to filter out much of the insignificant artifacts seen on stop-frame analysis. By integrating information over many cardiac cycles, useful diagnostic data are available. On the other hand, a single color frame can never convey a complete depiction of the true jet dimensions and often results in the measurement of artifact or noise rather than real flow information. For that reason, planimetry of color jets on a stop-frame image is not recommended.

**Doppler Artifacts**

As is the case with two-dimensional imaging, the creation of the Doppler image involves the potential production of artifacts. Some of these are related directly to the Doppler principle. For example, aliasing occurs when pulsed-wave Doppler techniques are applied to flow velocities that exceed the Nyquist limit. This topic has already been covered in detail. A commonly encountered artifact is called *mirror imaging*, also called *crosstalk*. As the name suggests, this is the appearance of a symmetric spectral image on the opposite side of the baseline from the true signal. Such mirror images are usually less intense but similar in most other features to the actual signal (Fig. 2.48). These artifacts can be reduced by decreasing the power output and optimizing the alignment of the Doppler beam with the flow direction.

**FIGURE 2.48.** Two examples of mirror image artifact are demonstrated. **A:** Descending aortic flow appears to occur both above and below the baseline. **B:** A
A stenotic porcine mitral valve is recorded with pulsed-wave Doppler imaging. The intensity of the signal results in a classic mirror image artifact.

Beam width artifacts are common to all forms of ultrasound imaging. With pulsed-Doppler imaging, it must be remembered that the sample volume(s) has finite dimensions that tend to increase with depth. A sample volume placed in the far field is large enough to straddle more than one flow jet. For example, left ventricular inflow and outflow can often be recorded simultaneously from the apical four-chamber view. This is because the sample volume at that depth is broad enough to simultaneously record both flow patterns. This is sometimes desirable, permitting the timing and velocity of different flow patterns to be compared (Fig. 2.49). However, beam width artifact often has less desirable effects. For example, a large sample volume may hinder one’s ability to distinguish aortic stenosis from mitral regurgitation.

**FIGURE 2.49.** Beam width artifacts in Doppler imaging can be clinically useful. In this case, the thickness of the Doppler beam allows simultaneous recording of both aortic outflow and mitral inflow. This permits the isovolumic relaxation time to
Color Doppler imaging can be affected by several types of artifacts. Shadowing may occur, masking color flow information beyond strong reflectors. Ghosting is a phenomenon in which brief swathes of color are painted over large regions of the image. Ghosts are usually a solid color (either red or blue) and bleed into the tissue area of the image (Fig. 2.50). These are produced by the motion of strong reflectors such as prosthetic valves. They tend to be very transient and do not correspond to expected flow signals. Ghosting is most problematic when color flow images are frozen to analyze or planimeter a jet.

Finally, it should be remembered that color Doppler imaging is very gain dependent. Too much gain can create a mosaic distribution of color signals throughout the image. Too little gain eliminates all but the strongest Doppler signals and may lead to significant underestimation of jet area. With experience, the operator learns to adjust the gain settings to eliminate background noise, without oversuppression of actual flow information.

FIGURE 2.50. Ghosting occurs when brief displays of color are painted over
regions of tissue, as shown in the illustration. See text for details.

**FIGURE 2.51.** An example of tissue Doppler imaging is provided. The sample volume is positioned within the mitral annulus and velocity of the annulus is recorded throughout the cardiac cycle.

**Tissue Doppler Imaging**

Another application of the Doppler principle is tissue Doppler imaging. By adjusting gain and reject settings, the Doppler technique can be used to record the motion of the myocardium rather than the blood pool. To apply Doppler imaging to tissue, two important differences must be recognized. First, because the velocity of the tissue is much lower than blood flow, the ultrasound instrument must be adjusted to record a lower range of velocities. Second, because the tissue is a much stronger reflector of the Doppler signal compared with blood, additional adjustments are required to avoid oversaturation. When these factors are taken into account, tissue velocity can be recorded. An example of tissue Doppler imaging is provided in Figure 2.51. Note how the sample volume is positioned within the mitral annulus to record the motion of the annulus throughout the cardiac cycle. One obvious limitation is that the incident angle between the beam and the direction of target motion varies from region to region. This limits the ability of the
technique to provide absolute velocity information, although direction and relative changes in tissue velocity are displayed.

Once tissue velocity has been determined, several derived parameters can be displayed, including displacement, strain, and strain rate. Strain is a measure of the deformation that occurs when force is applied to tissue. Strain rate is simply its temporal derivative. By measuring instantaneous velocity at two closely positioned points within the myocardium and knowing the initial distance between two points, both strain and strain rate can be determined. The Doppler tissue imaging technique has been used successfully to derive the velocity information needed to calculate strain. By comparing velocity at two closely located points, it has the potential advantage of avoiding the confounding effects of translational motion. However, because it is a Doppler technique, angle dependency remains an issue. The potential applications of strain and strain rate imaging are discussed more fully in Chapters 3 and 6.

BIOLOGIC EFFECTS OF ULTRASOUND

Some of the success and popularity of echocardiography can be attributed to the safety and risk-free nature of ultrasound. In addition to being completely noninvasive, the biologic effects of ultrasound, as used in routine clinical situations, pose minimal risks to the patient. Ultrasonic examination of many parts of the body, including potentially sensitive tissues such as a developing fetus and the eye, has been performed on millions of patients without documentation of a single serious adverse event. Still, the question of safety when an external energy source is transmitted into the body must be considered. Newer applications and instruments may involve higher levels of energy, so the potential impact of such approaches should also be examined.

The biologic effects of ultrasound depend on the total energy applied to a given region. Thus, both the intensity of the ultrasound beam and the duration of exposure are important factors. Acoustic energy is measured in joules, which is defined as the amount of heat generated by the transmission of ultrasound. Recall that power is the amount of acoustic energy per unit of time and intensity is the acoustic power per unit of area. For example, the power level is 1 W if 1 J of energy is produced in 1 s. A milliwatt is 0.001 W. The biologic effects of ultrasound are generally discussed in terms of power,
and the units of power are in the milliwatt range. Intensity is usually expressed as watts per meter squared (W/m\(^2\)) or in milliwatts per centimeter squared (mW/cm\(^2\)). The actual measure of intensity is complex in biologic systems and typically reported as spatial peak (SP) intensity, spatial average (SA) intensity, or intensity at a particular point. As discussed previously, intensity varies spatially across the ultrasound beam. Thus, SA intensity is equal to the total power emitted by the transducer divided by the cross-sectional area of the ultrasound beam. If the power output is 2.0 mW and the beam area is 1.0 cm\(^2\), then SA intensity would be 2.0 mW/cm\(^2\). SP intensity will usually occur at the center of the beam where power is most concentrated.

Measuring the intensity of the beam in a pulsed-mode system is more complicated. When ultrasound is transmitted in pulses, the intensity will vary both spatially and temporally, depending on the pulsing sequence. This latter factor depends on both the pulse duration and the pulse repetition period. To calculate the energy from a pulsed ultrasonic beam, it is necessary to know the duty factor, which is a measure of the fraction of time during which the transducer emits ultrasound (i.e., is “on”). If the duration is 1.5 ms and the pulse repetition rate is 1,000/s, then the pulse repetition would be 1,000 μs or 1 ms. In this case, the duty factor would be 1.5 divided by 1,000 or 0.0015. This means that the transducer is transmitting only 0.15% of the time. The average power of a pulsed echocardiograph would be the peak power multiplied by the duty factor. If the peak power was 10 W and the duty factor was 0.0015, then the average power would be 0.015 W or 15 mW.

When discussing intensity in pulsed-mode systems, a common measurement is the spatial averaged, temporal averaged intensity, which is obtained by measuring the power of the transducer over the pulse repetition period and then dividing it by the surface area of the transducer. This measure, frequently quoted by manufacturers, is the lowest of the various intensities measured with a pulsed system. The spatial averaged, temporal peak intensity is a measure of average power divided by the transducer surface area that occurs when the transducer is emitting. The SP intensity is usually two to three times greater than the SA intensity. Of course, the highest measure of intensity would be the SP, temporal peak intensity, which uses peak intensity that occurs when the transducer is “on.” Commercial ultrasound instruments operating in pulsed mode for two-dimensional
imaging have SP, temporal averaged intensities ranging from 0.001 to more than 200 mW/cm$^2$. Pulsed-Doppler imaging, however, may have an SP, temporal average as high as 1,900 mW/cm$^2$, considerably greater than 100 mW/cm$^2$ level that has been most extensively studied and has never been shown to produce a biologic effect.

The biologic effects of ultrasound energy are related primarily to the production of heat (a goal of ultrasonic therapy). With pulsed ultrasound, it is extremely unlikely that the duty factor is high enough for significant heat to be generated within the body. Heat is generated whenever ultrasound energy is absorbed, and the amount of heat produced depends on the intensity of the ultrasound, the time of exposure, and the specific absorption characteristics of the tissue. It should also be noted that the flow of blood and specifically the perfusion of tissue have a dampening effect on heat generation and physically allow heat to be carried away from the point of energy transfer.

The relatively short periods of pulsing, coupled with the fact that the transducer is constantly moving so that no single area is imaged for a long period, contribute to the low likelihood of delivering significant heat to the tissue. With transesophageal imaging, however, this is not always the case. For example, during intraoperative imaging, the probe may remain nearly stationary for extended periods. The heat generated by the transducer itself must also be considered. Although there are no reports of significant injury resulting from even prolonged intraoperative transesophageal echocardiography, attention to these issues is recommended. Limited imaging time, occasional repositioning of the probe, and constant monitoring of the probe temperature will all help to ensure an impeccable safety record.

Another physical effect of ultrasound is cavitation. This term refers to the formation and behavior of gas bubbles produced when ultrasound penetrates into tissue. It is very difficult to measure or even detect the phenomenon of cavitation in vivo. Because of the relatively high viscosity of blood and soft tissue, significant cavitation is unlikely. An important aspect of cavitation concerns its effect during the injection or infusion of contrast microbubbles. It is now well established that ultrasound energy causes such microbubbles to resonate, resulting in cyclical changes in bubble diameter and stability.

A variety of other physical forces may also be produced by ultrasound energy. These include oscillatory, sheer, radiation, pressure, and microstreaming. Although each of these effects can be demonstrated in vitro,
there is no evidence that any of these physical phenomena has a significant biologic effect on patients. Despite considerable study, virtually no clinically important biologic effects attributable to ultrasound at diagnostic power levels have been demonstrated. However, a few reports have suggested that some changes might occur at the chromosomal level that would be relevant to the developing fetus. These observations have caused considerable concern within the field of fetal echocardiography. The overwhelming evidence, however, supports the relative safety of ultrasound even in this critically sensitive arena.

Research will continue in this important area. All evidence to date suggests that diagnostic ultrasound, particularly that used in echocardiography, is an extremely safe tool with no demonstrated adverse effects even with the use of newer technology and more powerful instrumentation. Although this is reassuring and justifiably inspires continued confidence in ultrasound imaging, the desire for more and better diagnostic information should never occur at the expense of patient safety. Therefore, keeping the scan time to a minimum, especially when performing Doppler imaging, should always be a consideration. It is likely that ongoing reassessment of the safety of echocardiography will continue for the foreseeable future.

Suggested Readings

**GENERAL CONCEPTS**


**Doppler Principles**


**Harmonic Imaging**


**INSTRUMENTATION**


**SAFETY AND BIOLOGIC EFFECTS**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Chapter 3
Contrast Echocardiography

Ultrasound contrast agents were first used in conjunction with clinical echocardiography in the mid-1970s. Early agents consisted of either agitated saline or agitated saline stabilized with indocyanine green dye. Injections were done either intravenously or centrally at the time of cardiac catheterization. The resultant cloud of microbubbles was used to define cardiac borders and detect shunts (Fig. 3.1). After intravenous injection, these early contrast agents were isolated to the right heart and did not traverse the pulmonary circuit. As such, their appearance in the left heart was evidence of a right-to-left shunt.

**SOURCE OF ULTRASOUND CONTRAST**

Random ultrasound contrast targets were recognized in the early days of clinical echocardiography. It was theorized that the microbubble targets were created by cavitation at the time of injection of intravenous fluid. Although it is possible to create microbubbles due to a cavitation effect, the pressure with which fluid must be injected to create a cavitation effect is well beyond that encountered in routine clinical practice. Contrast occurring spontaneously at the time of an intravenous injection is more likely due to air contamination in the injection apparatus than to creation during injection.
FIGURE 3.1. Early M-mode contrast echocardiograms recorded in the cardiac catheterization laboratory. **A:** The orientation of the M-mode ultrasound beam. **B:** Image was recorded after injection of contrast into the left atrium and shows subsequent appearance of contrast in the aorta. **C:** Contrast injected into the right ventricular outflow tract is shown. **D:** Contrast appears in the aorta after a left ventricular injection. **E:** Image was recorded after a supravalvular injection into the aorta. Contrast is seen exclusively in diastole with a contrast-free area due to competitive flow during aortic valve opening. RVO, right ventricular outflow. (Reprinted with permission from Gramiak R, Waag RC, Simon W. Cine ultrasound cardiography. *Radiology.* 1973;107(1):175–180. Copyright © 1973 by the Radiological Society of North America, Inc.)

Gas-containing microbubbles reflect ultrasound at a level several orders of magnitude greater than non–gas-containing structures. Current ultrasound agents contain a variety of gases including air or, more recently, perfluorocarbons. The increased reflectivity from a microbubble target is due to the differential reflection of the contained gas compared with surrounding blood and tissue.

**CONTRAST AGENTS**
The simplest ultrasound contrast agent consists of saline microbubbles. Effective right heart contrast can be obtained by forcefully agitating a solution of saline between two 10-mL syringes, each of which contains 5 mL of saline and 0.1 to 0.5 mL of air (Fig. 3.2). Forceful agitation through a three-way stopcock creates a population of microbubbles that are highly variable in size and have a tendency to rapidly coalesce. After generation by agitation, they should be injected immediately to limit the time available for coalescence. These crudely produced microbubbles are intense echo reflectors and can be detected in the right atrium and right ventricle (Fig. 3.3). Their size precludes passage through the pulmonary capillary bed, and appearance in the left heart implies a pathologic right-to-left shunt. By analyzing the timing and location of appearance, the nature of this shunt can often be determined as being a patent foramen ovale, atrial septal defect (ASD), or pulmonary arteriovenous malformation (AVM). Creation of ultrasound contrast by this technique is widely used in clinical practice and has an excellent safety profile.

**FIGURE 3.2.** Two-syringe and three-way stopcock apparatus for preparation of agitated saline contrast for intravenous injection. The total volume in the syringe on the left is approximately 10 mL, which consisted initially of 9.5 mL of saline and 0.5 mL of air. The contrast was prepared by forcefully injecting the solution from one syringe to the other through the three-way stopcock. Turbulence within the stopcock results in the creation of a large number of microbubbles that are
suitable for intravenous injection. For opacification of right heart structures, a typical intravenous “dose” of contrast prepared in this manner ranges from 1.0 to 10.0 mL.
FIGURE 3.3. A: Apical four-chamber view recorded in a patient after injection of saline into a left upper extremity vein. B: After injection of intravenous contrast, there is uniform opacification of the right atrium and right ventricle with no
appearance of contrast in the left heart, implying the absence of an intracardiac right-to-left shunt. 

Video 3-3

Early attempts to create a more stable population of microbubbles involved reduction of surface tension. Surface tension increases the inward pressure on a bubble and is responsible for the tendency of a microbubble to collapse on itself. This tendency to spontaneously decrease in size due to surface tension results in a progressive increase in the pressure within the microbubble, which in turn increases the driving force for the contained gas to diffuse out of the bubble. These factors lead to an acceleration in the rate at which the microbubble shrinks and eventually disappears. By reducing and stabilizing surface tension, bubbles undergo less spontaneous collapse and a population of stabilized, longer lasting microbubbles can be created. Several agents including surfactant and indocyanine green dye have been used to reduce surface tension and create a population of smaller, more stable microbubbles. Many of the early fundamental observations in contrast echo were made using indocyanine green dye–stabilized microbubbles (Fig. 3.1). For practical purposes, there is little need to stabilize saline microbubbles. Because their size is relatively large, they do not pass the pulmonary capillary bed, and the safety record of this easily prepared agent has been remarkable.

Beginning in the early 1980s, an attempt was made to engineer microbubbles that would be uniform in size, have stability with respect to coalescence and size, and provide a homogeneous and reproducible degree of contrast. Recognition that high-intensity sonication of an albumin solution
resulted in microbubbles was a major breakthrough in contrast echocardiography. The stability of the resultant contrast agent depended on the solution that was sonicated and the gas it contained. Through trial and error, it was recognized that sonication of 5% human albumin resulted in creation of a relatively homogeneous population of small microbubbles consisting of a denatured albumin shell–containing air. These microbubbles were small enough to allow transpulmonary passage, resulting in an intense contrast effect, and could be commercially prepared as a sterile solution providing a reproducible contrast effect. The major limitations of the early air-containing contrast agents were their relatively large size and inconsistent transpulmonary passage. Refinements in manufactured microbubbles have included replacement of the contained gas with a high-density perfluorocarbon instead of air and replacement of the albumin shell by a lipid membrane. A number of other approaches to the manufacture of microbubbles have also been undertaken including saccharide particles that form gas microbubbles on their surface and engineered microbubble shells of various size and composition. In general, the commercially available microbubbles have an initial size of 1.1 to 3.5 μm and are prepared at a concentration of $5 \times 10^8$ to $1.2 \times 10^{10}$ microbubbles per milliliter. As such, the number of microbubbles injected per “dose” is substantially greater than that seen with agitated saline. Because of their stability (in a low–ultrasound intensity field), they have substantial persistence, and a single injection will provide a usable contrast effect in the cardiovascular system for 3 to 10 minutes.

An engineered microbubble has two basic components, the outer shell and contained gas (Fig. 3.4). Bubble shells can be designed to be either rigid or flexible and to have varying resistance to collapse at high pressure. Recognition of these phenomena allows creation of microbubble populations that can be resistant to ultrasound destruction and, therefore, provide persistent contrast effect or can be easily destroyed by the ultrasound, resulting in simulated acoustic emission and enhanced detectability by this mechanism. The shell can be designed to allow varying degrees of permeability and outward diffusion of the contrast gas. Finally, the composition of the shell can be altered to include nonreflective therapeutic compounds. Engineered microbubbles have been developed with shells which can be imbedded with specific antigenic-binding sites, allowing them
to be targeted to specific tissues. Application of the latter technology theoretically allows delivery of chemotherapeutic or biologically active agents, including gene transfection vectors, to targeted tissue.

The gas contained within the shell also affects the intensity and duration of the effect. Because the gas–blood interface is such a potent reflector, the intensity of contrast effect is substantially greater for any of the current generation of commercially available agents than that seen with agitated saline, largely because of the greater concentration of microbubbles. As is discussed subsequently, many ultrasound techniques either purposefully or incidentally disrupt the microbubble, allowing the gas to escape into the blood pool. Gases such as oxygen, nitrogen, and room air rapidly diffuse down a concentration gradient, resulting in rapid loss of contrast effect. High-density inert perfluorocarbons diffuse more slowly and, therefore, provide a longer lasting contrast effect even after bubble-shell disruption.

**Safety of Ultrasound Contrast**

Contrast echocardiography using both agitated saline and commercially developed agents for left ventricular opacification have had an excellent safety record. The major concern regarding agitated saline is that it represents a population of microbubbles of highly variable size subject to coalescence, and that if present in the arterial circulation could result in a clinical syndrome of air embolization. Early surveillance studies suggested a remarkably safe track record for this agent and there have been only infrequent isolated case reports of neurologic or other sequelae following saline contrast injection.
Similarly, the safety profile of the commercially available agents for left ventricular opacification has been superb. There were initial concerns regarding a very small number of adverse events noted following injection of these agents. A definite cause and effect between the events and agents was never demonstrated. Subsequently several large surveillance studies demonstrated an absence of any excess adverse events when used according to manufacturer’s recommendations. This safety record was shown in patients undergoing resting transthoracic echocardiography, stress echocardiography, and in patients in intensive care units.

Multiple animal studies have demonstrated a “dose response” of both contrast concentration and ultrasound delivery mode with respect to adverse effects. These studies have demonstrated the potential for creation of isolated ventricular arrhythmias as well as evidence of cellular damage related to contrast echocardiography when performed in higher than clinically relevant doses of the contrast agent and with algorithms for delivery of ultrasound exceeding those typically used in clinical practice. These studies suggest a substantial margin for safety of the agents when used as recommended according to clinical guidelines and also suggest parameters by which both the agent and the ultrasound instrumentation can be used for potentially therapeutic responses. This is briefly discussed in the section on experimental uses of ultrasound contrast.
ULTRASOUND INTERACTION WITH CONTRAST AGENTS

Microbubbles interact with the ultrasound beam in a variety of ways including direct reflection at the fundamental transmitted frequency and resonance with creation of reflected harmonic frequencies. The frequency at which a bubble has maximal reflectance is related to bubble diameter. For any ultrasound frequency, the amplitude of reflection from a microbubble decreases as the bubble diameter decreases. All bubbles have a diameter at which reflectance is maximal (the resonant diameter). Below the resonant diameter of the bubble, the amplitude of reflection again diminishes with the cube of the diameter. It is a fortuitous occurrence that bubbles having a diameter that allows transpulmonary passage have excellent reflectance when interacting with clinically relevant transmission frequencies.

Interaction of microbubbles with the ultrasound beam has three phases (Fig. 3.5). In its simplest form, ultrasound interacts with a microbubble by pure reflection of the ultrasound beam at its fundamental (i.e., transmitted) frequency. Maximal reflection from the microbubble is dependent on the relationship of the frequency and diameter as noted previously.

At higher ultrasound imaging intensities (typically ≥0.3 MI), microbubbles are not pure reflectors but begin to resonate. A resonating bubble will reflect ultrasound not only at the fundamental insonating frequency \(f_t\) but also at harmonics of that frequency. In this instance, a microbubble insonated with a 2-MHz interrogating beam will reflect back the 2-MHz fundamental frequency but also resonate, creating reflected frequencies at 4, 8, and 16 MHz. Each of these subsequent harmonic frequencies doubles in frequency and diminishes in amplitude. In routine clinical practice, only the first harmonic (i.e., twice the fundamental frequency) is typically used for anatomic imaging. Contrast-specific imaging often relies on either multiple harmonic frequencies or subharmonics of the first harmonic (i.e., four and eight times the fundamental frequency). This provides a more contrast-specific signal.

At increasing ultrasound energy levels, the bubbles are physically destroyed by the insonating beam. The process of destruction results in the creation of subpopulations of bubbles of variable diameters. The highly
variable diameter subpopulations result in a broad range of reflected frequencies. By this destructive bubble technique, a large amount of acoustic energy is generated both as reflected ultrasound and as multiple detectable Doppler shifts. This final phenomenon in which microbubbles are destroyed, thereby creating detectable ultrasound targets, is referred to as “stimulated acoustic emission.” This phenomenon can be maximized by the use of a microbubble with a fragile shell and containing nitrogen, or another rapidly diffusing gas, resulting in a rapid loss of the contrast effect after shell disruption.

**DETECTION METHODS**

Interaction of microbubbles with ultrasound is complex and can be divided into three types of interaction: fundamental reflection, harmonic creation and detection, and stimulated acoustic emission. The receiving characteristics of the ultrasound instrument can be altered to capitalize on any of these three phenomena. Table 3.1 outlines the different ultrasound domains (e.g., B-mode vs. Doppler, etc.) and several commonly used acquisition modalities. Virtually, any of the different ultrasound domains can be linked to any of the acquisition methods to register the contrast-enhanced image. The exact combination of ultrasound domain and acquisition protocol will depend on the nature of the examination (e.g., left ventricular border vs. myocardial perfusion) as well as the characteristics of the available contrast agent and imaging platform.

**Machine Settings**

All current manufacturers provide dedicated contrast-specific presets to account for sensitivity of ultrasound contrast agents in a high-intensity field. Many of them have, as optional add-ons, contrast-specific modalities suitable for detection of low-intensity contrast in the myocardium. The users should be aware of the specific nature of the contrast presets, which are proprietary and vary from manufacturer to manufacturer and from platform to platform.
FIGURE 3.5. Schematic representation of various microbubble responses to increasing ultrasound intensity. Above the diagonal line, depicting increasing intensity, is a graphic representation of reflected image intensity at each level of ultrasound intensity and below the line a stylized depiction of the frequency response noted with each technique. At low intensity, a linear response can be obtained that results in detection of a returning frequency identical to the transmit frequency \(f_t\). At higher incident pressures, bubble resonance occurs, resulting in the generation of a nonlinear or harmonic response such that signal is returned at the fundamental transmitted frequency as well as a series of its harmonics (e.g., \(f_t\)). At higher ultrasound intensities, bubble integrity is disrupted resulting in a subpopulation of smaller bubbles with a broad range of resonant frequencies. Because bubble destruction occurs at the higher insonating pressure, the duration of contrast effect is substantially less.

The simplest method for contrast detection is routine B-mode ultrasound. As noted previously, microbubbles are intense reflectors of ultrasound and the amount of reflected energy is substantially greater than that of the surrounding tissue or blood. Because of this, routine B-mode scanning is highly sensitive for the detection of isolated microbubble targets. This routine imaging technology is sufficient for detection of intracardiac shunts such as ASD using agitated saline. When used with newer perfluorocarbon-based
agents, detection is markedly facilitated by the use of harmonic and other advanced imaging algorithms (Fig. 3.6).

<table>
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<tr>
<th>Table 3.1</th>
<th>IMAGING MODALITIES FOR CONTRAST DETECTION</th>
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<tr>
<td><strong>Ultrasound Domain</strong></td>
<td><strong>Acquisition Mode</strong></td>
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<tr>
<td>B-mode</td>
<td>Continuous</td>
</tr>
<tr>
<td>Fundamental</td>
<td>Triggered</td>
</tr>
<tr>
<td>Harmonic</td>
<td>Fixed interval</td>
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<tr>
<td>High mechanical index</td>
<td>Variable, incremental interval</td>
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<tr>
<td>Low mechanical index</td>
<td>Triggered sequential</td>
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<tr>
<td>Doppler</td>
<td>Destruction/detection image sequence</td>
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<td>Harmonic versus fundamental</td>
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<td>Frequency shift</td>
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<td>Power spectrum</td>
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<td>Correlation techniques</td>
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**Intermittent Imaging**

It was recognized in the mid-1990s that the routine interrogating ultrasound beam destroyed ultrasound targets (Figs. 3.7 and 3.8). This was a fortuitous observation made when investigators recognized the absence of contrast effect in the left ventricular cavity or myocardium during continuous imaging. After brief interruption of scanning, contrast was again detectable without reinjection of the agent. This led to the technique of intermittent imaging in which ultrasound interrogation is triggered to the electrocardiogram. In between triggered imaging, no ultrasound energy is delivered, allowing time for restitution of the contrast effect and its subsequent detection when imaging is resumed. Obviously, with intermittent imaging, the ability to analyze wall motion is lost, and this imaging technique is typically used for evaluation of myocardial perfusion. There is a direct relationship between microbubble destruction, measured as loss of contrast effect, and the intensity of delivered ultrasound (Fig. 3.8).

**Low Mechanical Index Imaging**

Mechanical index (MI) is a measure of the power of an ultrasound beam and is defined as peak negative acoustic pressure/ft, where ft, is the transmitted
frequency. Routine B-mode scanning for anatomy and function typically is undertaken at an MI of 0.9 to 1.4, which results in optimal tissue signature but substantial contrast destruction. Typically, at an MI of 1.3 and above, perfluorocarbon-based ultrasound contrast agents are rapidly destroyed in the ultrasound beam. Initially this may result in an instantaneous burst signal due to stimulated emission, the ongoing destruction of the agent results in the inability to detect any contrast effect.

**FIGURE 3.6.** Four-chamber view recorded in a patient during harmonic (A) and fundamental (B) imaging often injection of a commercial contrast agent for left ventricular opacification. Note that with harmonic imaging, there is smooth opacification of the cavity and detection of contrast in all four cardiac chambers. **Bottom:** Recorded in the same patient using fundamental rather than harmonic imaging (arrows denote imaging mode). Note the lack of contrast detection with fundamental imaging. [Video 3-6]
FIGURE 3.7. Suprasternal view of a normal aorta after intravenous injection of ultrasound contrast. The electrocardiogram is provided for timing. A: A systolic frame in which contrast is clearly identified in the arch of the aorta. B: The diastolic portion of the same cardiac cycle, in which far less contrast is detected, is shown. In the real-time image, note the phasic appearance and disappearance of the contrast in the aorta. Note that, during systole, a “fresh bolus” of contrast is ejected into the arch from an area out of the plane of imaging. During diastole, when there is less flow in the aorta, there is more time for ultrasound interaction with the contrast agent and it is progressively destroyed.
FIGURE 3.8. Apical four-chamber view recorded in a patient demonstrating the impact of mechanical index on contrast appearance. **A:** Image was recorded with a mechanical index of 0.3 and reveals smooth opacification of all four cardiac chambers. **B:** Image was recorded 10 seconds later with a mechanical index of 1.0. Note the complete lack of contrast in the near field and the swirling nature of the partial filling in the far field.

**Video 3-8a**
Having recognized that the interrogating ultrasound beam is responsible for accelerated microbubble destruction and continuous imaging results in the loss of contrast effect, algorithms for continuous imaging at a low MI have been developed. Perhaps the single most important machine parameter to consider when using the modern generation of ultrasound contrast agents is the MI which was discussed previously. By imaging at a low MI (typically <0.3), contrast within the left ventricular cavity is not destroyed, and because imaging is continuous rather than intermittent, wall motion analysis can be undertaken in real time with boundaries enhanced by the opacified left ventricular blood pool (Fig. 3.8). Low MI imaging is also necessary when detecting very low concentrations of ultrasound contrast such as for myocardial perfusion. For myocardial perfusion imaging, intermittent high-MI imaging is often undertaken to purposefully destroy contrast in the blood pool to create a repeated bolus effect from which time appearance curves can be created.

In addition to MI, there are other machine settings that have an impact on detection of ultrasound contrast. In general, anything that increases delivery of the ultrasound energy to the contrast agent results in a greater degree of destruction and consequently a decrease in the magnitude of contrast effect. As such, high frame rates will result in greater ultrasound contrast destruction than low frame rates. There can be selective destruction of contrast at the point at which a transmit focal zone has been set. Because of increased ultrasound energy at shallow imaging depths, the near field is more susceptible to contrast agent destruction than is the far field.

A more advanced method for detection of contrast is the use of phase correlation techniques, in which an automatic correlation of the insonating and reflected frequencies is undertaken. Because microbubbles are nonlinear reflectors and result in variable frequency shifts, the characteristics of reflected ultrasound from two sequential pulses will contain different reflected frequency spectra. This nonlinear response is not seen after interaction with tissue where the characteristics of two sequential ultrasound pulses will be identical. This methodology is referred to as phase image analysis. For phase image analysis two ultrasound signals are sent out with
close temporal proximity (Fig. 3.9). The second pulse is 180 degrees out of phase with the first pulse and may have a different amplitude. When the two reflected signals are then received, they are summed, and the summed ultrasound signal is then displayed. If each of these signals is reflected from a linear, nonharmonic reflector, such as tissue or blood, they are then received back at the transducer precisely 180 degrees out of phase (exactly as transmitted), and when summed, they cancel each other to create zero signal. Conversely, if the signals interact with microbubbles, each signal is shifted in phase. Additionally, because microbubbles compress and expand at different rates in the ultrasound field, the contour of the reflected signal is altered compared with the transmitted signal. When summed, cancellation no longer occurs, and a signal is preserved. In theory, this provides a highly specific methodology for the detection of ultrasound contrast.
A: A transmitted wave interacts with a linear reflector (solid bar). The received wave is identical in configuration to the transmitted wave but will have less amplitude because of attenuation. The received signal is centered on the transmit frequency ($f_t$). B: The identical frequency transmitted 180 degrees out of phase with that in A. C: The interaction of two sequential pulses each of which is 180 degrees out of phase with the other (A + B transmitted nearly simultaneously) is depicted. When received and summed, the waveform is as demonstrated and the received signal consists of identical positive and negative amplitudes that result in zero signal, as denoted by the absence of shading. D: The interaction of a transmitted wave with
a microbubble is depicted. Because microbubbles contract and expand at different rates, they alter the contour of the transmitted wave. The received waveform has components of the fundamental frequency and harmonic frequencies at two and four times the transmit frequency. It is also altered in contour as noted. E: The interaction of two closely spaced pulses, 180 degrees out of phase (identical to the transmitted pulses in C), which then interact with a microbubble is represented. Because the two pulses interact in opposite manners with the microbubble, they result in a more complex received waveform. The fundamental frequencies are returned 180 degrees out of phase, and the harmonic signals are preserved. This results in a relatively contrast-specific signal.

**CLINICAL USES OF ULTRASOUND CONTRAST**

The use of contrast echocardiography can be divided into five broad categories: (1) detection of intracardiac shunts; (2) left ventricular opacification for chamber delineation; (3) refined definition of left ventricular structural abnormalities; (4) myocardial perfusion; and (5) enhancement of Doppler signals.

**Clinical Uses of Saline Contrast**

Detection of right-to-left shunts was one of the earliest uses of contrast echocardiography and remains a use for which agitated saline remains the agent of choice because of its low cost, long safety record, and lack of need for contrast opacification of the left heart structures. When evaluating a patient for right-to-left shunt, an agent that appears in the left ventricle because of normal transpulmonary passage is not appropriate. Causes of right-to-left shunts that can be documented by intravenous injection of agitated saline include ASDs of all types, patent foramen ovale, and pulmonary AVMs. Larger ventricular septal defects may allow some right-to-left shunting during diastole when pressure in the two ventricles is relatively equal.

Intravenous injection of agitated saline contrast remains one of the primary tools for detecting a patent foramen ovale or ASD. Figures 3.10 to 3.12 were recorded in patients with ASDs. In patients with smaller defects, contrast echocardiography may provide crucial information as to the presence of a shunt that is not directly visualized or has not resulted in a right ventricular volume overload. Detection of a patent foramen ovale or ASD
depends on shunting of contrast-enhanced blood from the right atrium across
the defect into the left atrium and left ventricle.

FIGURE 3.10. Apical four-chamber view recorded in a patient with a secundum
atrial septal defect. The central image was recorded after injection of agitated
saline and demonstrates a significant right-to-left shunt which was phasic with the
respiratory cycle. Note the contrast in the left ventricle but the absence of contrast
in the more superior portion of the left atrium. The side panel at the lower left was
recorded several cardiac cycles later and reveals diminished left ventricular
contrast consistent with the phasic nature of the shunt in an atrial septal defect.
FIGURE 3.11. Apical four-chamber view recorded after saline contrast injection in a patient with a sinus venosus atrial septal defect. The central image was recorded immediately after appearance of contrast in the right atrium and shows concurrent early appearance in the left atrium, prior to its appearance in the right ventricle. This would be consistent with sinus venosus defect location in which there is overriding of the superior vena cava with immediate communication to the left atrium. The inset at the upper left was recorded several cardiac cycles later in reveals equal opacification of all four cardiac chambers.
There are two factors which impact the likelihood and magnitude of right-to-left shunting with saline contrast. The first is the actual defect size and location. Larger defects are more likely to be associated with a significant right-to-left shunt than are small defects. During inspiration, right heart pressure and filling increase. This increases the tendency for transient right-to-left shunting to occur. If left atrial pressure is consistently higher than right atrial pressure, an ASD will be associated almost exclusively with a left-to-right shunt. In these instances, evaluating the appearance of contrast in the right atrium along the atrial septum may allow detection of a negative contrast effect (Figs. 3.12A and 3.13). The negative contrast effect occurs when noncontrast-enhanced blood from the left atrium flows across the ASD into the right atrium, displacing contrast-enhanced blood. Depending on the location this may be confused for normal inferior vena caval flow.

The timing of contrast appearance in the left atrium can be highly variable and as noted above is linked to the respiratory cycle. In primum and secundum defects there may be a several cardiac cycle delays in left atrial appearance depending on the phase of respiration at which contrast enters the heart. In patients with sinus venosus ASD, appearance of contrast may be nearly simultaneous in the right and left atrium and may be less dependent on the respiratory cycle. This is due to the functional overriding of the superior vena cava and left superior pulmonary vein as it enters the right atrium (Fig. 3.11).

A patent foramen ovale can be reliably detected with contrast
echocardiography using agitated saline (Figs. 3.14 to 3.16). A patent foramen ovale represents an unsealed overlap of the foraminal tissue with the more basal portion of the atrial septum. Variations of patent foramen include small fenestrations, which may be multiple. Atrial septal aneurysms are often associated with one or more small perforations (Fig. 3.16). Because left atrial pressure typically exceeds right atrial pressure, only a small and hemodynamically inconsequential left-to-right shunt is typically present in patients with patent foramen ovale. The magnitude of this shunt is below that which can be documented with oximetry or dye dilution techniques. Additionally, the shunt is often phasic with the respiratory cycle and limited to early inspiration. Maneuvers such as Valsalva and cough, which transiently increase right heart pressure, may allow the occult right-to-left shunt of a patent foramen ovale to become manifest with contrast echocardiography. Patients are best evaluated for a patent foramen ovale in the apical four-chamber or subcostal view with one or more contrast injections performed during quiet respiration, cough, and Valsalva. Using this fairly vigorous approach, approximately 25% of individuals with otherwise structurally normal hearts may be demonstrated to have trivial degrees of right-to-left shunting through a patent foramen ovale. Clinical studies suggest that only patients with a larger patent foramen ovale, that is, those with more substantial right-to-left shunting, are at risk of cardioembolic disease.

Figure 3.12. Saline contrast echocardiogram recorded in a patient with a secundum atrial septal defect. A: Four-chamber view with saline contrast recorded during quiet respiration. Note the opacification of the right atrium and right ventricle and the negative contrast jet from the left atrium to the right atrium (arrow). During quiet respiration there was no evidence of concurrent right-to-left shunt. The inset at the upper left is a transesophageal echocardiogram recorded in the same patient documenting the presence of an approximately 1-cm
secundum atrial septal defect (arrows). B: Apical four-chamber view recorded in the same patient presented in (A) after a repeat saline injection but during vigorous coughing. Notice with the provocation of cough there is now a definite right-to-left shunt with contrast targets in the left atrium and ventricle (arrows). Even with provocation the magnitude of the shunt appears small in spite of a documented atrial septal defect diameter of 1 cm.

Video 3-12

As noted above, detection of a right-to-left shunt implies transient or persistent elevation of right atrial over left atrial pressure. Only a minimal increase in right atrial pressure compared to left atrial pressure is required for a small right-to-left shunt to occur. On occasion, one encounters a patient in whom shunting is transient and related to volume status or body position. Figure 3.17 was recorded in a patient with a definite right-to-left shunt under basal conditions but an exclusive left-to-right shunt at the time of transesophageal echocardiography, presumably due to mild volume depletion.
FIGURE 3.13. Transesophageal echocardiogram recorded in a longitudinal view of the atrial septum. Agitated saline has been injected into an upper extremity vein and has completely filled the right atrium. Note a small number of individual contrast targets in the left atrium consistent with a limited right-to-left shunt. Also note the negative contrast effect (arrow) arising from the atrial septum and projecting into the contrast-enhanced right atrium. This effect occurs due to flow of noncontrast-enhanced blood from the left atrium through a small (4 mm) secundum atrial septal defect into the contrast-filled right atrium. 

coming soon
Video 3-13

On occasion, it may be difficult to distinguish shunting from a patent foramen from that due to an ASD. In general, an ASD will be associated with enlargement of right heart chambers and, if associated with a significant shunt, with abnormal septal motion. Typically the shunt magnitude is greater with an ASD than a patent foramen ovale. If a patent foramen ovale is associated with significant right heart disease or pulmonary hypertension the magnitude of right-to-left shunting can be substantial and functionally equivalent to an ASD. Occasionally, even a large ASD may be associated with minimal right-to-left shunting. This occurs in the setting of a large left-to-right shunt which overwhelms the likelihood of contrast-enhanced blood traversing the defect. In general, these defects will be unmasked by maneuvers such as a cough or Valsalva. The apparent magnitude of the right-to-left shunting however may understate the anatomical size of the defect (Fig. 3.12). The right-to-left shunt of a large ASD may be nearly continuous (Fig. 3.11). For smaller ASDs, or those with predominant left-to-right shunt, the appearance of contrast in the left atrium may be phasic, coordinated with the respiratory cycle, or provoked only by cough or Valsalva (Fig. 3.12).
FIGURE 3.14. Apical four-chamber view recorded in a young patient with a neurologic event. Saline contrast has been injected to evaluate the possibility of a patent foramen ovale. The presented central image was recorded approximately five cycles after appearance in the right atrium and shows definite phasic appearance of contrast in the left atrium and left ventricle consistent with an atrial level right-to-left shunt. In view of the absence of right heart enlargement or right ventricular volume overload the most likely diagnosis is patent foramen ovale. The inset at the lower right was recorded immediately after appearance of contrast in the right atrium and right ventricle, at which time there was no contrast on the left-sided chambers.
FIGURE 3.15. Real-time three-dimensional transesophageal echocardiogram of the atrial septum in a patient with a patent foramen ovale and a right-to-left shunt on saline contrast injection (B). A: Note the mobility of the foraminal tissue (arrows) in the real-time image.
A final type of right-to-left shunt that can be detected by contrast echocardiography is a pulmonary AVM. This can be seen in the presence of end-stage liver disease but also occurs as part of several medical syndromes. The classic contrast echocardiographic appearance of an AVM is that of a delayed right-to-left shunt in which contrast appears in the left atrium after a delay of 5 to 15 cardiac cycles (Fig. 3.18). This typically represents the time required for a transit of the contrast agent through the pulmonary arterial bed and the AVM and into the pulmonary veins. As the exact magnitude of delay is related to transpulmonary flow, patients with high cardiac output, as typified by the patient with end-stage liver disease, may have more rapid appearance of contrast in the left heart, superficially mimicking an atrial level
shunt. Other characteristics of a pulmonary AVM include the tendency of the contrast to build up persistently and slowly over time in the left heart and the lack of phasic appearance of contrast in the left atrium, which would be more characteristic of an atrial level shunt. This gradual nonphasic appearance is a more specific marker for a pulmonary arteriovenous malformation than is any predefined time delay. In the presence of larger or multiple AVMs, the magnitude of the right-to-left shunt can be substantial and may be associated with hypoxia. In these larger shunts, it is common to see contrast intensity continue to build in the left atrium and left ventricle at a time when it is diminishing in the right heart. This pattern of contrast appearance is virtually pathognomonic of an AVM. Finally, direct inspection of pulmonary veins can often identify contrast in the pulmonary veins and thereby establish the diagnosis.

**Detection of Miscellaneous Conditions**

Occasionally contrast echocardiography, typically using intravenous agitated saline, is useful for delineating abnormal extracardiac communications. Injection of agitated saline into a lower extremity vein in an individual withazygos continuation of the inferior vena cava allows detection of the contrast in the more superior portion of the right atrium, confirming the presence of this congenital anomaly. A more common scenario is to identify a patient with a dilated coronary sinus, typically best visualized in a parasternal long-axis view. The differential diagnosis of a dilated coronary sinus includes chronic elevation of right heart pressure due to chronic volume or pressure overload and persistence of a left superior vena cava with drainage directly into the coronary sinus. The later anomaly can be documented by injecting agitated saline into a left upper extremity vein resulting in opacification of the dilated coronary sinus before draining into the right atrium (Figs. 3.19 and 3.20). Injection into a right upper extremity vein will not opacify the coronary sinus.
FIGURE 3.16. Apical four-chamber view recorded after intravenous injection of agitated saline in a patient with an atrial septal aneurysm. A: In the central image, note the complete opacification of the right atrium and right ventricle and the bulging of the atrial septal aneurysm (arrow) into the right atrium. There is no evidence of right-to-left shunting. The inset at the upper right was recorded without contrast and depicts right-to-left bowing of the atrial septal aneurysm. B: Image was recorded later and demonstrates a small amount of contrast in the left atrium and ventricle (arrows) consistent with an associated patent foramen ovale.
FIGURE 3.17. Transthoracic and transesophageal echocardiogram recorded in the same patient on two separate days. A: The upper panel was recorded under basal, nonfasting conditions and reveals a definite right-to-left shunt. B, C: The lower panels (transesophageal study) were recorded after a 16-hour fast. Note the persistent left-to-right shunt on color flow Doppler and absence of any right-to-left shunt on saline contrast injection.

Video 3-17a

coming soon

Video 3-17b

coming soon
FIGURE 3.18. Apical four-chamber view recorded over a prolonged imaging period in a patient with a large pulmonary venous malformation after injection of saline contrast into an upper extremity vein. The upper left panel was recorded immediately after filling of the right atrium and right ventricle. Note the absence of any contrast in the left chambers. The upper right panel was recorded approximately 5 seconds later and shows faint but rather homogenous and nonphasic filling of the left ventricle. The lower left panel was recorded
approximately 20 seconds after injection and reveals equivalent filling of both the right and the left ventricles with saline contrast. Also note the contrast in the pulmonary veins. The lower right panel was recorded 40 seconds after injection at which point contrast has cleared from the right heart but has persisted in the left heart courtesy of the flow from the pulmonary vascular reservoir. Also, again, note the contrast effect in the pulmonary vein (arrows).

Artifacts and Pitfalls

Normal quiet respiration results in phasic variation of right atrial pressure. Typically, a phasic shunt associated with either a patent foramen ovale or ASD will be augmented in the cardiac cycles in early inspiration and may be absent in expiration. Depending on the relative heart rate and respiratory rate this may result in an apparent delayed appearance of contrast in the left atrium. For this reason, relying only on the delay in appearance of shunting to diagnose a pulmonary AVM may result in an erroneous diagnosis if fortuitous timing results in an inspiration-related shunt occurring multiple cardiac cycles after appearance of contrast in the right atrium.

Blood flow–related artifacts include competitive flow and marginated flow. Because contrast is contained within the bloodstream, its appearance will parallel that of the blood flow. If there is competing flow from another vessel that is not contrast enhanced, a negative contrast effect will occur. Typically, superior vena caval flow (assuming an arm injection) enters the right atrium as a bolus that merges with the noncontrast-enhanced flow from the inferior vena cava. The noncontrast IVC blood creates an area devoid of
saline contrast that can be confused for a negative contrast effect related to an ASD (Fig. 3.21). This creates a swirling matrix of contrast and nonenhanced blood, which is often maximal along the interatrial septum. This effect may be accentuated in a high-flow state in which there is greater than usual inferior vena cava flow such as is seen in chronic hepatic disease or pregnancy. On occasion, this effect has been confused with a pathologic shunt at the atrial level.

A prominent eustachian valve may result in a negative contrast effect within the right atrium, leading to the false impression that a defect with left-to-right shunting is present (Figs. 3.22 and 3.23). Because the eustachian valve marginates flow in the right atrium, it may result in only inferior vena cava flow (i.e., not contrast enhanced if the injection is into an upper extremity view) coming in contact with the atrial septum in the area of an ASD or patent foramen ovale. This may result in a false-negative evaluation for right-to-left shunting. When in doubt, injection of a contrast agent into a lower extremity vein will circumvent this problem.
In the parasternal long-axis view, note the dilated circular structure bordered by the mitral annulus and left atrium. This structure has a relatively thin wall and represents dilated coronary sinus. B: Image was recorded after injection of agitated saline into a left upper extremity vein and reveals prompt opacification of this structure before the appearance in the right ventricle, confirming that it represents a persistent left superior vena cava connecting directly to the coronary sinus. In the real-time image, note the early appearance of contrast in the right ventricle as well. CS, coronary sinus.
FIGURE 3.20. Apical four-chamber view recorded in a patient with a persistent left superior vena cava to coronary sinus communication. The central image was recorded without contrast. Note the oval-shaped echo-free space in the lateral atrioventricular groove representing a dilated coronary sinus (arrows). The inset at the upper right was recorded after injection of intravenous saline into the left upper extremity and demonstrates opacification of the coronary sinus. The inset at the upper left was recorded several cardiac cycles later and demonstrates opacification of the right side of the heart without contrast in the left atrium or left ventricle.
FIGURE 3.21. Apical four-chamber view recorded in a patient after injection of agitated saline into an upper extremity vein. Note the area of absent contrast effect (large arrow) along the most superior portion of the atrial septum, which is due to competitive flow from noncontrast-enhanced inferior vena cava blood flow. Such an area of absent contrast could be confused with a true negative contrast effect due to an atrial septal defect. This position of the atrial septum is noted by the smaller arrow.
DETECTION AND UTILIZATION OF LEFT VENTRICULAR CONTRAST

Detection of contrast in the cardiac chambers was the first clinical use of ultrasound contrast (Fig. 3.1). It remains a valuable adjunct to the clinical examination for detection of shunts and more recently for enhanced visualization of left ventricular wall motion. Perfluorocarbon-based microbubbles easily pass through the pulmonary circulation in quantities sufficient to fully opacify the left ventricular cavity. As noted previously, scrupulous attention to machine settings and technique is necessary to optimize contrast visualization for left ventricular opacification. Numerous studies have demonstrated the enhanced visualization of the left ventricular endocardial border after intravenous contrast injection and the ability to “salvage” echocardiograms that otherwise may have been suboptimal for diagnostic purposes. When compared to a standard such as magnetic resonance imaging, left ventricular contrast has been shown to improve accuracy and reproducibility for determining left ventricular volumes, ejection fraction, and regional wall motion analysis. In general, intravenous contrast is recommended for enhanced definition of left ventricular walls and wall motion when two or more segments are not visualized.

When used with appropriate attention to detail, intravenous contrast for left ventricular opacification is a valuable adjunct to the clinical echocardiographic examination. Current recommendations are that it be used
when 2 or more of 16 segments are not ideally visualized. Numerous studies have demonstrated that use of contrast in the setting of a suboptimal transthoracic echocardiogram “salvages” an otherwise nondiagnostic study, increases accuracy for determination of ventricular volumes and ejection fraction and allows accurate detection of otherwise nonrecognized functional abnormalities (Figs. 3.24 to 3.26). This advantage of left ventricular opacification has been demonstrated in the ambulatory outpatient setting, in general inpatient populations, and in the intensive care unit where imaging is often problematic. Addition of left ventricular contrast in suboptimal studies has been demonstrated to have a significant impact regarding decisions regarding anticoagulation and the need for pressors. In a similar fashion, ventricular opacification with intravenous contrast has been demonstrated to enhance the accuracy of wall motion analysis during stress echocardiography (Fig. 3.27). Selective use of intravenous contrast for left ventricular opacification should be considered part of the standard of care in the modern echocardiography laboratory.
FIGURE 3.22. Apical four-chamber view recorded in a patient with a vague linear echo traversing the right atrium (arrows). This represents a complete eustachian valve which effectively subdivides the right atrium into two segments (upper panel). After injection of saline contrast, (lower panel) note that the bulk of the contrast is confined to the less superior aspect of the right atrium with an echo-free area in the area adjacent to the inferior vena cava (arrow). In this example, note the small number of contrast targets in the left ventricle consistent with a right-to-left shunt, the magnitude of which cannot be accurately determined from this study because of the margination of contrast-enhanced blood away from the atrial septum.

Selection of patients for left ventricular opacification should be based on the need for incremental information. When the endocardial border is completely visualized, there is little incremental yield from the use of left
ventricular contrast agents. Similarly, if the echocardiogram is technically limited to the point of nonvisualization of any of the cardiac structures, it is unlikely that an intravenous contrast agent will provide a fully diagnostic image. The maximal yield of contrast for left ventricular opacification appears to be in individuals in whom 20% to 60% of the endocardial border is suboptimally visualized at baseline.
FIGURE 3.23. Transesophageal echocardiogram demonstrating a prominent eustachian valve and margination of contrast-enhanced blood flow. **A:** Image was recorded before injection of contrast into an upper extremity vein. Note the prominent eustachian valve adjacent to the inferior vena cava (arrow). **B:** Image was recorded after injection of contrast agent into an upper extremity vein. Note the appearance of contrast in the superior vena cava and the main portion of the right atrium but the absence of contrast in the area delineated by the eustachian valve. This absence of contrast could be confused with a negative contrast effect due to an atrial septal defect.

Video 3-23a

coming soon

Video 3-23b

coming soon
FIGURE 3.24. Example of left ventricular opacification after intravenous injection of a perfluorocarbon-based contrast agent. **Top left:** A baseline apical four-chamber view. Note the poor visualization of the apex and lateral wall. The other three panels were recorded after intravenous injection of a perfluorocarbon-based contrast agent. Note the excellent delineation of the left ventricular cavity and the ability to fully identify the apex and lateral walls. Video 3-24
FIGURE 3.25. Transthoracic echocardiogram recorded in an obese patient undergoing mechanical ventilation in the surgical intensive care unit. A: Apical four-chamber view from which no accurate visualization of cardiac chambers or assessment of ventricular function can be made. Note the approximate 5 cm distance between the transducer and the apparent left ventricular apex. B was recorded after injection of intravenous contrast for left ventricular opacification. While not ideally visualized the left ventricle is demonstrated to be dilated and globally hypokinetic confirming the presence of significant left ventricular systolic dysfunction which otherwise could not have been diagnosed from the echocardiogram.
FIGURE 3.26. Parasternal long-axis echocardiogram recorded in a morbidly obese patient in an intensive care unit undergoing mechanical ventilation. A: Image was recorded before injection of a perfluorocarbon-based contrast agent. Even in the real-time image, it is difficult to identify any cardiac structures. B: Image was recorded after intravenous injection of a perfluorocarbon-based agent, also in a parasternal long-axis view. Note the excellent opacification of the right ventricular outflow tract and left ventricular cavity. In the real-time image, note the normal left ventricular size and systolic function.

In addition to assessment of left ventricular size and function, left ventricular
contrast can be used for a number of other less common purposes, including detection or exclusion of intracavitary thrombus or tumor, identification of unusual entities such as ventricular noncompaction, diagnosis of atypical forms of hypertrophic cardiomyopathy, specifically the apical variant, and detection of abnormal communication to the ventricular chamber.

**FIGURE 3.27.** Demonstration of left ventricular contrast during a dobutamine stress echocardiogram. In this quad screen format, the lower right image was recorded during recovery when the majority of contrast is no longer present. Notice the suboptimal image quality with adequate visualization only of the proximal ventricular septum. The other three images were recorded during different phases of dobutamine stress at which point endocardial borders are well defined and left ventricular function can be accurately assessed.
FIGURE 3.28. Apical view recorded in a patient with a vague echo density on noncontrast imaging. After intravenous injection of a perfluorocarbon-based agent, a distinct spherical filling defect is noted in the apex, consistent with a pedunculated apical thrombus (arrows).
In marginal quality studies, one occasionally encounters an apparently hypokinetic or akinetic apex with vague, ill-defined echoes that may suggest the presence of an apical thrombus. Use of high-frequency, short-focus transducers or color B-mode imaging can occasionally resolve the issue. An additional mechanism for confirming the presence or absence of left ventricular thrombus is to use contrast for left ventricular opacification. Once completely and homogeneously opacified, the true boundary of the left ventricle can be identified and a thrombus, if present, will appear as a filling defect (Fig. 3.28). Similarly, if there is complete filling of the ventricular apex, the source of the vague echo density is likely to be artifact (Fig. 3.29).
FIGURE 3.29. Apical long-axis view recorded in a patient with an ischemic cardiomyopathy and left ventricular systolic dysfunction. In the central image note the dilated hypokinetic left ventricle and the vague echo density at the left ventricular apex (arrows). The inset at the upper left was recorded after injection of intravenous contrast for left ventricular opacification in the same view. Note the complete homogeneous filling of the left ventricular apex by contrast in this equivalent view which confirms that the vague echo noted at the apex is an artifact and not thrombus. Video 3-29a
An additional entity that results in vague, confusing echoes in the left ventricle is myocardial noncompaction. This is a form of congenital cardiomyopathy in which the embryologic myocardium, which is naturally filled with sinusoidal spaces, does not “compact” into normally structured myocardium. This results in a network of sinusoids within the ventricular myocardium and is associated with a dilated cardiomyopathy. With routine two-dimensional scanning, one encounters vague, irregular thickening of the apical and lateral walls, although the distribution of noncompaction can be highly variable. The differential diagnosis of the echo appearance is that of complex thrombi versus ventricular noncompaction. Injection of intravenous contrast and opacification of the left ventricular cavity will allow identification of the multiple sinusoidal cavities within the apparently “spongy” myocardium, confirming the diagnosis of myocardial noncompaction (Fig. 3.30).

The apical variant of hypertrophic cardiomyopathy may be missed on routine two-dimensional scanning. Because the hypertrophied muscle is of relatively low density and by definition in the near field when the heart is examined from an apical transducer position and the endothelial boundary is parallel to the ultrasound beam, the true thickness of the myocardium may not be appreciated. The ultrasound beam, especially if using low-frequency transducers, may “burn through” the apical myocardium leading to the impression that the epicardial boundary is the endocardial border. As with suspected thrombus, use of high-frequency, short-focal length transducers or
B-mode color scanning may resolve this issue, as will scrupulous attention to technical detail. Left ventricular opacification with contrast is a very effective mechanism for identifying the true endocardial boundary in this situation and may allow confident establishment of this diagnosis in otherwise confusing instances (Fig. 3.31).
FIGURE 3.30. Apical four-chamber view recorded in a patient with ventricular noncompaction. A: Without contrast, note the irregular thickening of the apex and lateral wall (arrow). After injection of a contrast agent for left ventricular opacification (B), note the contrast in the multiple sinusoids in the apex and lateral wall (arrows).
FIGURE 3.31. Apical four-chamber view recorded in a patient with an apical variant of hypertrophic cardiomyopathy. A: Recorded with standard B-mode imaging, from which pathologic thickening of the apex is not appreciable. After injection of contrast for left ventricle opacification (B), the pathologic thickness of the apical left ventricular walls (double-headed arrows) can be appreciated.
Ventricular pseudoaneurysms and small isolated apical aneurysms occasionally can be difficult to visualize with respect to the orientation of the communication to the left ventricular cavity, and on occasion one identifies an extracardiac space for which it is unclear whether there is a communication between the space and the left ventricular cavity. Occasionally, the issue can be resolved with color Doppler flow imaging. Use of contrast for left ventricular endocardial detection can also be helpful in this situation (Fig. 3.32).

Occasionally, either in the presence of normal or abnormal left ventricular systolic function, one encounters confusing echoes in the apex of the left ventricle. The differential diagnosis often includes thrombus versus muscle
trabeculae. Intravenous contrast for left ventricular opacification can be diagnostic in this situation. Thrombi typically are characterized by a definite filling defect within the cavity of the left ventricle while trabeculae will present as muscle bridges with contrast between the outer wall of the left ventricular cavity and the trabeculae (Fig. 3.33).

Similar confusion can occur when evaluating the right ventricular apex, especially in situations in which right ventricular hypertrophy is present such as in right ventricular hypertension or congenital heart disease. In this instance one may encounter a dilated right ventricle with a mass of echoes which appears to obliterate the apex. Use of contrast either saline contrast or a manufactured contrast agent can be used to assist in differentiating hypertrophied muscle in the right ventricle apex from an obliterative thrombus (Fig. 3.34).

**FIGURE 3.32.** Off-axis apical view recorded in a patient with a nonobstructive apical hypertrophic cardiomyopathy. The central image was recorded after injection of intravenous contrast for left ventricular opacification. Note the marked hypertrophy of the ventricular septum (*double-headed arrow*) with abrupt thinning toward the apex and the discrete apical outpouching consistent with an apical aneurysm (*arrows*). The *shorter inward-facing arrows* denote the boundary of the
hypertrophied left ventricular cavity in systole. The inset at the lower left is the baseline image without contrast in which neither the ventricular hypertrophy nor aneurysm are as well documented.

Video 3-32 LVO

Video 3-32

Intracardiac tumors are infrequently encountered but may also pose difficulty in diagnosis in less than ideal quality studies. Use of contrast for left ventricular opacification can help define the boundaries of an intracardiac mass, and if noted to be pedunculated the diagnosis is somewhat more likely to be tumor than thrombus, especially in the presence of normal ventricular function (Fig. 3.35). Other miscellaneous and rare entities for which intravenous contrast may be helpful is in the diagnosis of a valvular blood cyst (Fig. 3.36).
On occasion intravenous contrast may be useful in evaluating the left atrial appendage. Figure 3.37 was recorded in a patient in whom the left atrial appendage was being evaluated for possible thrombus. Note the confusing anatomy as discussed in the legend. In this instance use of intravenous contrast for opacification of the left atrium allows identification of the confusing mass as a side lobe of the left atrial appendage within the oblique sinus.

**FIGURE 3.33.** Apical four-chamber view recorded in a patient with an ischemic cardiomyopathy and an anteroapical myocardial infarction. In the central image note the vague complex echo densities at the left ventricular apex potentially representing thrombus (*arrows*). The inset at the upper left is the contrast-enhanced view. Note the complete opacification of the left ventricle and the bridging myocardial muscle bundles (*black arrows*) with contrast clearly between the true apex and the margin of the muscle bundle.
FIGURE 3.34. Apical view recorded in a young patient with L-transposition of the great arteries. A: Note the dilated anatomical right ventricle which is markedly hypertrophied related to systemic pressure. Note that the distal half of the right
ventricle appears obliterated (arrows). The etiology of the obliteration represents either marked hypertrophy of muscle bundles or possibly thrombus. B: Recorded after injection of intravenous contrast for opacification and nicely demonstrates continuity of the right ventricular cavity with the apparently obliterated apex (arrows), confirming that the mass of echoes represents marked hypertrophied trabeculae at the right ventricular apex rather than thrombus. 

Video 3-34a

Video 3-34b
FIGURE 3.35. Apical four-chamber view recorded in a patient with metastatic malignancy and a neurologic event. In the central image note the hint of an apical mass (arrow). The inset at the upper left was recorded following injection of contrast for ventricular opacification in which a pedunculated mobile mass can clearly be identified in the left ventricular apex (arrows).
FIGURE 3.36. Transesophageal echocardiogram recorded in a young patient prior to an electrophysiologic procedure. Incidental note was made of a small cystic mass on the tip of the anterior mitral valve leaflet (arrows in the central illustration). The inset at the upper right is an expanded view of the same area after injection of intravenous contrast and demonstrates that the mass is cystic and not in continuity with the blood pool, making the diagnosis of mitral valve blood cyst most likely (arrows).
Enhancement of Doppler Signals

The interaction of ultrasound with contrast agents results in a substantially higher magnitude of Doppler signal than interaction with red blood cells or tissue structures. It is assumed that the frequency shift itself remains stable as a microbubble is insonated and that it is only the intensity (power or energy) of the reflected signal that is increased. Therefore, the frequency shift and calculated velocities will accurately reflect the physiologic state; however, the intensity of the signal will increase dramatically. Low concentrations of contrast agents can be used to intensify Doppler signal strength in instances in which there is a suboptimal spectral signal. Excessive contrast effect will
result in substantial noise in the signal and may be counterproductive. The first use of this was on the right side of the heart for enhancing the tricuspid regurgitation jet (Fig. 3.38). Caution is advised when using intravenous saline to enhance the tricuspid regurgitation jet. Ideally the intensity of the contrast should be such that a normal-appearing spectral profile is preserved from which an accurate peak velocity can be ascertained. Not infrequently the presence of contrast results in a markedly distorted flow profile with multiple instantaneous “spikes” which may have erroneously high velocities which do not represent the velocity of the actual tricuspid regurgitation jet (Fig. 3.39). When this type of distorted spectral signal is noted, no inference regarding jet velocity should be made. The new transpulmonary agents can provide a similar degree of enhancement for pulmonary vein flow (Fig. 3.40) or for increasing the spectral image intensity of a relatively weak aortic stenosis jet. The operator should be cautioned that excess or even unusual gain settings will result in regurgitation of erroneous, excessively high-velocity signals, and increased noise in general.

FIGURE 3.37. Transesophageal echocardiogram recorded in a patient being evaluated for possible left atrial thrombus prior to catheter ablation for atrial
fibrillation. Note the complex anatomy of the left atrial appendage with what appears to be a small mass (small arrows) confined within an additional space bordered by the large arrows. The inset at the lower left was recorded after injection of intravenous contrast which fully opacifies the left atrium and left atrial appendage. Note that the space surrounding the apparent mass is devoid of contrast (arrows) suggesting that it represents fluid in the oblique sinus and that the “mass” is actually a side lobe of the left atrial appendage.

Video 3-37a

Video 3-37b
FIGURE 3.38. Contrast enhancement of a faint tricuspid regurgitation jet by agitated saline injected into an upper extremity vein. A: In the spectral images, note the faint tricuspid regurgitation signal from which it is not possible to ascertain the complete spectral profile or maximal velocity. B: The spectral profiles were recorded after enhancement of the jet with agitated saline. Note the substantially more robust signal and the ability to identify the maximal velocity with confidence.

FIGURE 3.39. Example of erroneous Doppler signals which are seen following injection of saline contrast. Orientation of the Doppler beam is as noted in the four-chamber view at the top of the figure. Note the numerous high-velocity
instantaneous “spikes” occurring in systole with velocities of 5 m/s and greater. These clearly do not represent a true tricuspid regurgitation jet velocity in this patient who had no evidence of pulmonary hypertension. The inset at the upper right is the noncontrast-enhanced Doppler signal in the same view.

FIGURE 3.40. Example of enhancement of pulmonary vein spectral Doppler imaging with intravenous contrast. The spectral signals (A) were recorded from an apical view. Note the very poorly defined pulmonary vein inflow signal. B: Image recorded in the same patient after injection of a perfluorocarbon-based intravenous agent demonstrates marked enhancement of the spectral signal of pulmonary vein flow. Note that both the systolic (S) and the diastolic (D) antegrade flows as well as the retrograde A-wave flow (A) are clearly seen after contrast enhancement.

Because the ultrasound contrast agent interacts with all forms of Doppler imaging, caution should be exercised when color flow imaging is employed. The addition of even very low concentrations of ultrasound contrast to the blood pool results in a substantially greater color flow area than would be
recorded without contrast (Fig. 3.41). Because the color flow jet area is used to estimate regurgitation severity, the increase in jet area caused by interaction with contrast will result in systematic overestimation of regurgitation severity. As such, contrast agents should not be used in conjunction with color Doppler in clinical practice.

**Contrast Artifacts**

Successful use of ultrasound contrast requires careful attention to technical detail and the use of machine settings that often differ from those used for routine clinical scanning. Even with meticulous attention to detail, there are a number of pitfalls and artifacts that can diminish the clinical yield of contrast echocardiography. Contrast artifacts can be divided into two broad categories: those due to the agent and its interaction with the ultrasound beam, and physiologic artifacts, both of which may interfere with interpretation (Table 3.2).

As contrast agents are very potent reflectors of ultrasound, their presence in high concentration results in nearly complete attenuation of ultrasound penetration. This phenomenon is particularly prominent when using the newer, more highly reflective perfluorocarbon-based agents. Attenuation occurs when there is an abnormally high concentration of ultrasound targets in the near field, beyond which the ultrasound beam cannot penetrate (Figs. 3.42 and 3.43). This results in detection only of the initial layer of contrast-enhanced blood, with all areas of the heart behind this area being shadowed. Attenuation is common during bolus injections of perfluorocarbon-based agents. It can be avoided by delaying scanning until later in the infusion protocol, after the peak contrast concentration has declined, or preferably by the use of a smaller bolus or lower concentration of the ultrasound agent. Clinically, the attenuation phenomenon is most problematic when imaging the basal lateral wall in an apical four-chamber view. This region is often an area of contrast dropout which should not be confused for the ventricular boundary, either for wall motion analysis or for volumetric determination. Similarly, this area of greatest attenuation can be remarkably problematic for assessing myocardial perfusion.
FIGURE 3.41. Apical four-chamber view recorded in a patient with mild tricuspid regurgitation before (A) and after (B) injection of a perfluorocarbon-based contrast agent. **A:** Note the relatively disorganized tricuspid regurgitation jet consistent with mild regurgitation. **B:** Note the dramatic increase in the size and intensity of the color flow signal jet when intracavitary contrast is present. [Video]

### Table 3.2

<table>
<thead>
<tr>
<th>CONTRAST ARTIFACTS</th>
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<tbody>
<tr>
<td>Agent/ultrasound related</td>
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<tr>
<td>Attenuation</td>
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<tr>
<td>Shadowing</td>
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<tr>
<td>Apical destruction</td>
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Physiologic
Competitive flow
SVC–IVC
Margined flow
Incomplete blood pool mixing
Eustachian valve

FIGURE 3.42. Example of an attenuation effect following injection of intravenous contrast. The central image is an apical four-chamber view recorded shortly after appearance of contrast in the left ventricle. Note the dense contrast effect at the apex of the left ventricle (downward-pointing arrows) and the absence of contrast and anatomical definition below the contrast (downward-pointing arrows). This shadowing is related to excess contrast effect and reflection of ultrasound prior to penetration into the left ventricle itself. The inset at the lower right is the same patient recorded 10 seconds following injection after dilution of the contrast in the blood pool, in which smooth homogeneous opacification of left ventricular cavity (arrows) is now apparent with definition of more distal structures.
Another well-recognized artifact is that created by shadowing from a papillary muscle when imaging in the four-chamber view. The shadow created at the proximal boundary of the contrast with the papillary muscle extends toward the left atrium in a straight line. This shadow can be confused with the lateral endocardial border (Fig. 3.44).

Shadowing from a papillary muscle is not the only source of an artifactual, contrast-free region within the left ventricular cavity. If a patient has areas of dense fibrosis or calcification between the transducer and the blood pool, a shadow will occur behind the echoreflective focal area mimicking a contrast-free area. Figure 3.45 was recorded in a patient with a chronic apical aneurysm with areas of intramural calcification. Note the two separate areas
of apparent lack of contrast effect that radiate down from the apex into the cavity of the blood pool. These are the result of shadowing from calcific deposits in an apical aneurysm.

Because the contrast agent interacts with ultrasound, irrespective of the analysis mode, it has a profound impact on the appearance and validity of Doppler signals (Fig. 3.41). For this reason, if use of a contrast is anticipated, the operator should collect all required color Doppler images before using intravenous contrast. Imaging of even limited amounts of contrast markedly distorts the color Doppler signal and results in erroneous registration of data.

FIGURE 3.43. Parasternal long-axis view recorded immediately after injection of ultrasound contrast. Note the significant attenuation of ultrasound signal behind the dense bolus of contrast in the right ventricular outflow tract, which precludes visualization of any posterior structures.
coming soon

Video 3-43
FIGURE 3.44. Apical four-chamber view demonstrates a papillary muscle shadow. A: Image was recorded in diastole. Note the location of the papillary muscle (black arrows) and the faint shadow behind it. Also note the true location and thickness of the lateral wall (white arrows). B: Image was recorded in systole and demonstrates a more exaggerated papillary muscle shadow. Mistaking the papillary muscle shadow for the lateral wall will result in dramatic underestimation of the size of the left ventricle. PAP, papillary muscle.

Video 3-44
Detection and quantitation of myocardial perfusion have been a goal of echocardiography since the ability to opacify the myocardium was first recognized in the 1980s. Early animal laboratory work confirmed that contrast distribution paralleled myocardial blood flow and confirmed that the absence of contrast accurately reflected the ultimate size of a myocardial infarction in animal models and in patients (Figs. 3.46 and 3.47). Subsequent work demonstrated that the newer contrast agents could be used to identify coronary collateral circulation and that a preserved contrast effect in the myocardium was evidence of microvascular integrity and blood flow to the area. The presence of microvascular blood flow was shown to correlate with recovery of function after myocardial infarction and is an accurate manner of hibernating myocardium in the chronic setting.

The contrast effect can be seen in the cavity and as a fainter effect within the myocardium. It can also be directly visualized in either epicardial or intramural coronary arteries (Fig. 3.48). When there is evidence of contrast
perfusing the intramural coronary arteries, this is excellent evidence of their patency. This finding has been correlated with the presence of a patent epicardial artery after coronary intervention. Contrast within these intramural arteries can also be used to enhance Doppler flow signals.

Detailed analysis of myocardial flow characteristics requires different imaging methodologies than does the simple detection of contrast within the myocardium. To create a time of appearance curve requires a bolus effect in the coronary circulation, which can be obtained in several ways. After intravenous injection of a contrast agent, the microbubbles will appear first on the right side of the heart, then in the left heart, and last in the aorta, the coronary arteries, and the myocardial capillaries. Thus, a single intravenous injection results in the ability to record only one time appearance curve in the myocardium. For detailed evaluation, multiple time appearance curve analyses are necessary, often targeted to different regions of interest or performed under basal conditions and after vasodilator stress. Obviously, in view of the persistence of the newer contrast agents, one would need to wait 10 minutes or more before repeating an intravenous injection to obtain a second bolus. An alternate strategy for obtaining multiple bolus effects is to rely on purposeful destruction of the contrast agent. This can be accomplished by delivering a burst of high-intensity (high MI) ultrasound to the image field. This has the effect of destroying contrast that is present in relatively low concentration in the myocardium and reducing the contrast intensity in the myocardium to near zero. Imaging is then continued either in a continuous or intermittent format while myocardial replenishment occurs, from which a time intensity curve can be generated. If ultrasound contrast is present in the bloodstream at a steady-state concentration, this technique allows the creation of multiple “pseudo-boluses” for the evaluation of different regions of interest from different views or of repeated analysis under basal and stress conditions.
FIGURE 3.46. Contrast echocardiogram demonstrates myocardial contrast effect in an animal model of acute myocardial infarction. A: A short-axis image immediately after occlusion of a coronary artery. Note the opacification of the majority of the myocardium and the absence of contrast effect in the posterior wall (arrowheads). B: Image was recorded immediately after release of the coronary occlusion (brief occlusion) and demonstrates hyperemic flow in the previously occluded zone. Note the dramatic increase in contrast intensity in the previously occluded zone compared with the contrast intensity in the remaining myocardium. C: Image was recorded in the chronic phase of coronary occlusion and again demonstrates a distinct contrast-free zone in the posterior wall. D: The corresponding anatomic specimen shows excellent correlation between the anatomic location and extent of myocardial infarction and that predicted by absence of flow with contrast echocardiography.
FIGURE 3.47. Myocardial contrast echocardiogram performed using a continuous infusion in continuous low mechanical index imaging. This frame was recorded approximately 20 cardiac cycles after a burst phase and reveals the absence of contrast effect in the apical myocardium (arrows) and a robust myocardial contrast effect in the remaining walls. This patient was subsequently demonstrated to have a total occlusion of the distal left anterior descending coronary artery.
Analysis of myocardial perfusion with ultrasound contrast requires specific acquisition algorithms. As mentioned previously, interaction of the high-intensity ultrasound with a contrast agent results in bubble destruction and lack of contrast effect; therefore, if one wishes to detect contrast within the myocardium, standard imaging algorithms will be counterproductive. The two commonly used methods for detecting contrast, without resulting in counterproductive destruction, are continuous low MI imaging and intermittent triggered imaging. Either of these methods may be used with any of the ultrasound domains including B-mode imaging, harmonic or ultraharmonic imaging, power Doppler imaging, and phase correlation techniques. Continuous low MI imaging is the easiest to understand because it provides continuous imaging of all targeted cardiac structures with real-time visualization of wall motion, ventricular function, and myocardial thickening, simultaneously with the ability to observe contrast flow into the myocardium. Note in the real-time images for Figures 3.49 and 3.50 the instantaneous burst that represents purposeful destruction of ultrasound contrast, followed by the progressive reappearance of contrast within the
myocardium. This imaging format allows simultaneous evaluation of left ventricular systolic function and regional wall motion. If this method for myocardial contrast analysis is used, generation of a curve can be undertaken either by continuous frame-by-frame analysis within regions of interest or by analyzing only a fixed time point with reference to the electrocardiogram in sequential images after the burst. The advantage of analyzing intensity only at one time point at each cardiac cycle is that it results in less motion artifact and hence a smoother appearance curve.

A second method for detecting contrast in the myocardium without its destruction is to use intermittent triggered imaging. As mentioned previously, continuous high MI imaging results in continuous destruction of microbubbles. By imaging only intermittently, time is allowed for replenishment of the ultrasound contrast agent within the myocardium, and, hence, it can be detected with each subsequent ultrasound pulse. Intermittent imaging capitalizes on this phenomenon by imaging, triggered to the QRS, at progressively longer intervals. If one images with each cardiac cycle, there is continuous destruction of ultrasound in the myocardium and no time for contrast replenishment in the myocardium. If the imaging interval is doubled, there will be twice as much time for replenishment, and, hence, each subsequent pulse detects twice the contrast effect. Similarly, if the triggering interval is increased further to 1:4, 1:8, 1:16, and so on, then progressively longer periods of time will be provided for replenishment. With progressively longer triggering intervals, a greater myocardial contrast intensity will be noted (Fig. 3.51). Although not allowing a simultaneous assessment of function and flow, triggered imaging may provide a more visibly obvious contrast effect. With either technique, one or more regions of interest can be drawn in the myocardium and the intensity of contrast tracked either continuously or at each level of sequential imaging. Either method results in an appearance curve demonstrating a baseline low level of myocardial contrast effect, a slope of appearance, and a plateau phase from which various parameters can be extracted that directly relate to myocardial blood volume and flow (Fig. 3.52).
FIGURE 3.49. Apical four-chamber view recorded after intravenous injection of a perfluorocarbon-based contrast agent for the purpose of myocardial perfusion echocardiography. A: Image was recorded at the time of a high mechanical index “burst.” B: Image was recorded immediately after the burst and demonstrates the diminished contrast effect both in the cavity and especially in the ventricular myocardium. C: Frame was recorded four cardiac cycles later and demonstrates restitution of contrast effect in the left ventricular cavity and a faint contrast effect developing within the ventricular myocardium. D: Frame was recorded 10 cardiac cycles after the burst and demonstrates further opacification of the left ventricular myocardium.
FIGURE 3.50. Apical four-chamber view recorded in the same patient depicted in Figure 3.39 at the time of hyperemia due to dipyridamole infusion. The format and timing are identical to those for Figure 4.44. In the presence of a hyperemic state, note the increase in the contrast effect in the ventricular myocardium and the more rapid development of significant contrast effect in the ventricular myocardium.
The intensity of contrast in the myocardium is directly related to myocardial blood volume but only indirectly to coronary blood flow. The flow rate is related to the slope of appearance. As with any indicator technique, a contrast time appearance curve can be generated and multiple parameters of such a curve can be correlated with myocardial perfusion. Once curves are generated, either by continuous low MI imaging or by intermittent imaging, analysis can be undertaken for determination of myocardial blood volume and flow. Figures 3.52 and 3.53 schematize stylized contrast echo appearance curves and the different characteristics of the curve that can be related to coronary blood flow. The two most important features of the curve are \( \alpha \), which is the intensity at which the contrast effect plateaus, and \( \beta \), which is the time constant of contrast appearance. \( \alpha \) is directly related to myocardial blood volume, whereas \( \beta \) is related to flow rate. The product of \( \alpha \) and \( \beta \) (\( \alpha \times \beta \)) is directly proportional to myocardial blood flow. Under basal conditions, all areas of ventricular myocardium have roughly equivalent contrast intensity. Because of far-field attenuation and shadowing, the apparent contrast effect may be less in the more basal portions of the heart depending on the imaging plane. Subtraction techniques may assist in demonstrating the contrast effect in these areas. In the absence of a significant coronary stenosis, infusion of a vasodilator increases the flow rate (\( \beta \)), whereas the absolute myocardial blood volume as reflected by \( \alpha \) does not change significantly. In the presence of a total coronary occlusion, there will be a diminished or absent contrast effect. Generally speaking, a coronary
stenosis of less than 90% is not flow restrictive at rest and results in normal contrast appearance kinetics under basal conditions. The addition of a vasodilator such as dipyridamole or adenosine results in an increase in flow velocity only in those areas not perfused by a stenosed artery, and the appearance of the contrast curves will, therefore, differ in the normal and diseased beds. By comparing characteristics of the flow curve including α, β, and their product, a hyperemic ratio can be calculated by comparison of basal and vasodilator contrast injections. Figure 3.53 outlines stylized contrast appearance curves in normal arteries and with various degrees of coronary obstruction.

**FIGURE 3.51.** Graphic demonstration of the intermittent imaging technique for creating a time intensity curve. Four pairs of images are presented. For each, the schematic on the left represents the amount of contrast before imaging and on the right the amount of contrast after contrast imaging. In each instance, there is a decrease in the amount of contrast due to interaction with the ultrasound beam. **Top left:** Imaging is occurring with each cardiac cycle, which allows little time for replenishment of contrast within the target zone. As such, a relatively small amount of contrast is detected with each imaging pulse, and all contrast is destroyed by the subsequent imaging pulse. **Top right:** This example (ratio of 1:2) depicts the effect of imaging every other cardiac cycle. This allows for a greater degree of replenishment of contrast within the target zone, not all of which
is destroyed by the ultrasound beam. **Bottom:** Imaging at ratios of 1:4 and 1:8, which allow for progressively greater amounts of contrast replenishment and hence a greater contrast image intensity, is shown. These results are presented graphically in the center.

Numerous clinical studies have demonstrated the feasibility of using myocardial perfusion contrast echo to identify areas of nonperfused myocardium when compared with radionuclide perfusion imaging or known coronary artery anatomy, or to provide data regarding relative flow in coronary territories. Several clinical studies have shown that preserved perfusion on contrast echocardiography is an accurate marker of viable (i.e., hibernating) myocardium. It should be emphasized that, although myocardial contrast perfusion echocardiography has shown tremendous promise and has demonstrable accuracy in rigorously controlled clinical trials, its ability to detect coronary stenoses in a wide range of patients is still undergoing validation.

**FIGURE 3.52.** Stylized time appearance curve of contrast within the ventricular myocardium depicting the different parameters of a contrast appearance curve. See text for details.
FIGURE 3.53. Stylized time appearance curves depict normal coronary flow and different disease states. The curves on the left are all depictions of baseline appearance curves and those on the right are the anticipated appearance curves at the time of vasodilator stress. Two separate coronary territories representing a normal reference area (A) and an area of coronary obstruction (B) are depicted as solid lines and dotted lines, respectively. **Top:** In the two graphs, both territories A and B are normal and have virtually identical contrast appearance curves. Note that during vasodilation, both curves have a plateau (α) equivalent to that seen at baseline, but the rate of increase of contrast effect (β) is substantially steeper. **Middle:** The appearance curves in the presence of a total coronary occlusion and
myocardial infarction in area B are depicted. Note that curve A is identical to that at baseline but that curve B has a substantially blunted contrast effect. After vasodilatation, there is no change in curve B and curve A behaves as a normal flow territory. **Bottom:** The impact of a significant coronary stenosis in area B is shown. After vasodilatation, territory A has an increased rate of appearance, whereas both the rate of appearance and the plateau contrast intensity for area B are significantly diminished compared with baseline.

Myocardial perfusion contrast echocardiography also remains intensely equipment and protocol specific with respect to results. Finally, it should be emphasized that, while technically feasible and providing a high degree of accuracy for high-resolution analysis of myocardial perfusion, the techniques and agents for its specific use remain unapproved by the FDA for these indications at this time.

A final use of myocardial contrast echocardiography is in monitoring transcatheter alcohol septal ablation performed for treatment of obstructive hypertrophic cardiomyopathy. This is an interventional technique in which a catheter is placed in the first septal perforator (typically) of the left anterior descending coronary artery. Alcohol is then injected to create a controlled myocardial infarction for reduction of the proximal septal mass. This has the effect of reducing the magnitude of dynamic left ventricular outflow tract obstruction and has shown promise for nonoperative treatment of patients with obstructive hypertrophic cardiomyopathy. The goal of this therapy is controlled septal mass reduction. Contrast echocardiography, with the agent injected directly into the septal perforator, plays a major role in determining the feasibility of the procedure and in following its progress (Figs. 3.54 and 3.55). Before injection of ethanol, dilute ultrasound contrast agent is injected into the selected artery. This serves two purposes. The first is to ensure that there is no significant reflux of the contrast into the body of the left anterior coronary descending artery or into the bloodstream itself. Additionally, in some individuals, there may be a significant amount of contrast that appears in the right ventricular cavity. In any of these noted instances, one would anticipate that injection of ethanol into the selected artery would result in the ethanol being delivered not to the localized area of myocardium but more diffusely to the myocardium or the right ventricle. In these instances, the procedure may not be feasible. The second role that the contrast plays is to confirm the presence and size of the perfused bed. The goal of this procedure is that the proximal septum and ideally the area resulting in dynamic
obstruction are selectively “reduced.” Because the contrast serves as a marker of the eventual route of the destructive ethanol injection, myocardial contrast echo serves as an excellent guide for monitoring this procedure.
**FIGURE 3.54.** Apical four-chamber view recorded in a patient with a hypertrophic cardiomyopathy undergoing alcohol septal reduction therapy. **A:** Image recorded under basal conditions. There is a pacemaker catheter in the right ventricle (*arrow*) and systolic anterior motion of the mitral valve. Note the marked hypertrophy of the ventricular septum. **B:** Image was recorded after injection of a diluted perfluorocarbon-based contrast agent into a septal perforator artery in the cardiac catheterization laboratory. Note the distinct contrast in the proximal ventricular septum (*double-headed arrow*) maximum at the area of mitral valve contact with the septum in systole. This patient subsequently underwent successful reduction therapy for treatment of hypertrophic cardiomyopathy.
FIGURE 3.55. Parasternal long-axis view recorded in a patient with hypertrophic cardiomyopathy being considered for alcohol septal reduction therapy. A: Image was recorded at baseline. Note the hypertrophy of the ventricular septum and the systolic anterior motion of the mitral valve. B: Image was recorded after injection of a diluted perfluorocarbon-based contrast agent into a septal perforator artery. Note the absence of contrast effect in the ventricular septum but the appearance of contrast in the right and left ventricular cavity and the marked contrast effect in right ventricular muscle trabeculae (arrow). This patient was not considered a candidate for alcohol septal reduction therapy and the procedure was not performed.

Video 3-55

Suggested Readings

**General Principles**


**Left Ventricular Opacification**


**SAFETY OF CONTRAST ECHOCARDIOGRAPHY**


**MYOCARDIAL PERFUSION CONTRAST ECHOCARDIOGRAPHY**


**Miscellaneous/Experimental Studies**


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To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Echocardiography is a highly versatile technique that can be applied in various clinical settings. It includes several modalities that, taken together, comprise a comprehensive echocardiographic examination (Table 4.1). Patients are usually referred for echocardiography to investigate symptoms or abnormalities found on a physical examination, to evaluate a known or suspected clinical condition, or to screen a subject for the possibility of disease. The value of the diagnostic information depends on the quality of the study and the likelihood that the results will provide new information that will have an impact on the patient’s management or well-being. Guidelines have been jointly published by the American Society of Echocardiography and other national and international organizations that critically evaluate the strength of evidence for the use of echocardiography in various clinical situations. In addition, Appropriate Use Criteria have been developed and critically assessed for numerous clinical situations. Throughout this book, the recommendations provided by these guidelines are highlighted.

The ability to record high-quality echocardiographic images and obtain accurate Doppler flow recordings are essential determinants of the overall value of the echocardiographic examination. As such, echocardiography is highly operator dependent. It is difficult to overemphasize the critical role of the person who performs the imaging. Echocardiography can also be regarded as a partnership between the individual who obtains the data and the one who interprets the study. To obtain a comprehensive and accurate echocardiogram, the operator must understand the anatomy and physiology of the cardiovascular system, have a thorough knowledge of the ultrasound
equipment to optimize the quality of the recording, know the specific diagnostic questions that are being asked, and be able to apply the technology to the individual patient so that optimal imaging can be achieved.

### Table 4.1 IMAGING DOMAINS IN CLINICAL ECHOCARDIOGRAPHY

<table>
<thead>
<tr>
<th>Anatomic imaging domains</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single-line interrogation</td>
</tr>
<tr>
<td>M-mode echocardiography</td>
</tr>
<tr>
<td>Multiple-line interrogation</td>
</tr>
<tr>
<td>Two-dimensional echocardiography</td>
</tr>
<tr>
<td>Multiple-dimensional imaging</td>
</tr>
<tr>
<td>Three-dimensional imaging</td>
</tr>
<tr>
<td>Reconstructed</td>
</tr>
<tr>
<td>Real-time three-dimensional imaging</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Doppler domains</th>
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</thead>
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<td>Pulsed Doppler methods</td>
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<tr>
<td>Single-interrogation volume</td>
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<td>Multiple-interrogation volume</td>
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<tr>
<td>Saturated-interrogation volume area</td>
</tr>
<tr>
<td>Color flow imaging</td>
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<tr>
<td>M-mode color interrogation</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Continuous wave Doppler</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Analysis domains</th>
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</thead>
<tbody>
<tr>
<td>Frequency shift</td>
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<tr>
<td>Power spectrum</td>
</tr>
<tr>
<td>Variance</td>
</tr>
<tr>
<td>Correlation methods</td>
</tr>
<tr>
<td>Tissue velocity imaging</td>
</tr>
<tr>
<td>Strain rate imaging</td>
</tr>
</tbody>
</table>

| Speckle/tissue signature tracking |

### A QUALITY ECHOCARDIOGRAPHIC LABORATORY

An echocardiography laboratory is, by its very nature, a service organization. To provide a consistent and high-quality level of service, preparation, expertise, adherence to best practices, quality monitoring, standardization, and a service-oriented philosophy are essential. To paraphrase from an Institute of Medicine report on quality in health care, quality in echocardiography can be defined as “the degree to which the service
increases the likelihood of a desired outcome in a manner consistent with current professional knowledge.” Over the past decade, greater attention has been focused on defining high-quality echocardiography, setting minimum performance standards, reducing variability, and creating processes to achieve and document excellent service.

The basic components of a modern echocardiography laboratory include the physical space and equipment. Accreditation of the facility can be achieved through the Intersocietal Accreditation Commission (IAC), a rigorous but valuable process that is recommended for all laboratories. Among other things, IAC accreditation mandates that cardiac ultrasound equipment is able to perform two-dimensional (2D), M-mode, transesophageal echocardiography (TEE), and Doppler (spectral, tissue, and color flow) imaging. In recent years, the ability to offer three-dimensional (3D) and strain imaging is also necessary. IAC also sets standards for those who perform and interpret echocardiograms. Sonographers should be credentialed through the American Registry of Diagnostic Medical Sonographers (ARDMS) or Cardiovascular Credentialing International (CCI). Physicians who independently interpret echocardiograms in an accredited laboratory should have at least level II training as defined by the Core Cardiology Training Statement (COCATS) and should actively participate in ongoing educational efforts to maintain competency. Demonstrating special competency by successfully completing a National Board of Echocardiography examination is another means for practicing physicians to contribute to a quality service.

An essential component of a quality echocardiography service is the service itself, which can be broken down into a series of steps all necessary to provide a consistently excellent product. The steps in the process include the following:

1. Patient/test selection—Proper test application, and avoiding inappropriate testing, is best accomplished through awareness of Appropriate Use Criteria. Does the patient need testing? Which test would be most likely to yield the necessary diagnostic information? Can the test that was ordered be performed safely and with a high likelihood of answering the clinical questions? Will the findings lead to a change in management or a better outcome?
Image acquisition—This requires modern and well-maintained imaging equipment and a well-trained sonographer knowledgeable of the patient’s medical condition and the reason for the study. Technical expertise, quality equipment, standard operating protocols, and an understanding of the clinical question are essential to ensure acquisition of a complete set of imaging data.

Storage and access—Secure, HIPAA-compliant, and redundant storage of digital imaging information assures ongoing availability and access to the echocardiographic images and reports. Remote access to images facilitates patient care and reduces the need for unnecessary repetition of echo studies, but must be done in a manner to ensure security of protected patient data.

Interpretation—This requires clinical and imaging expertise on the part of the physician, the ability to perform and analyze measurements, and the skill to integrate imaging and clinical data to arrive at an accurate and meaningful interpretation.

Report generation—The report should be compiled in a timely manner and include all the key elements in a consistent and clear format. Structured reporting implies a comprehensive, unambiguous, and standardized template that includes both qualitative and quantitative information.

Dissemination and implementation—The final step is communication of the findings. Depending on the critical nature of the results and the urgency of the situation, the findings may be provided directly to the referring physician, or in a written or electronic format. A policy for rapid communication of critical findings is essential. The interpreting physician should also be readily available to discuss and clarify any issues raised by the echocardiographic findings.

**APPROPRIATE USE CRITERIA**

In response to a growing concern about utilization of imaging services, Appropriate Use Criteria for echocardiography were developed initially in 2007 and then updated in 2011. These documents are evidence-based guidelines that examine, for specific clinical situations, whether the test is justified on the basis of a rigorous set of criteria. Using this approach, an appropriate study is one in which the expected incremental information, combined with clinical judgment, exceeds the expected negative
consequences by a sufficiently wide margin for a specific indication that the procedure is generally considered acceptable care and a reasonable approach for the indication. Using this definition of “appropriate,” the assessment of value of echocardiography in over 200 representative clinical scenarios was performed. For each case, the application of echocardiography was deemed appropriate, rarely appropriate, or uncertain.

The objective of these Appropriate Use Criteria is to provide support for the use of echocardiography in situations where the test result is expected to improve patient care. Alternatively, the criteria also define clinical situations in which echocardiographic results may not alter patient care, improve outcome, or provide important incremental diagnostic information. Revisions and updates to the current set of appropriateness criteria can be expected in the future. In addition, it is likely that appropriate use criteria will be developed comparing the relative value of the different imaging modalities in various clinical settings.

APPROACH TO THE ECHOCARDIOGRAPHIC EXAMINATION

Most echocardiographic examinations are comprehensive. That is, a thorough and fairly standardized approach is undertaken with the goal of recording a complete array of images and Doppler data that address the full spectrum of possible diagnoses (Table 4.2). 3D imaging is being incorporated into the echocardiographic (both transthoracic and transesophageal) examination with increasing frequency. Currently, 3D imaging is best regarded as an adjunct to the 2D examination. 3D imaging complements, but does not take the place of the 2D examination, much like Doppler imaging. That is, it does not take the place of 2D echocardiography, but is a supplement to it. This is in contrast to TEE, which usually is a distinctly separate examination.

Occasionally, a more targeted or focused examination is undertaken that is only concerned with a specific diagnostic issue, often comparing the current situation with a previous examination. In other situations, an entirely different approach is required, such as when evaluating an infant with suspected complex congenital heart disease. Clearly, echocardiography requires an individualized approach and each patient represents a unique set
of problems and challenges. The technical details involved in obtaining a high-quality echocardiogram are unique, and the examination must be customized for each patient. It is not feasible to simply place the transducer at routine locations on the chest and expect standardized, high-quality images to be available in each patient. The examiner must rely on experience, persistence, and creativity to record the most comprehensive and highest-quality data. Additional factors, including transducer selection, instrument settings, patient comfort and positioning, and even the patient’s breathing pattern, will also affect the quality of the recording.

Most ultrasound systems are equipped with a selection of transducers with a range of capabilities and limitations. With the exception of dedicated continuous-wave Doppler transducers (called nonimaging or Pedoff), most probes are capable of performing M-mode imaging, 2D imaging, and Doppler imaging (Fig. 4.1). It is rare that one transducer setting is ideal for every aspect of a given examination. For instance, a high-frequency imaging transducer may provide optimal resolution for near-field imaging (such as the right ventricular free wall or the cardiac apex) but will offer inadequate penetration to allow imaging of the far field.

In addition to transducer frequency, transducer size or “footprint” is also a consideration. The footprint refers to the dimensions of the surface area coming in contact with the patient’s skin. Because of the relatively narrow spaces between the ribs, the footprint can be a limiting factor in transducer selection (Fig. 4.2). In this illustration, the distal septum and posterior left ventricular wall are obscured by the rib shadow along the left side of the image. If the transducer surface is too big to fit between ribs or to maintain continuous contact with the skin, suboptimal imaging will be obtained. In all cases, the footprint area of current-generation 3D transducers is larger by 30% to 50% compared with standard 2D transducers.
A variety of transducers are available for use in clinical echocardiography. Five transthoracic probes are shown, illustrating the range of capabilities.

### Table 4.2
TRANSTHORACIC ECHOCARDIOGRAPHIC VIEWS

<table>
<thead>
<tr>
<th>Two Dimensional</th>
<th>Three Dimensional</th>
<th>Doppler</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Parasternal</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Long axis</td>
<td>Full volume of the long axis</td>
<td>CFI of MR, AR, VSD</td>
</tr>
<tr>
<td>Medially angulated long axis</td>
<td>Full volume of the short axis</td>
<td>RV inflow, TR</td>
</tr>
<tr>
<td>Short axis (multiple levels)</td>
<td>Narrow angle of the AV and MV</td>
<td></td>
</tr>
<tr>
<td>Basal</td>
<td>3D color of the valves, septa</td>
<td></td>
</tr>
<tr>
<td>MV level</td>
<td>Zoom on any region of interest</td>
<td>CFI of AR, TR, PS, PR, VSD</td>
</tr>
<tr>
<td>Papillary muscle level</td>
<td></td>
<td>CFI of MR</td>
</tr>
<tr>
<td>Apical</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Apical</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Four chamber</td>
<td>Full volume of the four chambers</td>
<td>Mitral, tricuspid inflow; MR, TR</td>
</tr>
</tbody>
</table>

**FIGURE 4.1.** A variety of transducers are available for use in clinical echocardiography. Five transthoracic probes are shown, illustrating the range of capabilities.
<table>
<thead>
<tr>
<th>Two chamber</th>
<th>Narrow angle of the valves, septa</th>
<th>MR, AR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long axis</td>
<td>3D color of the valves, septa</td>
<td>MR, AR</td>
</tr>
<tr>
<td>“Five” chamber</td>
<td>Zoom on any region of interest</td>
<td>LV outflow, AR</td>
</tr>
</tbody>
</table>

**Subcostal**

- **Four chamber**: Full volume of the four chambers, RV inflow, TR, ASD
- **Short axis**: 3D color of the septa, TR, pulmonary flow, PR
- **Basal**: IVC, hepatic veins
- **Mid ventricular**

**Suprasternal**

- **Arch in long axis**: Full volume of the aortic arch, Ascending/descending aortic flow
- **Arch in short axis**: 3D color of the aorta

AR, aortic regurgitation; AS, aortic stenosis; ASD, atrial septal defect; CFI, color flow imaging; IVRT, isovolumic relaxation time; LVOT, left ventricular outflow tract; MR, mitral regurgitation; PDA, patent ductus arteriosus; PR, pulmonic regurgitation; PS, pulmonic stenosis; SVC, superior vena cava; TR, tricuspid regurgitation; VSD, ventricular septal defect.

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**PATIENT POSITION**

The transthoracic examination can be performed with the echocardiographer (or sonographer) sitting on the patient’s left or right side. This is largely a matter of personal preference, comfort, and custom. When seated to the right side of the patient, scanning is performed with the right hand. If the left side is used, usually the operator scans with his or her left hand and manipulates the machine settings with the right hand. Developing experience scanning from both sides is recommended. Not only does this minimize the risk of repetitive use injury, but it prepares the sonographer for room situations where only one side of the bed may be available for approaching the patient.

One of the goals of the echocardiographic examination is to obtain the highest-quality images without creating unnecessary discomfort or anxiety for the patient. Because transthoracic echocardiography (TTE) can take as long as an hour, the comfort and well-being of both the examiner and the patient are important. The transthoracic echocardiographic examination
usually requires more than one patient position. For most adult patients, imaging is performed with the patient either supine and/or in the left lateral decubitus position (Fig. 4.3). By tilting the patient to the left, the heart is brought forward to the chest wall and more to the left of the sternum thereby improving the ultrasound windows. The degree to which the patient should be rotated to the left must be individualized, and occasionally excellent images can be obtained with the patient in the supine position.

**FIGURE 4.2.** An example of rib shadowing (arrows). The presence of the rib relative to the transducer footprint obscures the distal septum and posterior wall of the left ventricle.
FIGURE 4.3. Proper positioning for the echocardiographic examination. The transducer is placed over the apical window, and the patient is tilted in the left lateral decubitus position.

Additional patient positions are often necessary. Tilting the patient into the right lateral decubitus position may be necessary in some forms of congenital disease or to record aortic valve flow (Fig. 4.4). To facilitate subcostal
imaging, a supine position with the legs flexed at the knees generally provides the greatest relaxation of the abdominal muscles so that the transducer can be properly positioned (Fig. 4.5). To use the suprasternal notch as an ultrasound window, it is often necessary to place a pillow behind the patient’s shoulders so that the neck can be comfortably hyperextended, thereby creating an opening for transducer placement (Fig. 4.6). Finally, even the sitting position may sometimes be required, especially for some forms of congenital heart disease.

![FIGURE 4.4. The right lateral decubitus position is shown, and a Pedoff transducer is applied to record ascending aortic flow.](image)

Patient cooperation is an important consideration in the echocardiographic examination. Explaining the purpose of the examination, ensuring the patient’s comfort, and stressing the safety and noninvasive aspects of ultrasound will alleviate anxiety and enhance cooperation. It is sometimes necessary for the patient to suspend breathing or perform a Valsalva maneuver, in order to obtain optimal imaging. In children and infants in whom anxiety and lack of cooperation can be anticipated, special approaches
are necessary. Enlisting the assistance of a parent is frequently adequate, although sedation may occasionally be necessary to complete the examination.

**PLACEMENT OF THE TRANSDUCER**

The goal of the transthoracic echocardiographic examination is to acquire a complete ultrasound interrogation from all the available acoustic windows. In doing so, the heart can be visualized in multiple orthogonal planes, allowing tomographic and volumetric data to be integrated in a coherent manner. The transducer locations endorsed by the American Society of Echocardiography for transthoracic imaging in the adult include the left and right parasternal locations, the cardiac apex, the subcostal window, and the suprasternal notch location. The examination is frequently begun with the patient lying in the supine position, rotated into the left lateral decubitus position, and the transducer located at the left parasternal position. Depending on body habitus, the presence or absence of lung disease, and the position of the heart within the thorax, the optimal intercostal space for recording the “parasternal views” will vary. Imaging from the cardiac apex frequently requires tilting the patient into a steep left lateral decubitus position. By palpation, the point of maximal impulse is located and used as the starting point for apical imaging. The subcostal approach is particularly important in patients with advanced lung disease or thick chest walls and provides the unique opportunity to view the inferior vena cava, hepatic veins, and many of the important congenital anomalies. The suprasternal notch is most useful to visualize the great vessels and left atrium (Fig. 4.7).
FIGURE 4.5. The transducer is applied to the subcostal window, with the patient in the supine position.
FIGURE 4.6. To record aortic flow from the suprasternal notch, it is often necessary to elevate the shoulders using a pillow to tilt the head backward.

Less commonly used windows include the right parasternal location. This position is useful to examine the aorta or interatrial septum and is also useful in patients with congenital malposition of the heart, such as dextrocardia. It plays a major role in the assessment of aortic stenosis. This approach usually requires positioning the patient in the right lateral decubitus position. The right apical, right supraclavicular fossa, and even the back are potential acoustic windows that must occasionally be used. For example, the right supraclavicular examination often provides the best opportunity to visualize the superior vena cava.

It should be emphasized that the standard patient positions and transducer locations serve only as a general guide, applicable to most patients. In patients with chest deformities, such as pectus excavatum, or those with chronic obstructive lung disease, these standard approaches may be inadequate. Likewise, some anomalies within the thorax, including dextrocardia, pleural effusion, and pneumothorax may also render the standard approaches ineffective. In such cases, it is the experience and
creativity of the examiner that will often determine the value of the information derived from the transthoracic study. Using the transducer as an exploratory camera will occasionally reveal unexpected acoustic windows that will yield important diagnostic information.

**FIGURE 4.7.** The suprasternal notch provides a unique opportunity to record the aortic arch and the origin of the great vessels.
In most laboratories, the parasternal window serves as a starting point for the study. Beginning at the third left intercostal space, the transducer is applied and rotated to record the parasternal long-axis view. To optimize the image, it may be necessary to move up or down one or two intercostal spaces and to rotate the patient into a left lateral decubitus position. When properly recorded, this view depicts the mid portion and base of the left ventricle, both leaflets of the mitral valve, the aortic valve and aortic root, the left atrium, and the right ventricle (Fig. 4.8). The left ventricular apex is rarely visualized from this window. The transducer position should be adjusted so that the scanning plane is parallel to the major axis of the left ventricle and passes through the center of the left ventricular chamber. This is the point where the minor-axis diameter is maximal and the mitral valve leaflet excursion is greatest. This is best accomplished by gradual medial to lateral angulation until left ventricular size is at its maximum. From this view, an M-mode cursor can be placed to record minor-axis dimensions (Fig. 4.9). This orientation will record the full excursion of the mitral valve, aortic valve opening and closing, right ventricular free wall motion, and the left ventricular septal and posterior wall motion. The coronary sinus will be visualized in the posterior atrioventricular groove, just below the base of the posterior mitral leaflet. An example of this is shown in Figure 4.10, which demonstrates the normal relationship between the coronary sinus, the atrioventricular groove, and the descending aorta. Behind the left atrium, a portion of the descending aorta will often be recorded. This view is also ideal to confirm the presence or absence of a pericardial effusion. A narrow, echo-free space behind the posterior left ventricular wall but anterior to the descending aorta is strongly suggestive of pericardial fluid.
FIGURE 4.8. The parasternal long-axis view.
PARASTERNAL LONG-AXIS VIEWS

An imaging plane aligned parallel to the long axis of the left ventricle will not, in most cases, be exactly parallel to the left ventricular outflow tract and aortic root. This is illustrated in Figure 4.11, which demonstrates that slight counterclockwise rotation of the transducer is needed to follow the long axis of the left ventricle into the long axis of the aorta. In this illustration, the true dimensions of the proximal aorta are underestimated in the left panel, which shows a properly aligned parasternal long-axis view. By slightly rotating the transducer (right panel), the aortic root is “opened up” and the true long axis of the aorta is demonstrated. In most patients, some angulation of the scan plane from medial to lateral is required to obtain a complete interrogation of the aortic valve, including the leaflets, annulus, and sinuses.
FIGURE 4.10. This parasternal long-axis view illustrates the relationship between the coronary sinus (arrow) and the descending aorta (DA). [Video 4-10 coming soon]
FIGURE 4.11. A: The parasternal long-axis view is adjusted so that the scan plane is parallel to the long axis of the left ventricle. In this plane, the proximal aorta appears normal. B: The plane is rotated slightly counterclockwise to better align with the long axis of the ascending aorta. By doing so, the true dimension of the aortic root is apparent.

An important advantage of the parasternal long-axis view is that it orients many of the structures of interest perpendicular to the ultrasound beam, which improves target definition by increasing resolution. By moving the transducer to a lower interspace, the left ventricular apex can be included in the field of view and an apical long-axis plane can be recorded. The advantage of this view is, of course, the ability to include the apex. The major disadvantage is that major structures, particularly the walls of the left ventricle, now lie more parallel to the transducer beam, thereby reducing endocardial definition and making wall motion analysis more difficult. This issue is covered in detail later in this chapter.

Starting from the parasternal long-axis view, medial angulation of the scan plane affords an opportunity to examine the right atrium and right ventricle (Fig. 4.12). As the plane is swept under the sternum, the posterior segment of the interventricular septum is recorded, as is the posteromedial papillary muscle, and eventually the right ventricular inflow tract. Because the right
ventricular inflow tract is not parallel to its left ventricular component, slight clockwise rotation of the transducer is generally required. In this plane, the important landmark is the tricuspid valve and the plane is considered optimized when the full excursion of the anterior and posterior tricuspid leaflets is recorded and the right ventricular dimension is greatest. This recording permits the inferior portion of the right atrium, including the eustachian valve and occasionally the inferior vena cava, to be visualized. By further rotation of the transducer, a plane that records the right ventricular outflow tract, pulmonary valve, and main pulmonary artery is obtained (Fig. 4.13A). In this example, the entire length of the main pulmonary artery is seen and trivial pulmonary regurgitation is demonstrated. To record the bifurcation of the main pulmonary artery, either this view or the basal short-axis view (Fig. 4.13B) is ideal.

**FIGURE 4.12.** Two examples of the right ventricular inflow view are shown. A: A
portion of the left ventricle is preserved within the scan plane. B: Further angulation excludes the left ventricle and only the right atrium and right ventricle remain.

**FIGURE 4.13.** A: The right ventricular outflow view records the right ventricular outflow tract and the main PA. Trivial pulmonary valve regurgitation (arrow) is illustrated. B: The bifurcation of the main pulmonary artery is seen from the basal short-axis view.
Doppler evaluation of the parasternal long-axis view is useful to record blood flow through the mitral and aortic valves (Fig. 4.14). Because the flow of blood is not parallel to the ultrasound beam, quantitation of flow velocities
is generally not possible. However, color flow Doppler from this view is routinely used to detect aortic or mitral regurgitation. In this example, a systolic frame demonstrates acceleration of blood in the left ventricular outflow tract, toward the aortic valve. No evidence of mitral regurgitation is recorded. Slight medial angulation provides an excellent opportunity to detect flow through a ventricular septal defect. Further medial angulation permits Doppler recording of tricuspid valve inflow and both qualitative and quantitative assessment of tricuspid regurgitation.

A volumetric 3D recording from the parasternal window has many of the same advantages and limitations as the 2D view (Fig. 4.15). That is, the mid and basal portions of the left ventricle and the aortic and mitral valves are well visualized, but the apex is often excluded. Valve structure, wall motion, and chamber sizes can be evaluated with 3D echocardiography using this acoustic window.
FIGURE 4.15. A three-dimensional image from a normal subject, recorded from the parasternal window. The image is oriented in the long-axis plane and illustrates how the thickness of slice can be used to record three-dimensional depth.
PARASTERNAL SHORT-AXIS VIEWS

From the parasternal long-axis transducer position, clockwise rotation of the transducer approximately 90 degrees moves the imaging plane to the short-axis view. By rotating the transducer clockwise, the patient’s lateral wall is placed to the observer’s right and the medial wall to the observer’s left. Although theoretically an infinite number of short-axis planes exist between the base and apex of the heart, in practice, three or four representative views are recorded from this general transducer position. Because these different planes span several centimeters, some repositioning of the transducer is necessary, requiring moving from the second through the fourth intercostal spaces and tilting the transducer at various angles. The relationship of the various short-axis planes to the long-axis view is demonstrated in Figure 4.16.

A useful reference point to begin the short-axis examination is the tip of the anterior mitral valve leaflet. By rotating the transducer slightly and adjusting the tilt of the plane, the left ventricle can be made to appear circular and both leaflets of the mitral valve will demonstrate maximal excursion (Fig. 4.17A). As in all short-axis views, the left ventricle is displayed as if viewed from the apex of the chamber. When properly recorded, the short-axis view in this plane corresponds roughly to the mid left ventricular level and allows optimal recording of mitral leaflet excursion, mid left ventricular wall motion, and visualization of a portion of the right ventricle. The normal
Interventricular septal curvature can be appreciated and any abnormalities of septal position, shape, or motion can be assessed. Minor base-to-apex angulation is useful to record the orifice of the mitral valve, the coaptation of the leaflets, and the mitral chordae and their insertion into the anterolateral and posteromedial papillary muscles. Using real-time 3D echocardiography, a volumetric recording from the parasternal window permits a series of short-axis planes to be derived. From this family of planes, selected short-axis 2D images can be displayed and analyzed. One practical application of this approach is the precise recording of the mitral orifice in patients with mitral stenosis (Fig. 4.18).

Moving to a more basal plane, the short-axis view approaches the level of the aortic annulus and allows simultaneous visualization of several important structures (Fig. 4.17B). In addition to the annulus, the aortic valve, coronary ostia, left atrium, interatrial septum, right atrium, tricuspid valve, right ventricular outflow tract, pulmonary valve, and proximal pulmonary artery can also be recorded. Occasionally, the left atrial appendage can also be visualized from this plane. When properly aligned, the three cusps of the aortic valve can be seen to open and close in systole and diastole, respectively. Immediately superior to the annulus, the ostia of the left and right coronary arteries can be seen. If the annulus is regarded as a clock face, the left main artery originates at approximately 4 o’clock and the right coronary artery at 11 o’clock (Fig. 4.19). The nearly orthogonal relationship between the aorta and the pulmonary artery and the relative positions of the aortic and pulmonary valves can be appreciated. With slight superior angulation, the pulmonary artery can be followed to its bifurcation and both the right and left branches identified (Fig. 4.13B).
FIGURE 4.16. This schematic demonstrates the various short-axis planes that can be derived from the parasternal long-axis view. Note that the planes are not exactly parallel but provide views of anatomy from apex to base.
FIGURE 4.17. Two short-axis views are provided. A: The short-axis view at the level of the MV. B: A basal short-axis projection at the level of the aortic valve.
By moving the transducer to a lower interspace and angling the scan plane more apically, the image will sweep through the papillary muscle level and then the left ventricular apex (Fig. 4.20). This series of views is ideal for assessing the contractile pattern of the left ventricle at the midventricular and apical levels. When recording these views, adjustments are aimed at maintaining the near-circular appearance of the left ventricular cavity as the overall cavity size decreases toward the apex.

FIGURE 4.18. Three-dimensional views of a stenotic mitral valve are shown. In (A), the valve is shown from the perspective of the left atrium. In (B), from the perspective of the left ventricle. AL, anterior mitral leaflet; PL, posterior mitral leaflet.

Video 4-18a
FIGURE 4.19. From the basal short-axis view just above the aortic valve, the origins of the left coronary artery (LCA) and right coronary artery (RCA) can be recorded.
The Doppler evaluation of the various parasternal short-axis views serves several purposes. At the base of the heart, the scan plane can be adjusted so that blood flow is oriented nearly parallel to the ultrasound beam through both the tricuspid and pulmonary valves. Both tricuspid inflow and tricuspid regurgitation can be recorded from this position. Slight angulation permits a similar assessment of pulmonary valve flow from the same basal view (Fig. 4.21). Conversely, aortic flow is nearly perpendicular to the scan plane; therefore, quantitative Doppler assessment of aortic flow is not possible. However, color flow imaging just below the aortic valve (at the level of the left ventricular outflow tract) may allow visualization of the aortic regurgitant jet as it emerges from the regurgitant orifice (Fig. 4.22). An assessment of regurgitant jet area at this level is useful. By moving to the mitral valve level, a similar approach using color flow imaging to assess the mitral regurgitant jet is also possible (Fig. 4.23). This may be of particular value to localize the source of mitral valve regurgitant jets. By scanning carefully through the plane of the mitral leaflets, the location and extent of the regurgitant orifice can often be identified.
FIGURE 4.20. A short-axis plane at the level of the papillary muscles (arrows).

Video 4-20

coming soon
With the patient rotated to the left and the transducer placed at the cardiac apex, a family of long-axis images is available. A useful starting point for this part of the examination is the apical four-chamber view, illustrated in Figure 4.24. Once the apical window is located, the transducer is pointed in the general direction of the right scapula and then rotated until all four chambers of the heart are optimally visualized. This occurs when the full excursion of both mitral and tricuspid valves is recorded and the “true” apex of the left ventricle lies in the near field. The normal true apex can be identified by its relatively thin walls and lack of motion. Incorrect transducer position will lead to foreshortening of the left ventricle and failure to visualize the true apex. A common variant seen in normal hearts is the false tendon in the left ventricular apex (Fig. 4.25). Such structures are benign anomalies but must be differentiated from pathologic findings, including a thrombus or tumor. When properly adjusted, this image includes the four chambers, both atrioventricular valves, and the interventricular and interatrial septa. While examining the crux of the heart, it should be noted that the insertion of the septal leaflet of the tricuspid valve is several millimeters more apical than the insertion of the mitral leaflet. In a properly oriented four-chamber view, the anterior mitral leaflet is recorded medially and the smaller posterior leaflet is seen as it arises from the lateral margin of the atrioventricular ring. On the right side, the septal leaflet of the tricuspid valve inserts medially and the larger anterior leaflet arises laterally. Confirming this relationship is useful for orientation of the image and is critical in diagnosing several congenital conditions, such as Ebstein anomaly and endocardial cushion defects. The moderator band is often seen in the right ventricular apex (Fig. 4.26), and the descending aorta can frequently be visualized behind the left atrium. Although the left atrium lies in the far field, the junction of the pulmonary veins into the posterior wall of the chamber can often be seen.
FIGURE 4.21. The basal short-axis view is ideal to record flow through the pulmonary valve using pulsed Doppler imaging.
FIGURE 4.22. Color flow imaging from the short-axis view in diastole, just below the aortic valve, records an aortic regurgitation jet in cross section as it emerges from the valve. "Video 4-22" coming soon
FIGURE 4.23. At the level of the tips of the mitral leaflets, the short-axis view permits the mitral regurgitation jet to be recorded. **A:** Two-dimensional imaging shows thickened mitral leaflets. **B:** Color flow imaging shows the extent of the regurgitant jet at the same level.

coming soon

Video 4-23
FIGURE 4.24. The apical four-chamber view. Video 4-24
FIGURE 4.25. An example of a false tendon (arrows) in the left ventricular cavity.

Video 4-25

By tilting the transducer into a shallower angle relative to the chest wall, resulting in a more anterior scan plane, the left ventricular outflow tract, aortic valve, and aortic root can be recorded (Fig. 4.27). This is frequently referred to as the “five-chamber view,” recognizing the obvious inaccuracy of
the term. Despite the unfortunate terminology, the view has several practical uses. It places both the left ventricular inflow and left ventricular outflow roughly parallel to the ultrasound beam, permitting quantitative Doppler assessment of both patterns simultaneously (Fig. 4.28). In addition, both aortic and mitral regurgitation can be detected from this view, and it is often the best perspective to distinguish between subvalvular and valvular aortic stenosis.

FIGURE 4.26. An apical four-chamber view demonstrates a moderator band (arrow) in the right ventricular apex.
Starting from the four-chamber view, the transducer can be tilted to a shallower angle to produce a plane that includes the left ventricular outflow tract and proximal aorta.

Using the apical four-chamber view as a reference, the other apical views are readily derived. By rotating the transducer counterclockwise approximately 60 degrees, an apical two-chamber view is recorded (Fig. 4.29). The objective here is to completely exclude the right atrium and ventricle from the recording so that only the left ventricle, left atrium, and
mitral valve are visualized. The two-chamber view is also similar in orientation to the right anterior oblique angiographic view. For this reason, it is sometimes referred to as the right anterior oblique equivalent. Although not truly orthogonal to the four-chamber view, the apical two-chamber image records different walls of the left ventricle and the combination of these two views often provides an accurate representation of left ventricular size, shape, and function. The two views are often used in combination for biplane quantitative approaches to left ventricular function. This view also permits the left atrial appendage to be recorded in some patients (Fig. 4.30). Although TEE will always be superior for this purpose, this is one of the few opportunities on transthoracic imaging to visualize this structure.

If the transducer position is returned to the four-chamber orientation and then rotated clockwise approximately 60 degrees, an apical long-axis view is recorded, characterized by the presence of both the mitral and aortic valves in the same plane (Fig. 4.31). This is a similar plane to the parasternal long-axis view except recorded from the apex. An important difference between the two long-axis views is the relationship between the endocardial surface and the ultrasound beam. From the parasternal view, the endocardium is roughly perpendicular to the beam, thereby facilitating endocardial definition. From the apical window, the left ventricular walls and the ultrasound beam are more parallel, which in some cases results in endocardial dropout and poorer visualization of wall motion. An advantage of this view is its utility in detecting and quantifying aortic valvular and subvalvular obstruction, including hypertrophic cardiomyopathy.
From the apical five-chamber view, simultaneous recording of aortic outflow and mitral inflow can be performed. This permits isovolumic relaxation time (IVRT) to be measured.
FIGURE 4.29. An apical two-chamber view. Video 4-29
FIGURE 4.30. The two-chamber view sometimes allows the left atrial appendage (*) to be visualized.
It is sometimes helpful to relate these three apical views as relative positions on a clock face (Fig. 4.32). Starting with the four-chamber view, the left ventricular walls are imaged at the 10 o’clock and 4 o’clock positions. The two-chamber view records left ventricular walls at the 2 o’clock and 8 o’clock positions, whereas the apical long axis bisects the left ventricle at approximately 12 o’clock and 6 o’clock positions. These are only approximate guidelines but serve to orient the three views and underscore the fact that each records different segments of the left ventricle.

Another approach to apical imaging employs real-time 3D echocardiography to capture a volumetric data set. Because this recording includes the entire left (and potentially right) ventricle, it is often used for the calculation of left ventricular volume, mass, and ejection fraction (Fig. 4.33). This specific application of 3D echocardiography has proven to be one of its strengths and is covered in more detail in Chapter 5.
FIGURE 4.31. The apical long-axis view is similar to the parasternal long-axis view but is recorded from a lower interspace.
Doppler evaluation from the apical views has several important applications. The orientation of blood flow relative to the scan plane permits recording of mitral, aortic, and pulmonary venous blood flow profiles from the apex. From the four-chamber view, the Doppler sample volume is first placed at the tips of the mitral leaflets to record mitral inflow (Fig. 4.34). An analogous approach can be taken to sample tricuspid inflow. Aortic outflow is then recorded from the five-chamber view, with the sample volume positioned at the level of the aortic annulus (Fig. 4.35). Pulsed Doppler interrogation of pulmonary venous flow is usually obtained from the apical four-chamber view, despite the considerable distance between the transducer and target (Fig. 4.36). Using a low-velocity scale and keeping the wall filters at a low level, the sample volume is placed within the mouth of the pulmonary vein. In the example shown, the systolic and diastolic filling waves and the slight retrograde flow during atrial systole are all clearly

**FIGURE 4.32.** The relationship among the various apical long-axis views and the parasternal short axis. See text for details.
recorded. Finally, from the apical views, color Doppler imaging should be routinely performed to assess for regurgitation of the mitral, aortic, or tricuspid valve.

FIGURE 4.33. This composite image illustrates how volume and ejection fraction are derived from the three-dimensional image. By tracking the endocardial border over the course of the cardiac cycle, calculation of instantaneous volume of the left ventricle is performed. This represents a semi-automated approach to quantify end-systolic (ESV) and end-diastolic (EDV) volumes from which ejection fraction (EF) is derived.
Tissue Doppler imaging of the mitral annulus should be routinely performed to aid in the assessment of diastolic function and filling pressures. To record annular velocities, use a small sample volume and adjust gain and filter settings to a low level. From the four-chamber view, position the sample volume over the mitral annulus medially in the area of the septum (Fig. 4.37). Annular velocities in the region of the lateral wall should also be recorded. The velocity scale should be turned to its lowest level. Motion of the annulus throughout the cardiac cycle can be recorded in most patients. Finally, color M-mode recording of mitral inflow and left ventricular filling is another approach to the assessment of diastolic function (Fig. 4.38). Using routine color flow imaging for orientation, the M-mode cursor is placed at the center of the inflow jet. The M-mode display reveals the acceleration of blood in early diastole through the mitral valve toward the apex. The slope of the red–blue interface represents the propagation velocity of left ventricular inflow and correlates with the rate of chamber relaxation.
THE SUBCOSTAL EXAMINATION

The subcostal views provide valuable and, in some cases, unique diagnostic information, but cannot be obtained in all patients, primarily due to body habitus. In most patients, placement of the transducer in the subcostal location provides an opportunity to record a four-chamber and a series of short-axis planes. The subcostal four-chamber view is similar to the corresponding apical view with two exceptions. First, the ultrasound beam is oriented perpendicular to the long axis of the left ventricle and thus often provides better endocardial definition of the ventricular walls. Second, because of the position of the transducer relative to the cardiac apex, foreshortening or inability to visualize the left ventricular apex is more likely from the subcostal position (Fig. 4.39). Because of the orientation of the interventricular and interatrial septa relative to the scan plane, this view is particularly useful to examine these structures and to search for septal defects. In adult patients, this is frequently the only echocardiographic view that visualizes the superior portion of the atrial septum, permitting sinus venosus defects to be detected. The proximity of the right ventricular free wall to the transducer also makes this view ideal for assessing right ventricular free wall thickness and motion and may be helpful in evaluating abnormal wall motion in patients with suspected pericardial tamponade (Fig. 4.40).
FIGURE 4.35. The apical five-chamber view allows recording of aortic outflow using the pulsed Doppler technique.

FIGURE 4.36. From the apical four-chamber view, pulsed Doppler imaging can
often be used to record pulmonary venous flow by positioning the sample volume at the junction of the pulmonary vein and LA. In this example, pulmonary venous flow has three phases: a systolic phase (PVs), a diastolic phase (PVd), and a small wave of flow reversal during atrial systole (PVa).

FIGURE 4.37. Tissue Doppler lateral imaging of the lateral mitral annulus demonstrates velocity away from the transducer in systole and two waves toward the transducer (e′ and a′) in diastole.
From the four-chamber view, the transducer can be rotated approximately 90 degrees counterclockwise to record a series of short-axis images. Figure 5.41A demonstrates a short-axis plane at the papillary muscle level. The plane can usually be adjusted to provide an excellent view of the right ventricular outflow tract, pulmonary valve, and proximal pulmonary artery (Fig. 4.41B). This is a useful alternative to the parasternal short-axis view for the assessment of these structures. The orientation of blood flow parallel to the ultrasound beam facilitates quantitative Doppler analysis. From this view, inferior angulation of the transducer can provide multiple short-axis views of the left and right ventricles moving from base to apex. The subcostal view is also useful for direct recording of the inferior vena cava and hepatic veins by modification of the short-axis plane (Fig. 4.42). The dimensions of the inferior vena cava and its response to “sniffing” should be analyzed. Hepatic vein flow is recorded using pulsed Doppler imaging. To record flow in the hepatic veins, it is first necessary to visualize the inferior vena cava, a few centimeters below the diaphragm. Then, using color Doppler imaging, the
liver can be interrogated until a vein is identified oriented parallel to the ultrasound beam. Pulsed Doppler imaging can then be used to record flow velocities within the hepatic vein. For maximal value, hepatic vein flow must be assessed in conjunction with the respiratory cycle.

FIGURE 4.39. A subcostal four-chamber view.
FIGURE 4.40. A subcostal four-chamber view from a patient with a large pericardial effusion (PE). From this window, diastolic right ventricular free wall collapse (arrow) can be demonstrated.
SUPRASTERNAL VIEWS

The primary use of the suprasternal views is to examine the great vessels. Extending and rotating the patient’s head allows positioning of the transducer so that the aortic arch is readily recorded. This can be uncomfortable for the patient and care should be taken to minimize pressure on the patient’s throat. Orientation of the scan plane is based on the position of the arch relative to the ultrasound beam. Although a variety of terms have been used to define the various transducer positions, describing the imaging plane as either parallel or perpendicular to the arch is most intuitive.

When the plane is oriented parallel to the aortic arch, it is often possible to visualize both ascending and descending segments of the aorta as well as the origin of the innominate, left common carotid, left subclavian, and right pulmonary arteries (Fig. 4.43). Because of the proximity of the arch to the transducer, a 90-degree sector may not be wide enough to simultaneously record both ascending and descending segments of the aorta. Angulation of the transducer is necessary for a complete recording in such patients. From this position, the transducer can be rotated 90 degrees to provide the perpendicular plane, which demonstrates the arch in short-axis orientation. From this view, the right pulmonary artery and left atrium can usually be recorded. By adjusting the scan plane leftward and slightly anteriorly, the superior vena cava can also be visualized. Figure 4.44 illustrates the suprasternal short-axis view, demonstrating the aortic arch in cross section,
and, below it, the right pulmonary artery and left atrium can be seen.

**FIGURE 4.41.** A: A subcostal short-axis view at the level of the papillary muscles. B: A short-axis view at the base. This view provides a clear recording of the interatrial septum and the right ventricular outflow tract, pulmonary valve, and main pulmonary artery.

Video 4-41

It should be clear from the previous sections that numerous echocardiographic views can and should be routinely recorded. Using digital techniques, it is common to display multiple views in a single quad screen format. Although any views can be included in the four quadrants, it has become customary to display the parasternal long- and short-axis and the
apical four- and two-chamber views (Fig. 4.45). This format has several advantages, including providing a thorough display of the left ventricular walls. This makes it particularly useful for wall motion analysis and in stress echocardiography. These topics are covered in later chapters.

FIGURE 4.42. A: The subcostal view is adjusted to demonstrate the long axis of the IVC joining the RA. B: Color flow imaging of hepatic vein flow. Video 4-42
ORIENTATION OF TWO-DIMENSIONAL IMAGES

Orientation of the echocardiographic image has been addressed by the American Society of Echocardiography. In the parasternal long-axis view, for example, the aorta is positioned to the right side of the sector scan. In the short-axis view, the right ventricle is displayed to the left side, as if the observer were viewing the heart from the apex. From the apex, the four-chamber view is most often displayed with the right heart to the left of the screen and the left heart to the right. One common variation is to invert the apical images so that the atria are displayed at the top of the screen and the ventricular apex at the bottom (Fig. 4.46). This may be regarded as more anatomically “correct” and is favored by pediatric echocardiographers. As a result, some of the illustrations in the chapter on congenital heart diseases (Chapter 19) follow this convention.

To account for the multiple possibilities with respect to orientation, the
American Society of Echocardiography has recommended a standardized approach to 2D echocardiographic imaging. The society further suggests that all 2D imaging transducers have an index mark that clearly indicates the edge of the ultrasonic plane, that is, the direction in which the ultrasound beam is swept. It is conventional for this index mark to be located on the transducer to indicate that the edge of the image will appear on the right side of the display screen (Fig. 4.47). For example, in parasternal long-axis examination, the index mark should be oriented in the direction of the aorta and the aorta should appear to the observer’s right of the image display. Furthermore, it is recommended that the index mark should point in the direction of either the patient’s head or his or her left side. The effect of this convention is to position the parasternal long-axis view so that the aorta is to the right, the short-axis view so that the right ventricle is to the left side, and the apical four-chamber view so that the left heart is to the right. Finally, the subcostal four-chamber view shows the two ventricles to the right of the screen. These conventions are followed throughout this text.

**ECHOCARDIOGRAPHIC MEASUREMENTS**

2D echocardiography lends itself to quantitation and routine measurements should be a part of most comprehensive echocardiographic examinations. In 2015, the American Society of Echocardiography and the European Association of Echocardiography jointly published recommendations for chamber quantification (Lang, 2015). This provides an excellent compendium of the quantitative analyses and measurements that should be performed, guidelines on how to obtain such measurements, and tables on the normal and abnormal values. A partial list of standard measurements available with TTE is provided in Table 4.3. Quantitation of 3D echocardiography is also performed in many clinical situations, the most common of which involves the determination of left ventricular volumes and ejection fraction. With further experience and continued improvement in technology, additional quantitative applications of 3D echocardiography will likely emerge.
FIGURE 4.44. The suprasternal notch also permits the aortic arch (AA) to be recorded in cross section. This plane allows visualization of the superior vena cava and demonstrates the right pulmonary artery (RPA) coursing below the arch and above the left atrium. (See Video 4-44.)
FIGURE 4.45. In a quad screen format, the four views most often included are the parasternal long- and short-axis and the apical four- and two-chamber views. LAX, long axis; SAX, short axis; 4C, four chamber; 2C, two chamber.

The American Society of Echocardiography has previously made recommendations regarding the descriptive items and quantitative components that constitute a standard report of an adult transthoracic echocardiogram (Gardin et al., 2002). This document offers a comprehensive listing of the various features that should be routinely analyzed and reported. The goal of such a listing is to encourage standardization of echocardiographic reports and to ensure that examinations are thorough and comprehensive. In this text, guidelines for performing and interpreting such measurements are provided in the chapters corresponding to the chamber or valve being analyzed.
FIGURE 4.46. The apical four-chamber view is sometimes displayed with this “anatomically correct” orientation that places the apex down and the atria (RA and LA) above. [Video 4-46]
LEFT VENTRICULAR WALL SEGMENTS

Although the left ventricle could be divided into any number of segments, the American Society of Echocardiography has adopted a set of standards and recommended terminology. The scheme begins by dividing the left ventricle into thirds along the major axis from base to apex (Fig. 4.48). The most basal third of the left ventricle extends from the atrioventricular groove to the tip of the papillary muscles. The middle third is identified as that portion of the left ventricle containing the papillary muscles, and the apical third begins at the base of the papillary muscle and extends to the apex. The society also identifies the left ventricular outflow tract as the area extending from the free edge of the anterior mitral leaflet to the aortic valve annulus.

The next step is to divide each region into segments around the circumference of the minor axis. The basal and mid thirds are customarily divided into six segments each, and the apical region is divided into four segments, as illustrated in Figure 4.49. The result is the creation of 16 segments that comprise the left ventricle. The rationale for this approach was intended to reconcile the short-axis planes at each level with the three corresponding longitudinal views: the parasternal long-axis, the apical four-chamber, and the apical two-chamber views. In addition, this segmentation approach was intended to acknowledge the importance of coronary artery anatomy to wall motion analysis. As is discussed in Chapter 15, this scheme provided a logical and rational correlation between coronary distribution and left ventricular segmentation.
FIGURE 4.47. For orientation of transducer position, most ultrasound manufacturers provide an index mark along one side of the transducer. It is conventional that this index mark be located on the transducer to indicate that edge of the image that will appear to the right side of the display screen (arrow).

Table 4.3  TWO-DIMENSIONAL ECHOCARDIOGRAPHIC MEASUREMENTS

<table>
<thead>
<tr>
<th>Direct Measurement</th>
<th>Derived Data</th>
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<tbody>
<tr>
<td></td>
<td>Fractional shortening (%)</td>
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<td></td>
<td>LV mass</td>
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<td></td>
<td>Stroke volume, aortic valve area</td>
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As is seen in Figure 4.49, one practical advantage of this approach is that each segment can be visualized in both a long-axis and a corresponding short-axis projection. Using three short-axis planes (one corresponding to each of the thirds of the left ventricle) and the three longitudinal projections, a total of six basal, six mid, and four apical segments are recorded. Thus, whether one assesses the left ventricular segments from a series of three short-axis planes or three longitudinal projections, the total number of segments and their interrelationships are preserved. This occurs because the parasternal long-axis view does not visualize the apex, thereby accounting for the fact that there are only four segments in the apical short-axis projection. Even so, the apex is relatively overrepresented in this scheme. This is commonly referred to as the 16-segment model and has become the standard approach for assessing regional left ventricular function and wall motion analysis.
FIGURE 4.48. To define the left ventricular segments, it is first necessary to divide the left ventricle into apical, mid, and basal thirds, as shown in the schematic.
Several years ago, in an attempt to standardize terminology among the various imaging modalities and to improve consistency with respect to left ventricular segmentation, a task force representing various organizations has recommended a 17-segment model of the left ventricle. This document (Cerqueira et al., 2002) addresses nomenclature and segmentation in an effort
to reconcile differences among echocardiography, nuclear imaging, and the newer cardiac modalities such as computed tomography, magnetic resonance imaging, and positron emission tomography. The major recommendation of this document was to identify the apex as a separate (the 17th) segment (Fig. 4.50).

**M-MODE EXAMINATION**

With the development of 2D and Doppler echocardiography, the M-mode examination has been subjugated to a supporting role. Although it is rarely, if ever, performed as a freestanding study, the ancillary information provided by M-mode echocardiography still has a role to play in many clinical situations. To obtain an M-mode image, a single raster line from the 2D image is selected and displayed. Distance, or depth, is displayed along the vertical axis and time along the horizontal axis. One of the strengths of M-mode echocardiography is the very high temporal resolution that it provides. This yields a very rapid sampling rate and affords the ability to record subtle and/or high-frequency motion.

Figure 4.51 demonstrates four M-mode positions that can be obtained from the parasternal window. In each case, the ultrasound first penetrates the chest wall, then the right ventricular cavity, and finally into the left heart structures. Depending on the level selected, different left heart structures are recorded, from apex to base in the figure. Because of the high rate of sampling, some rapidly moving structures may be optimally imaged with this technique. For example, abnormalities of interventricular septal motion, such as those due to left bundle branch block, right ventricular volume overload, or other abnormal right ventricular filling patterns, can be readily demonstrated. Subtle abnormalities of mitral valve motion can be seen only using the M-mode technique. These include the fine fluttering associated with aortic regurgitation and the B bump caused by elevated left ventricular diastolic pressure. Figure 4.52 demonstrates a B bump from a patient with dilated cardiomyopathy. The subtlety and brief duration of the B bump make it impossible to appreciate with 2D imaging. Abnormalities of posterior wall and/or interventricular septal motion, as would occur in patients with constrictive pericarditis, can also be detected.

It is clear that M-mode echocardiography has a limited role to play in the modern comprehensive echocardiographic examination. However, several specific clinical situations are optimally assessed using this modality. For
example, the early diastolic right ventricular free wall collapse that occurs in patients with tamponade is best recorded using an M-mode view that simultaneously demonstrates right ventricular free wall motion and motion of one of the cardiac valves. Including valve motion in the scan allows precise timing and identification of early diastole. If the right ventricular wall motion is collapsing during this time, evidence of a hemodynamically significant effusion has been demonstrated.

Another important application of M-mode echocardiography involves the study of hypertrophic cardiomyopathy. Several of the subtle hemodynamic abnormalities of this condition, such as partial midsystolic closure of the aortic valve due to subvalvular obstruction and systolic anterior motion of the mitral valve, are demonstrated best using M-mode echocardiography. Partial midsystolic closure of the aortic valve may be the best way to differentiate subvalvular from valvular aortic stenosis (Fig. 4.53). M-mode also provides unique information on the pulmonary valve. An example of a pulmonary valve M-mode echocardiogram is shown in Figure 4.54. The small letters indicate the various motions of a normal pulmonary leaflet. For example, the downward motion labeled “a” corresponds to atrial contraction and corresponds to the A-wave of mitral valve Doppler inflow. One of the earliest echocardiographic signs associated with valvular pulmonary stenosis was an exaggerated A-wave. Another finding, midsystolic notching of pulmonary valve echocardiography, is indicative of pulmonary hypertension. This was a valuable finding before the availability of Doppler imaging and was often the only echocardiographic indication of elevated pulmonary artery pressure. More recently, the accurate quantitative information provided by the Doppler approach has relegated this M-mode application to historic interest only.
FIGURE 4.51. With a transducer (T), placed on the chest wall (CW) in the parasternal window, a variety of possible M-mode views can be recorded. See text for details. ARV, anterior right ventricular wall; AMV, anterior mitral valve leaflet; PMV, posterior mitral valve leaflet; PLV, posterior left ventricular wall; PPM, posterior papillary muscle; S, sternum. (Reprinted with permission from Feigenbaum H. Clinical applications of echocardiography. Prog Cardiovasc Dis 1972;14:531–558. Copyright © 1972 Elsevier.)
FIGURE 4.52. M-mode recording at the level of the mitral valve. A B bump is indicated by the arrows. PW, posterior wall.
Although less important now than in the past, one of the earliest advantages of M-mode echocardiography was its use for quantifying chamber sizes and function. Much of this has now been supplanted by 2D echocardiography, which provides better spatial orientation for proper alignment of the measurements. In some laboratories, M-mode measurements are still performed, particularly the measurements of chamber dimension, left ventricular wall thickness, and left ventricular fractional shortening. Several other specific applications of M-mode echocardiography continue to play a role in the practice of echocardiography. These are discussed in the respective chapters dealing with valvular and congenital heart diseases.
Although TEE has become an integral part of echocardiography, it is most often performed as a separate examination. Since becoming popular in the late 1980s, TEE has changed the diagnostic approach to several cardiovascular diseases. It is complementary to TTE in some situations (such as in the evaluation of infective endocarditis) and has clearly supplanted the transthoracic approach in others (such as the detection of left atrial thrombi or the assessment of aortic dissection). Today, approximately 5% to 10% of all echocardiographic studies are transesophageal. Guidelines for performing a transesophageal examination, including recommended views, were recently
The clinical success of TEE is the result of several factors. First, the close proximity of the esophagus to the posterior wall of the heart makes this approach ideal for examining several important structures. The closeness and absence of intervening tissues, such as bone or lung, allow the use of high-frequency transducers and ensure high-quality imaging in most patients. Second, the ability to position the transducer in the esophagus or stomach for extended periods provides an opportunity to monitor the heart over time, such as during cardiac surgery. Third, although more invasive than other forms of echocardiography, the technique has proven to be extremely safe and well tolerated so that it can be performed in critically ill patients and very small infants. As a result, TEE provides unique and important diagnostic data in a variety of clinical settings (see Table 4.4).

TEE has proven to be a safe and generally well-tolerated procedure. Because of the semi-invasive nature of the procedure and the unusual views that can potentially be recorded, special training is required of the operator as well as the nurse monitor. TEE is essentially a form of upper endoscopy. Complications are rare but include aspiration, arrhythmia, perforation of the esophagus, laryngospasm, and hematemesis. Complications, such as hypotension, hypertension, or hypoxia (see later), may also arise from the effects of the medications that are administered as part of the examination. Death can occur but is very rare.

<table>
<thead>
<tr>
<th>Table 4.4</th>
<th>UNIQUE DATA FROM TRANSESOPHAGEAL ECHOCARDIOGRAPHY</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Atrial thrombi/masses</strong></td>
<td></td>
</tr>
<tr>
<td>- Left atrial appendage clot</td>
<td></td>
</tr>
<tr>
<td>- Left atrial appendage spontaneous contrast</td>
<td></td>
</tr>
<tr>
<td>- Clot in body of left atrium</td>
<td></td>
</tr>
<tr>
<td>- Right atrial thrombus</td>
<td></td>
</tr>
<tr>
<td>- Thrombus/mass on pacemaker wire or indwelling catheter</td>
<td></td>
</tr>
<tr>
<td><strong>Mitral valve</strong></td>
<td></td>
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</tbody>
</table>
Precise mechanism of mitral regurgitation
Refined suitability for valvotomy in severe mitral stenosis
Define eccentric jets
Function of prosthetic valves
Paravalvar regurgitation

**Aorta**
- Detection/characterization of dissection
- Detection of atheroma

**Aortic trauma/transection**

**Chambers**
- Refinement of patent foramen ovale characteristics

**Online monitoring**
- Intraoperative left ventricular size/function
- Monitoring interventional procedures
  - Atrial septostomy
  - Balloon valvotomy
  - Pulmonary vein/left atrial interventions
  - Percutaneous aortic and mitral valve replacement or repair

**Endocarditis**
- Detect aortic abscess
- Identify smaller vegetations
- Detect valve perforation
- Detect endocarditis of prosthetic valves

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### Table 4.5 RISKS AND CONTRAINDICATIONS FOR TRANSESOPHAGEAL ECHOCARDIOGRAPHY

<table>
<thead>
<tr>
<th><strong>Topical anesthesia</strong></th>
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<tbody>
<tr>
<td>Allergic reactions</td>
<td>Toxic methemoglobinemia</td>
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<table>
<thead>
<tr>
<th><strong>Conscious sedation</strong></th>
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<tr>
<td>Respiratory compromise/hypoxia</td>
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<tr>
<td>Hypotension</td>
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<tr>
<td>Paradoxical hypertension</td>
<td></td>
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<tr>
<td>Paradoxical agitation</td>
<td></td>
</tr>
<tr>
<td>Idiosyncratic reactions</td>
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<table>
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<tr>
<th><strong>Probe insertion: immediate</strong></th>
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<tbody>
<tr>
<td>Oral trauma</td>
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<tr>
<td>Dental trauma</td>
<td></td>
</tr>
<tr>
<td>Esophageal perforation</td>
<td></td>
</tr>
<tr>
<td>Vagal reaction</td>
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<table>
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<tr>
<th><strong>Probe insertion: delayed</strong></th>
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<tbody>
<tr>
<td>Aspiration</td>
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Preparation of the patient is critical to a successful procedure. A list of risks and contraindications to TEE is provided in Table 4.5. First, the patient should be thoroughly informed about the indications and procedure. Informed consent should be obtained. The patient should fast for at least 4 to 6 hours before undergoing TEE. Any history of dysphagia or other forms of esophageal abnormalities should be sought and evaluated prior to the procedure. All patients should have intravenous access and both supplemental oxygen and suction should be available in all cases. Before intubation, the use of a topical anesthetic to numb the posterior pharynx is recommended. Either lidocaine or Cetacaine is typically used for this purpose. Although safe and well tolerated, rare cases of toxic methemoglobinemia have been reported and should be considered whenever significant oxygen desaturation complicates the procedure. Treatment of this condition is intravenous administration of methylene blue, usually given in a dose of 1 mg/kg as a 1% solution over 5 minutes. Various intravenous agents are also frequently used for conscious sedation, for pain prevention, and as an anxiolytic. The combination of midazolam and fentanyl is popular in many laboratories. Bacteremia induced by upper endoscopy during TEE is very rare. Although such decisions should always be made on an individual basis, the routine use of antibiotic prophylaxis has generally been abandoned.

To perform the procedure, the patient is placed in the left lateral decubitus position (Fig. 4.55). The head of the bed is elevated approximately 30 degrees to improve comfort and help avoid aspiration. If the patient has dentures, these should be removed, and in most patients, a bite block is placed between the teeth to prevent damage to the probe. After the probe has been lubricated with surgical jelly, it is introduced into the oropharynx and gradually advanced while the patient is urged to “swallow” to facilitate
intubation. Once the probe has passed into the esophagus, a complete examination can usually be performed in 10 to 30 minutes. During this time, monitoring by a nurse is considered the standard of care. Special attention should be paid to the patient’s blood pressure, heart rate and rhythm, and oxygen saturation. Suctioning of the oropharynx is often required, and additional intravenous medications may be needed to maintain the proper level of conscious sedation and comfort.

Early transesophageal echocardiographic transducers were capable of imaging from only one tomographic plane in the transverse orientation and were called monoplane devices. The second-generation instruments had biplane capability and were able to record images in both the transverse and longitudinal orientations (Fig. 4.56). Using these transducers, the various transesophageal views were obtained by moving the transducer to various levels of the esophagus and stomach and by flexing the tip of the transducer via hand controls on the device. All current-generation transesophageal echocardiographic transducers have multiplane capability (Fig 4.57). The
image is rotated, either electronically or mechanically, around a 180-degree arc to yield an infinite number of possible imaging planes. This development not only increased the number of planes that could be recorded but reduced the need for extreme flexion of the transducer tip to record all necessary information. The availability of multiplane imaging effectively provides a 360-degree “panoramic” reconstruction of the heart and great vessels. All ultrasound modalities, including Doppler and 3D data, can be obtained using this technique. The probes operate at multiple frequencies, typically 3.5 to 7.0 MHz. Pediatric probes are smaller in diameter (5 to 7 mm) and operate at higher frequencies (5 to 10 MHz). Transesophageal probes capable of real-time 3D imaging have also been developed. This technology combines the image quality of the transesophageal approach with the spatial advantages of 3D imaging.

**FIGURE 4.56.** A biplane transesophageal echocardiogram. **A:** The long-axis view of the left ventricle with the transducer located in the patient’s stomach. **B:** The orthogonal view demonstrating a short-axis plane. TEE, transesophageal echocardiography.
FIGURE 4.57. A multiplane transesophageal probe and a bite block are shown.

TRANSESOPHAGEAL ECHOCARDIOGRAPHIC VIEWS

TEE does not lend itself to standardization of views as readily as TTE. As with any technique, a clear awareness of potential pitfalls and normal variants is essential. Because the examination is often oriented toward answering a
specific question or making a particular diagnosis, care must be taken to perform a thorough assessment and avoid missing important ancillary findings. The targeted nature of the test, together with the constraints imposed by the esophagus and its relation to the heart, limit our ability to define and describe standard views using this modality. Despite these limitations, some degree of standardization is both appropriate and beneficial to ensure a complete and comprehensive examination. This is accomplished by advancing the probe to the level of the superior portion of the left atrium and then recording a series of transverse and longitudinal views at sufficient levels to provide a comprehensive assessment of the entire heart.

A useful starting point is the four-chamber view, which is recorded with the transducer positioned immediately superior and posterior to the left atrium and flexed in a way to provide a long-axis plane through all four chambers (Fig. 4.58). Because of the relationship between the heart and esophagus, a true long-axis plane is often difficult to achieve. By adjusting the degree of flexion, variations on the four-chamber view are recorded (Fig. 4.59). This transesophageal echocardiographic view provides similar information to the corresponding transthoracic view, seen from the opposite direction. Each modality has its advantages and limitations. For example, the transthoracic four-chamber view places the left ventricular apex in the near field and is ideally suited to detect apical thrombi. In contrast, the transesophageal four-chamber view places the left atrium in the near field and is ideally suited for assessing left atrial and mitral valve pathology.
FIGURE 4.58. A four-chamber view from the mid-esophagus is displayed, illustrating the close proximity of the esophagus to the LA. (Video 4-58)
By anteflexing the probe tip, the long-axis orientation can be gradually converted into a more short-axis view for the evaluation of the left ventricular outflow tract and aortic valve (Fig. 4.60). This view is similar to a parasternal basal short-axis view obtained from the chest wall. By gently flexing and relaxing the probe, the aortic root, aortic valve, and left ventricular outflow tract can be thoroughly assessed (Fig. 4.61). By rotating the array angle from 0 degrees (transverse) to approximately 90 degrees, a two-chamber view can be obtained (Fig. 4.62). Further angle rotation, to approximately 135 degrees, will approximate a left ventricular long-axis view (Fig. 4.63). This plane is closely aligned to the long axis of the heart and provides an excellent assessment of the aortic valve and aortic root. Rotation of the probe clockwise will sweep the imaging plane toward the right heart, eventually recording the bicaval view in which the right atrium, and inferior and superior vena cava are visualized (Fig. 4.64). This view also provides a thorough assessment of the atrial septum and is especially helpful to interrogate the superior portion of the atrial septum for sinus venosus defects. By slight angulation, the right atrial appendage can also be examined from this approach (Fig. 4.65).
FIGURE 4.59. Three of the echocardiographic views that can be obtained with the horizontal probe in the midesophageal location. LPA, left pulmonary artery; RPA, right pulmonary artery; S, stomach; PV, pulmonary vein; CS, coronary sinus.
FIGURE 4.60. From the esophagus, the probe can be flexed to yield a basal short-axis projection. [Video 4-60]
FIGURE 4.61. Four of the short-axis views that can be obtained with the horizontal probe in the upper esophagus. S, stomach; LUPV, left upper pulmonary vein; RUPV, right upper pulmonary vein; LAA, left atrial appendage; RAA, right atrial appendage; RLPV, right lower pulmonary vein; LLPV, left lower pulmonary vein; LCA, left coronary artery; RCA, right coronary artery; FO, foramen ovale; N, noncoronary cusp; R, right coronary cusp.
FIGURE 4.62. By adjusting the array angle to approximately 90 degrees, a two-chamber view is recorded.

Video 4-62

The left atrial appendage, a frequent target of TEE, can be visualized in several of the views just described. Because the appendage is often
multilobed and varies considerably in size and shape, it should be assessed from multiple angles to ensure complete interrogation (Fig. 4.66). By withdrawing the probe slightly and adjusting to a more horizontal plane (approximately 0 degrees), the bifurcation of the main pulmonary artery can be visualized adjacent to the ascending aorta (Fig. 4.67). The thoracic aorta, another structure uniquely suited to transesophageal echocardiographic inspection, lies in close proximity to the esophagus and on the opposite side from the heart. With the array angle at 0 degrees, the transducer itself is rotated 180 degrees to view the aorta in short axis. Beginning distally, gradual withdrawal of the transducer will follow the descending aorta in a retrograde manner up toward the arch (Fig. 4.68). Some degree of rotation is often required to maintain visualization, but the entire course of the vessel can generally be recorded. At any point, adjusting the array angle to a vertical plane will provide a corresponding longitudinal view. At the level of the aortic arch, the origin of the branch vessels can be recorded (Fig. 4.69). Then, by rotating the transducer and gradually advancing the probe further into the esophagus, a portion of the ascending aorta can be recorded. Because of the interposition of the trachea, some portion of the ascending aorta will not be seen in most patients. These series of views provide an excellent opportunity to detect aortic aneurysm, dissection, and atherosclerosis.
FIGURE 4.63. By increasing the array angle to approximately 130 degrees, the left ventricular outflow tract, aortic valve, and proximal aorta can be included in the plane, yielding a long-axis view.
FIGURE 4.64. With the probe relatively high within the esophagus, a vertical plane allows both atria and the interatrial septum to be recorded. This plane is called the bicaval view and also records the entrance of the superior vena cava (SVC) into the RA.

The junction of the four pulmonary veins and the posterior wall of the left atrium can often be visualized with TEE. To record the left pulmonary veins, the transducer angle is adjusted to approximately 100 degrees and the transducer is rotated to the far leftward plane (counterclockwise rotation of the probe). Color flow imaging can be used to assist in locating the mouth of the veins. The two left veins drain into the left atrium in close proximity to each other, and the left upper pulmonary vein is often recorded adjacent to the left atrial appendage (Fig. 4.70). To record the right pulmonary veins, adjust the transducer angle from 50 to 60 degrees and rotate the probe to the patient’s far right. Again, the two veins appear to originate together, sometimes as a bifurcation. Figure 4.71 is an example of a transesophageal 3D echocardiogram of the mitral valve and left ventricle from a patient with cardiomyopathy.
The transducer can also be advanced into the patient’s stomach to provide a family of views from this unique perspective. Beginning from the transverse plane (0 degrees), extreme anteflexion and gradual withdrawal of the probe will bring the transducer in contact with the superior portion of the stomach, with the ultrasound beam directed upward toward the heart. A series of short-axis views of the ventricles can then be recorded by sequential anteflexion and retroflexion to visualize the various levels of short-axis planes (Fig. 4.72). Often, some angle adjustment is required to optimize the true short-axis view. Then, by increasing the array angle to a more vertical plane, a long-axis view is recorded, often providing excellent visualization of the left ventricular outflow tract and aortic valve.

**FIGURE 4.65.** From the vertical plane, the right atrial appendage (*) can be visualized (A). By slight adjustment of the probe (B), the multilobed shape of the structure is demonstrated. [Video 4-65](Video 4-65)
FIGURE 4.66. Five views (A–E) of the left atrial appendage are recorded at various angles of rotation of the scan plane. Note how the appearance of the appendage varies in the different views. The relationship of the appendage to the left upper pulmonary vein (*) is also demonstrated.
FIGURE 4.67. From a high esophageal position, the horizontal plane will permit the relationship between the main pulmonary artery (MPA) and aorta to be recorded. This view also allows the bifurcation of the MPA into the right pulmonary artery (RPA) and left pulmonary artery (LPA) to be demonstrated. The ascending aorta is shown in cross section.

THREE-DIMENSIONAL ECHOCARDIOGRAPHY

The inherent 3D nature of the heart has, for many years, fueled a desire to collect and display cardiac images in three dimensions. It has only been in the past decade, however, that improvements in transducer design, engineering, and computer power have allowed a robust and clinically useful approach to this challenging problem. These technical developments have reduced transducer size, shortened acquisition time, improved image quality, and allowed for better display options.

The major difference between 2D and 3D ultrasound begins with the transducer. Unlike 2D transducers, in which the piezoelectric crystals are arranged linearly (in a row), in 3D probes, the individual elements are
arranged in a rectangular grid just beneath the surface of the transducer (Fig. 4.73). Each piezoelectric crystal can be activated separately and the phasing of activation is electronically controlled. As the ultrasound beam propagates away from the transducer, it can be steered simultaneously in both the x and y planes. Through sophisticated beamforming technology, a staggering amount of ultrasound energy can be simultaneously transmitted and received over a pyramid-shaped region, with the transducer located at the apex of the pyramid. Current-generation transducers consist of approximately 3,000 piezoelectric elements in the rectangular grid. A major advance in recent years has been the ability to construct fully sampled 3D transducers. If each element is utilized for the formation of the ultrasound image, the array is considered “fully sampled,” as opposed to “sparsely sampled” if only a fraction of the crystals are utilized (Fig. 4.73).
FIGURE 4.68. The descending aorta is recorded in a series of short-axis views at various levels by withdrawing the transducer from the level of the diaphragm to the distal arch (panels A–D). In panel E, a biplane recording of the proximal thoracic aorta is shown, demonstrating short- and long-axis views.

This leads to the important concept of image resolution in 3D echocardiography. The amount of analyzable ultrasound information is a function of both temporal and spatial resolution. Remember that the fundamental limiting factor in 3D echocardiography, like all ultrasound modalities, is the speed of sound, which is approximately 1,540 m/s. Each
piezoelectric crystal is limited by the time it takes for sound to travel out and back from the target, which sets the depth (or distance) of the image. Therefore, it is the depth of imaging that determines the number of pulses that can be emitted per second.

**FIGURE 4.69.** The aortic arch can be visualized using transesophageal echocardiography. In (A), a long-axis view of the arch shows the typical curved contour. In (B), a short-axis projection shows an orthogonal view which can sometimes demonstrate the origin of one of the branch arteries (*).

The other two variables that determine the amount of ultrasound data collected (hence, the resolution) are the shape of the pyramid (often described by the angle, such as 60 degrees or 80 degrees) and the density of beam spacing (**Fig. 4.74**). Ultimately, it is the tradeoffs among these three variables that determine the resolution, and therefore the value and quality of the resulting image. A practical way to conceptualize these factors is to regard depth as a determinant of temporal resolution, also referred to as volume rate. All other things being equal, the greater the depth of imaging, the lower the volume rate. Spatial resolution depends on the shape of the pyramid and the beam density of each plane within the pyramid. The larger the pyramid (i.e., the bigger the angle), the lower the spatial resolution. Thus, the utility of the resulting data set depends on optimizing the tradeoffs between the three variables.
FIGURE 4.70. This view demonstrates the relationship between the left atrial appendage (*) and the left upper pulmonary vein (LUPV) from the two-chamber view.  

Video 4-70
Recognizing these limiting factors that combine to affect resolution and volume rate, several techniques have been developed to optimize image quality and utility of 3D echocardiography. Some of these, such as multiline beamforming, are technical advances within the transducer and/or beamformer that maximize the amount of ultrasound information that is received and processed to produce each volume of data. Other techniques, such as multibeat acquisition, involve acquisition of a portion of the pyramid from each of 3 or 4 consecutive beats which are then stitched together to assemble the full pyramid (Fig. 4.75). This allows a higher volume rate for any level of spatial resolution parameters.

A fully sampled 3D data set results in a volume of echocardiographic data, including cardiac and extra cardiac structures, and typically, prior to processing, appears as an indistinguishable pyramidal mass of ultrasound signature. Processing the 3D data set, in order to yield useful diagnostic information, can be undertaken in multiple. At least three approaches to acquisition and display are currently available. Perhaps the simplest to understand is multiplane imaging (Figs. 4.76 and 4.77). In this mode, multiple high-resolution 2D images are acquired and displayed. The planes can be predetermined or user selected and often orthogonal planes are chosen to display a structure simultaneously from two different angles, but acquired from the same cardiac cycle. The images can be adjusted, or fine-tuned, to optimize visualization of specific areas of interest, such as the mitral leaflets in mitral valve prolapse (Fig. 4.78). Alternatively, a series of parallel short-axis planes can be obtained and displayed (Fig. 4.79). This may be particularly useful for analysis of left ventricular wall motion and function, including during stress echocardiography. Thus multiplane imaging is a form of “real-time” 3D echocardiography, with the additional capability of off-line post-processing of the data set.
FIGURE 4.71. An example of a transesophageal three-dimensional echocardiogram. The MV and left ventricle are shown. A portion of the left ventricular wall is removed to show the cavity of the chamber.

Video 4-71
FIGURE 4.72. Transgastric imaging demonstrates a short-axis view at the mid left ventricular level.

FIGURE 4.73. The piezoelectric crystals in an ultrasound transducer can be arranged linearly (left panel), or in a two-dimensional array. The middle panel demonstrates a sparsely sampled matrix array in which half of the elements are...
active. In the right panel, a fully sampled array, in which all elements are active, is shown. See text for details.

FIGURE 4.74. The fundamental difference between two-dimensional and three-dimensional scanning is illustrated. On the left, a phased array probe creates a 2D sector scan. On the right, the third dimension is added to create a volumetric data set, from which real-time 3D imaging is derived. See text for details.
A second option is multibeat imaging. In this mode, a portion (or thick slice) of the pyramidal data set is acquired sequentially from 3 or 4 consecutive cardiac cycles, using ECG gating (see Fig. 4.75). Once acquired, the individual slices are reconstructed, or stitched, into a fully aligned pyramid of ultrasound information, including color Doppler. As such, it is not considered “real time” and the resulting images are not able to be manipulated once acquired. The main advantage of this mode is that it allows a larger volume of data to be assembled at a higher volume rate than would otherwise be possible.
FIGURE 4.76. Schematic demonstrates various methods for processing real-time three-dimensional full-volume data sets. A: Schematic represents the total volume in a pyramidal scan that contains all four cardiac chambers. Note that distinct cardiac structures are not visualized. B: A “cropped” full-volume data set in which the full-volume data set has been “sliced” through its midpoint. This effectively exposes the middle of the 3D data set in the cropped plane. In this example, this results in a 3D image of an apical four-chamber view. C: Three images represent extracted two-dimensional images from the full-volume data set. Each has been created by extracting a discrete imaging plane along either the long- (A) or short-axis (B and C) plane of the 3D data set. In reality, an infinite number of imaging planes could be selected from the 3D data set, resulting in creation of any of the traditional or nontraditional 2D imaging planes.

The third approach is true real-time 3D echocardiography, sometimes called live 3D. In this mode, a full-volume, pyramidal-shaped data set is acquired from a single cardiac cycle and viewed in real time, very similar to
2D recording (Fig. 4.80). An advantage of this method is that the image can be fully manipulated while scanning to obtain the desired diagnostic information. In theory, the entire heart can be captured in a single acquisition, without the degrading effects of respiratory motion or arrhythmia. A disadvantage is that sheer amount of ultrasound data being gathered will limit both volume rate and spatial resolution.

Real-time 3D echocardiography has several applications. For example, full-volume acquisition (90 degrees × 90 degrees) can be used for determining, either automatically or manually, chamber volume, especially the left ventricle (Fig. 4.81). Assuming acceptable endocardial definition, this approach has been shown to be the most accurate echocardiographic method for measuring left ventricular ejection fraction and may be particularly useful in patients with arrhythmias, such as atrial fibrillation, where beat-to-beat variability is a problem.

FIGURE 4.77. Multiplane imaging involves the simultaneous recording of (in this case) three two-dimensional images, from which a volumetric reconstruction of the left ventricle is created. Volumes and ejection fraction can then be calculated.
Another application uses live 3D to interrogate specific intracardiac structures in real time. This capability can be especially useful during structural heart interventions (Fig. 4.82). By switching back and forth between 2D and 3D imaging, sometimes incorporating color Doppler, unique and valuable diagnostic information can be obtained in real time. In most 3D echocardiographic systems, a zoom feature permits more targeted viewing of the desired structure, in order to optimize resolution and detail (Fig. 4.83).

A key requirement of live 3D is the ability to manipulate the pyramid of ultrasound data which, when unprocessed, is a featureless blob with little utility. In order to visualize desired intracardiac anatomy, the outside layers must be peeled away, a process called “cropping.” Current echocardiographic systems allow this to be done both in real time, that is, during acquisition, or at a later time (Fig. 4.84). Cropping not only allows the removal of undesirable external structures but also the rotation and reorientation of the image. This allows the viewing perspective to be tailored to the clinical situation. For example, in the operating room, display of the mitral valve can be adjusted to provide the surgeon with a view of the valve from the perspective of the left atrium, looking “down” at the structure, as he or she would see it during the surgery (Fig. 4.85). Live 3D has also become virtually indispensable during most structural heart procedures and many of the interventions involving patients with congenital heart diseases.
FIGURE 4.78. Once a three-dimensional data set is recorded, the displayed planes can be manipulated to show a specific area of interest. In this case, a 3D display of a prolapsed mitral valve (arrow) is shown, along with the corresponding two-dimensional planes. 

Video 4-78

coming soon
FIGURE 4.79. Series of nine equidistant short-axis images automatically extracted from a three-dimensional data set. Moving left-to-right and top-to-bottom are recorded at the apex through the papillary muscle and, finally, mitral valve level. Note that in the real-time image, wall motion can be accurately analyzed at each of these imaging planes in real time. Video 4-79
FIGURE 4.80. Using live 3D, a dehisced mitral ring from a patient with endocarditis is displayed. The ring is shown from the perspective of the left atrium. A large region of dehiscence is seen superior to the ring, with the large arrow demonstrating an intact suture. The small arrowhead points to a vegetation.

Video 4-80

A frequently cited limitation of 3D echocardiography is that it simply
provides a series of 2D imaging planes which could theoretically be obtained by a comprehensive 2D examination. In reality, 3D echocardiography has the advantage of ensuring precise alignment of a 2D interrogation plane in atypical orientations. It also provides a more complete spatial orientation. This may be very useful for localization of ventricular septal defects, assessment of the size of an atrial septal defect, and defining the atrial septal rim which has implications for percutaneous closure. This is done by creating imaging planes that are not feasible using a single 2D plane such as an en face view of the atrial septum for visualization of the fossa ovalis or an atrial septal defect. A major advantage is in the evaluation of complex congenital abnormalities. 3D echocardiography has also shown superiority in assessing the mechanism of mitral regurgitation and in precise identification of flail leaflets. Another area in which reconstructed 3D echocardiography has shown substantial promise is in evaluating prosthetic valves for paravalvular regurgitation. It is frequently difficult to precisely identify the location and determine the size of a paravalvular leak, even with TEE. 3D reconstruction of the face of the annulus frequently provides superb visualization of the entire circumference of the annulus and localization of multiple paravalvular leaks. Finally, quantitative accuracy for chamber volume determination is greater for 3D than for 2D echocardiography. This advantage is most obvious when dealing with irregularly shaped chambers such as the right ventricle or abnormal left ventricles. This is discussed further in Chapter 5 on the evaluation of left ventricular size and function.

FIGURE 4.81. Real-time three-dimensional echocardiography is recorded from a patient with atrial fibrillation. On the left, five consecutive cardiac cycles are shown, with the end-diastolic (ED) left ventricular volumes on top and the corresponding end-systolic (ES) volumes directly below. On the right, the instantaneous left ventricular volumes are plotted, illustrating the beat-to-beat variability and range of ejection fraction.
FIGURE 4.82. A Mitra Clip device (arrow) is seen positioned between the two leaflets of a myxomatous mitral valve using three-dimensional echocardiography.
FIGURE 4.83. Detailed anatomic information on intracardiac pathology is possible with real-time three-dimensional echocardiography. In this case, from a patient with mitral valve prolapse, the specific prolapsing scallop (arrow) is shown clearly, along with the relationship of the mitral valve to the aorta (*).
FIGURE 4.84. A, B: Real-time three-dimensional transesophageal echocardiographic image of mitral regurgitation. B: This image can be rotated and cropped to provide a short-axis image of the actual regurgitant orifice. MR, mitral regurgitation.
Video 4-84

Additional imaging formats have been proposed for 3D echocardiography including real-time 3D holographic displays of the 3D data set. The feasibility of this approach has been demonstrated; however, the clinical availability of equipment to produce these images is limited. A final approach has been to create physical 3D models from the ultrasound data set. This relies on the technology used in plastics manufacturing and can provide a solid or hollow model of a 3D data set from cardiac images.
FIGURE 4.85. An advantage of live-3D imaging is the ability to display anatomic information during surgery or interventions. In this example, from a patient with mitral valve prolapse, the detailed anatomy of the mitral leaflets can be shown from both the left atrial and left ventricular perspective. Coming soon

Video 4-85

POINT-OF-CARE CARDIAC ULTRASOUND
Advances in technology and miniaturization now permit diagnostic quality ultrasound images to be obtained using smaller, more portable devices, including hand held. This practice involves all forms of diagnostic ultrasound. When used to evaluate the cardiovascular system, it is referred to as either point-of-care cardiac ultrasound or focused cardiac ultrasound. In this way, the term “echocardiography” is reserved for the more traditional, comprehensive assessment of the heart and great vessels. In current practice, it is usually reserved for the performance and interpretation of a bedside cardiac ultrasound examination, typically focused on a limited diagnostic issue or in follow-up to a recent echocardiogram.

Although the terminology does not specify the type of equipment utilized, it generally refers to a very small, hand-held device, as large as a laptop computer or as small as a cell phone (Fig. 4.86). As such, a range of features is possible, depending on the size of the device. Larger devices are capable of 2D, spectral, and color Doppler imaging and may allow recording and archiving of the images (Fig. 4.87). Smaller hand-held systems may offer only 2D imaging, possibly with color Doppler and may or may not permit the study to be saved or recorded. These are sometimes referred to as ultrasound stethoscopes, and are intended to provide immediate (although limited) ultrasound information that is used by the practitioner in the ongoing care of a patient. For example, they may be used following an invasive procedure to exclude a pericardial effusion, or in a hypotensive ICU patient for a rapid assessment of left ventricular contractility.

The most recent generation of point-of-care systems offers image quality that approaches, but does not quite match a modern, full-sized echocardiography system. Naturally, difficult to image patients will be even more challenging with a smaller, less powerful hand-held device. This suggests that the acquisition and interpretation of point-of-care ultrasound requires as much training and experience as a standard echocardiogram. However, if point-of-care cardiac ultrasound is limited to a smaller number of straightforward diagnoses, such as pericardial effusion, a more targeted training experience may be sufficient. Several professional societies, including the American Society of Echocardiography, have now developed and published guidelines for training in point-of-care ultrasound (Spencer et al., 2013).
FIGURE 4.86. One example of ultrasound miniaturization is shown. A transducer, containing all the necessary image-forming technology is interfaced with a smartphone. Two-dimensional and color Doppler images can be recorded and stored on the phone or tablet.

FIGURE 4.87. These images (A, B) from a patient with severe mitral regurgitation were recorded using the point-of-care device shown in Figure 4.86.
Point-of-care cardiac ultrasound has several advantages and attractive features, besides its much lower cost. In a teaching environment, the technology offers an excellent opportunity to demonstrate the correlation between physical diagnosis and echocardiographic findings. It is often used on rounds in a teaching hospital to demonstrate visually what is suspected from the history and physical. In the Emergency Department or Intensive Care Unit, it offers a rapid and accurate approach to a limited cardiac assessment in a critically ill or unstable patient. In the invasive laboratory, it permits focused questions, such as the presence or absence of an effusion, to be answered quickly and accurately.

It seems likely that this technology will continue to improve and
disseminate beyond the practice of cardiology. As it does, more and more groups will take an interest in the rules that govern its use. It will be important to develop a policy for utilization and training guidelines that all groups can agree on and adhere to. Point-of-care cardiac ultrasound is not a replacement for a complete echocardiogram. Its role in the practice of clinical medicine is still being defined.

ECHOCARDIOGRAPHY AS A SCREENING TEST

The availability, utility, and noninvasive nature of echocardiography have fostered its popularity as a diagnostic test. In this chapter, an approach to the echocardiographic examination that yields accurate and potentially important information in a variety of clinical situations is described. When properly applied, the diagnostic and prognostic utility of echocardiography is unmatched. As with any procedure, however, the potential for overuse exists and the decision to perform an echocardiogram must always be balanced by the expected value of the results. These should be judged in terms of the anticipated impact of the diagnostic data, the likelihood that the results will alter management, or the prognostic value of the results to reassure or persuade a patient in a given situation. Thus, the more specific and targeted the question being asked, the more likely it is that the test will provide useful new information. If applied too widely, the yield of the test will be offset by the cost and potential for misleading information.

When used as a screening tool, the benefits of echocardiography depend on the specific situation, some of which are listed in Table 4.6. It should be clear from the preceding discussion that echocardiography is not an appropriate test to screen the general population for heart disease. Although some important positive results might be found, the low yield and the potential for false-positive findings argue against this approach. In other situations, however, screening with echocardiography is clearly justified on the basis of clinical evidence. Examples include screening of athletes when the history and/or physical suggest a possible abnormality and the evaluation of first-degree relatives of patients with certain genetic cardiovascular diseases. As is always the case, the specific decision to perform the test is predicated on several factors. First, the ordering physician must understand
the expected value of the results and be able to apply the new information to the patient. The patient must be informed, both of the expected utility of the test results and the potential for inaccurate or incomplete results. Finally, the study must be performed and interpreted in an expert manner by professionals aware of the question being posed.

### Table 4.6

<table>
<thead>
<tr>
<th>INDICATIONS FOR ECHOCARDIOGRAPHY TO SCREEN FOR THE PRESENCE OF CARDIOVASCULAR DISEASE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class</td>
</tr>
<tr>
<td>1. Patients with a family history of genetically transmitted cardiovascular disease</td>
</tr>
<tr>
<td>2. Potential donors for cardiac transplantation</td>
</tr>
<tr>
<td>3. Patients with phenotypic features of Marfan syndrome or related connective tissue diseases</td>
</tr>
<tr>
<td>4. Baseline and reevaluations of patients undergoing chemotherapy with cardiotoxic agents</td>
</tr>
<tr>
<td>5. First-degree relatives of patients with unexplained dilated cardiomyopathy in whom no etiology has been identified</td>
</tr>
<tr>
<td>6. Patients with systemic disease that may affect the heart</td>
</tr>
<tr>
<td>7. The general population</td>
</tr>
<tr>
<td>8. Competitive athletes without clinical evidence of heart disease</td>
</tr>
<tr>
<td>9. Routine screening echocardiogram for participation in competitive sports in patients with normal cardiovascular history, electrocardiogram, and examination</td>
</tr>
</tbody>
</table>


### THE DIGITAL ECHO LABORATORY

Cardiac ultrasound examinations generate a tremendous amount of information consisting of moving gray-scale images, color images, and static images. There has been a rapid evolution of the manner in which these images are acquired, stored, and analyzed. During the early days of
echocardiography, only M-mode tracings were available, and these were typically recorded on a strip-chart recorder and stored as light-sensitive paper output. With the advent of 2D echocardiography, the need arose to store moving video images. For approximately two decades, these images were stored using standard video technology and reviewed from videotape. Although this provides “full-disclosure” registration and review of all available information, it is an inefficient method for reviewing studies, results in the need for substantial storage and archiving space, and has the disadvantage of the degradation of video information over time. In the early 1980s, high-speed digitizing devices became available that allowed analog data to be converted to a digital format and stored as such. The limitation of these early attempts was the relatively slow speed of computer processors and the limitations in both speed and cost of computer memory. Over time, there has been a dramatic reduction in cost and improvement in speed and reliability, so that storage of large amounts of digital information is now within the reach of virtually all echocardiography laboratories. Finally, all modern ultrasound scanners are intrinsically “digital” platforms. The information collected from the transducer face is done so in a digital format, processed as digital information, and displayed by a computer processor as digital information. It is converted to analog format only for purposes of recording on videotape. As such, the original, nondegraded digital images are available for both review and storage if the appropriate offline systems are made available.

Digital archiving systems are now available from a number of ultrasound and third-party vendors, all of which are capable of providing transfer from current-generation ultrasound platforms to a file server (and subsequently to bulk storage devices) and retrievable through standard computer workstations. A major breakthrough in digital medical imaging came with the advent of the DICOM standard (Digital Imaging and COmmunication in Medicine). The DICOM standard was negotiated among major medical manufacturers, regulatory bodies, and professional organizations in an effort to ensure that medical imaging information would be available in a standardized nonproprietary format so that it could be easily transferred from institution to institution and platform to platform for storage and analysis. Virtually all current-generation ultrasound platforms provide output of echocardiographic images in a DICOM-compatible format that can be
archived to and retrieved from offline analysis systems. The DICOM committee has also standardized image formats and recommended standards for image compression. Because a complete ultrasound examination may include 30 to 100 image clips and static images, it represents a substantial file size. In an effort to reduce file size, image clips are typically compressed. Compression can be either lossless (no information lost) or lossy, with the potential for image degradation. The DICOM committee has recommended motion JPEG, which provides as much as 20:1 compression as the accepted compression method of video images.

The modern digital echocardiography laboratory has several components as noted in Figure 4.88. In a typical installation, ultrasound platforms are connected to a file server by either a local area network (LAN) or Internet connection. Depending on the size of the laboratory, the “file server” may be an enhanced desktop computer or a standard file server with a high-speed hard drive capacity of several hundred gigabytes. In either instance, the ultrasound platform can be configured to either automatically or on command transmit the DICOM format ultrasound images to the file server for storage. Reading stations typically are desktop personal computers that can retrieve images for analysis and report generation. Because all information is digital and communicated over a computer network, echocardiograms recorded in remote sites can be transmitted almost instantaneously to the file server for review at the interpreting laboratory. Additionally, physicians can retrieve images at any appropriately equipped desktop computer, including physician offices, hospital ward stations, and catheterization laboratories.

The method for long-term storage of digital information is currently in evolution and can result in substantially more expense than the initial outlay for a basic digital echocardiography laboratory. Most hospital and regulatory agency stipulations require that medical information be stored with several layers of redundancy, and, as such, a “purely digital” laboratory must be prepared to provide storage at a minimum of two separate sites along with tape backup to ensure guaranteed access to medical information, even in the event of catastrophic failure at one site. In view of this, many laboratories that rely heavily on digital imaging still record standard analog videotape as a means of long-term archiving for regulatory purposes.

In migrating to a digital echo laboratory, it is important to recognize that even with the enhanced speed and availability of large-scale memory, it is
still not feasible to record 20 to 40 minutes of continuous video information in digital format for high-volume laboratories. In view of this, the digital laboratory must develop a protocol for acquisition of select images that may be specific to the clinical presentation or disease being investigated. Typically, a full digital echocardiographic examination will comprise 30 to 100 individual clips consisting of real-time 2D imaging, color Doppler flow imaging, and static images of M-mode echocardiography, spectral Doppler display, and so on. The precise images to be acquired are a matter of individual laboratory preference and policy, but, as a general rule, it should be possible to acquire representative clips of each of the standard views typically required during videotaping of an echocardiogram within the stipulated 30- to 100-image volume.

Once digitally acquired and archived, the images can be retrieved from any appropriately equipped computer including standard office desktops, laptops, and hospital workstations. A typical full-function workstation has the ability to retrieve and review studies, make multiple measurements, and generate reports. Additionally, in a fully digital hospital environment, it is anticipated that all other medical images including catheterization data, results of nuclear medicine studies, and electrocardiograms, will also be available in a DICOM format. Most vendors of digital archiving and review stations provide access for non-echocardiographic images. The review stations can be configured as either review-only systems or full-function analysis and report-generation systems. Typically, a laboratory that is heavily invested in the digital echocardiographic laboratory may have a number of view-only stations for use by requesting physicians, emergency department physicians, the operating room, among others, with a more limited number of more sophisticated workstations for measurement and report generation.

A major advantage of the digital environment is the ability to retrieve and compare in a side-by-side format multiple studies. This allows a side-by-side display of comparable images recorded at two different points in time for evaluation of serial changes and has particular relevance with respect to evaluating resolution of wall motion abnormalities after acute myocardial infarction, serial changes in valvular insufficiency, or serial changes in left ventricular function. This ability to rapidly evaluate echocardiograms recorded at different points in time in a side-by-side fashion has been a major advantage of the digital echocardiography laboratory. Finally, the digital
image is played as a continuous loop of anywhere from one to ten cardiac cycles. Because this is a controllable digital loop, the reviewer has the ability to evaluate wall motion, valvular function, and other parameters on a frame-by-frame basis and provide a highly detailed temporal resolution of cardiac events, which is typically not feasible when reviewing video images. Although the cost of instituting a digital archiving and review system can be significant, the majority of laboratories that have migrated from videotape to a digital environment have found that the improved efficiency of analyses and the cost-saving achieved by not archiving videotape substantially mitigates the increased cost of a digital laboratory.

The techniques and methods of echocardiography should not be viewed in isolation. All the techniques are interrelated, and, in theory, all the different
imaging modalities such as 2D imaging, M-mode imaging, color flow imaging, and spectral Doppler imaging are available from any given single transducer. Limitations in computational power and transducer design of necessity may limit the information at this time from any one device.

<table>
<thead>
<tr>
<th>Level</th>
<th>Duration of Training (months)</th>
<th>Cumulative Duration of Training (months)</th>
<th>Minimal Total no. of TTE Examinations Performed</th>
<th>Minimal Total no. of TTE Examinations Interpreted</th>
<th>TEE and Special Procedures</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3</td>
<td>3</td>
<td>75</td>
<td>150</td>
<td>Yes&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
<td>6</td>
<td>150</td>
<td>300</td>
<td>Yes&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>9</td>
<td>300</td>
<td>750</td>
<td>Yes</td>
</tr>
</tbody>
</table>

<sup>a</sup>Initial exposure to transesophageal echocardiography and other special procedures.

<sup>b</sup>Completion of level 2 and additional special training needed to achieve full competence in transesophageal echocardiography and special procedures.

TEE, transesophageal echocardiography; TTE, transthoracic echocardiography.


**TRAINING AND COMPETENCY IN ECHOCARDIOGRAPHY**

Standards for training in echocardiography, as well as all areas of cardiovascular medicine, are defined by the COCATS, now in its fourth iteration (see Table 4.7). The COCATS task force on echocardiography focuses on fellowship training and defines three levels:

- Level I—basic training required to become competent consultants; required of all fellows; defined as an introductory or early level of competency, but not sufficient to allow independent interpretation.
- Level II—sufficient training to allow independent performance and interpretation of basic and complex echocardiograms; does not imply
complete mastery of all special procedures, such as 3D, intracardiac, or intraoperative or intraprocedural TEE.

Level III—intended for those fellows who plan careers in echocardiography; includes an exposure to laboratory administration, research experience, and special procedures, including 3D, contrast perfusion, and intraprocedural echocardiography. Specific criteria for Level III training will be covered in an Advanced Training Statement.

Once trained, maintaining competency in a complex, rapidly advancing field is difficult. Lifelong Learning Competencies have recently been developed for all areas of cardiology, including echocardiography to provide assistance in this regard. These documents and tables define the key elements of competency expected to be maintained throughout a professional career. They are grouped into two categories: medical knowledge, and patient care and procedural skills. The competencies are further classified as those expected of all clinical cardiologists and those expected of individuals in specific subspecialty areas. Keeping abreast of new developments in a large, dynamic, and rapidly changing field such as echocardiography is challenging. These documents help guide the busy clinician by focusing on the key areas necessary for developing and maintaining excellence.

**Suggested Readings**

**GENERAL CONCEPTS**


**APPROPRIATE USE**


Applications


Reporting

Cerqueira MD, Weissman NJ, Dilsizian V, et al. Standardized myocardial segmentation and


**TRANSESOPHAGEAL ECHOCARDIOGRAPHY**


**TRAINING**


**FOCUSED CARDIAC ULTRASOUND**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Chapter 5
Evaluation of Systolic Function of the Left Ventricle

GENERAL PRINCIPLES

Most forms of acquired heart diseases may be associated with abnormalities of left ventricular systolic function at some point in their natural history. An assessment of left ventricular systolic function should be part of virtually all echocardiographic examinations. Assessment of systolic function provides valuable prognostic information, plays a crucial role in selection of medical therapy, and is instrumental in determining the timing of surgery for valvular heart disease.

This chapter will deal with echocardiographic techniques for evaluation of both global and regional left ventricular systolic function. Since its inception echocardiography has played a role for assessment of left ventricular systolic function, initially beginning with M-mode echocardiography and progressing to modern platforms providing comprehensive three-dimensional imaging of the left ventricle with the ability to extract detailed parameters of ventricular function. This chapter will concentrate on the currently utilized and commercially available methods for evaluation of left ventricular systolic function. Older techniques and techniques which have been utilized for investigational purposes only are mentioned for historical purposes, or for their relevance with respect to limitations which may still be present in modern analysis systems.
The first attempts to quantify left ventricular function involved linear measurements of the minor-axis dimension from a dedicated M-mode echocardiogram. Linear measurements have the disadvantage of determining ventricular function only along a single interrogation line. The precise location at which linear measurements are made has varied as the resolution of ultrasound instrumentation has improved. Initial ultrasound equipment had relatively poor gray-scale registration. As such, the precise boundary between the blood pool and tissue was often difficult to determine. One early approach to linear measurements involved a “leading-edge to leading-edge” technique. Using this technique, septal thickness was defined as the leading edge of the septum on its right ventricular side to the leading edge of bright endocardial echoes on the left ventricular side of the ventricular septum. Depending on gray scale, image intensity, and resolution, the leading edge itself could be as much as 1 or 2 mm in thickness. Refinements in image processing have allowed greater levels of gray-scale registration with a substantially refined visualization of the actual tissue–blood pool boundary. It is now common practice to measure chamber dimensions, as defined by the actual tissue–blood interface, rather than the distance between the leading-edge echoes. Table 5.1 outlines many of the linear measurements that can be made for assessment of left ventricular function. The location of these measurements is schematized in Figure 5.1 and further demonstrated in Figure 5.2.

Although the temporal resolution of a dedicated M-mode beam is superior to that of two-dimensional echocardiography, the ability to visualize the entire left ventricle, and to ensure a true minor-axis dimension, mitigates this potential advantage for most purposes. There are multiple limitations of linear measurements for determining ventricular performance. One of the most obvious is that many forms of acquired heart disease, especially coronary artery disease, result in regional variation in ventricular shape and function. By definition, a linear measurement provides information regarding dimension and contractility only along a single line. This may either underestimate the severity of global dysfunction if only a normal region is interrogated, or overestimate the abnormality if the M-mode beam exclusively transits the wall motion abnormality. An additional limitation of an M-mode measurement of the left ventricle is that it often does not reflect the true minor-axis dimension. This phenomenon is illustrated in Figure 5.2 and is very common in elderly patients in whom there is angulation of the
ventricular septum. In this instance, an M-mode beam traverses the ventricle in a tangential manner and overestimates the true internal dimension. As a two-dimensionally guided M-mode cursor must still adhere to beam direction from the transducer, it is often not possible to align the beam truly perpendicular to the long axis of the ventricle so that it reflects the true minor-axis dimension. Some platforms may allow an “anatomical M-mode” beam to be derived from a two-dimensional dataset and thereby remove this limitation. When comparisons are made between M-mode and two-dimensional minor-axis dimensions, the M-mode dimension typically overestimates the true minor axis of the left ventricle by 6 to 12 mm. This systematic discrepancy becomes greater with age and the attendant angulation of the heart. For any given patient, one can generally assume that the degree of off-axis interrogation will remain stable over time and this overestimation will remain constant. As such, in the absence of new regional abnormalities, differences in serial measurements retain their clinical validity, although the actual dimension may be incorrect.

| Table 5.1 | LINEAR MEASUREMENTS OF LEFT VENTRICULAR SIZE AND FUNCTION |

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Formula</th>
<th>Abbreviation</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV internal dimension in diastole</td>
<td>LVID_d</td>
<td>LVID_d</td>
<td>mm (or cm)</td>
</tr>
<tr>
<td>LV internal dimension in systole</td>
<td>LVID_S</td>
<td>LVID_S</td>
<td>mm (or cm)</td>
</tr>
<tr>
<td>Fractional shortening</td>
<td>(LVID_d – LVID_s)/LVID_d</td>
<td>FS</td>
<td>% or 0.XX</td>
</tr>
<tr>
<td>Meridional wall stress in systole</td>
<td>PR/h</td>
<td>σ_m</td>
<td>mm Hg or dyne-cm²</td>
</tr>
<tr>
<td>Cubed LV volume in diastole</td>
<td>(LVID_d)^3</td>
<td></td>
<td>cm³ or mL</td>
</tr>
<tr>
<td>Cubed LV + myocardial volume</td>
<td>(IVS + LVID_d + PW)^3</td>
<td></td>
<td>cm³ or mL</td>
</tr>
<tr>
<td>Velocity of circumferential shortening</td>
<td>(LVID_d – LVID_s)/(LVID_d × ET)</td>
<td>V Cf</td>
<td>Circumference/s</td>
</tr>
</tbody>
</table>

ET, ejection time; h, wall thickness; PR, pressure × radius; PW, posterior wall.
FIGURE 5.1. Schematic of a parasternal long-axis view of the left ventricle depicting linear measurements. By convention, linear measurements of the left ventricle are made at the level of the mitral chordae. From the linear internal dimension of the left ventricle in diastole and systole, fractional shortening can be calculated as noted. When measuring ventricular septal thickness, caution is advised to avoid measuring the most proximal portion of septum, which is frequently an area of isolated hypertrophy and angulation that does not truly represent ventricular wall thickness. FS, fractional shortening; LVID_d, left ventricular internal dimension in diastole; LVID_S, left ventricular internal dimension in systole; PW, posterior wall.
FIGURE 5.2. Parasternal long-axis echocardiogram and two-dimensional–derived M-mode echocardiogram in a patient with normal ventricular function. On the M-mode echocardiogram, note the internal dimension of the left ventricle of 5.5 cm and the derived values. On the two-dimensional echocardiogram, the longer white line represents the M-mode interrogation beam. Note that it traverses the left ventricle in a tangential manner and results in an internal dimension of 5.5 cm.
cm. The yellow line is the true short-axis dimension of the left ventricle which is substantially smaller at 4.5 cm. PW, posterior wall.

M-mode echocardiography provides a slight advantage for timing of events but confers no real advantage over direct two-dimensional measurements for chamber dimensions. There are several additional parameters of ventricular performance that can be derived from M-mode measurements. These include rates of systolic wall thickening of the posterior wall and calculation of velocity of circumferential shortening. For the latter calculation, the minor-axis is assumed to represent a circle of known diameter from which the circumference can be calculated and the rate of change of circumference determined. This measurement, typically standardized by normalizing to heart rate, is rarely used in contemporary practice.

An additional M-mode measurement that has been employed in the past is the descent of the base. During ventricular contraction, the base (annulus) of the heart moves toward the apex. In the presence of global left ventricular dysfunction, the magnitude of this motion is directly proportional to systolic function. M-mode interrogation is undertaken of the lateral mitral annulus, and annular excursion toward the transducer is then calculated (Fig. 5.3). There is a relatively linear correlation between the magnitude of systolic annular excursion and global systolic function. This technique is rarely used today, having given way to direct measures of ventricular volume and ejection fraction.
FIGURE 5.3. Apical view recorded in two patients demonstrates the measurement of the descent of the base with M-mode echocardiography (arrows). The M-mode interrogation beam has been directed from the apex of the heart through the lateral annulus. **A:** Note the approximate 1.6 cm of annular motion toward the apex in systole. **B:** Recording in a patient with severe systolic dysfunction reveals substantially decreased annular motion of <1.0 cm in systole.

This same principle is used in Doppler tissue imaging of the annulus for
determination of systolic velocities and excursion of the mitral annulus as a marker of ventricular function. Doppler tissue imaging relies on adjustment of Doppler gains and filters to selectively record velocities from within the myocardium itself rather than the blood pool. A sample volume can be placed within the mitral annulus and quantitative information extracted regarding annular velocity (Fig. 5.4). In a uniformly contracting ventricle, annular systolic velocity is a marker of global left ventricular function. Annular velocity data also play a major role in assessment of diastolic function, as discussed in Chapter 6.
FIGURE 5.4. Doppler tissue imaging of the lateral annulus performed in two patients. The upper panel was recorded in a patient with normal systolic function and an ejection fraction of 60%. Notice the S wave of 9 cm/s. Also noted are the diastolic e' and a' velocities. The lower panel was recorded in a patient with a dilated cardiomyopathy and ejection fraction of 27%. Notice the annular systolic velocity of 4 cm/s consistent with reduced global function.
Several indirect signs of left ventricular systolic dysfunction can be noted on M-mode echocardiography. These include an increased E-point to septal separation and gradual closure of the aortic valve during systole. The magnitude of opening of the mitral valve, as reflected by E-wave height, correlates with the volume of transmitral flow and, in the absence of significant mitral regurgitation, with left ventricular stroke volume. The internal dimension of the left ventricle correlates with diastolic volume. As such, the ratio of mitral excursion to left ventricular size parallels ejection fraction. Normally, the mitral valve E-point (maximal early opening) is within 6 mm of the left side of the ventricular septum. In the presence of a decreased ejection fraction, this distance is increased (Fig. 5.5).

**FIGURE 5.5.** M-mode echocardiograms recorded in two patients with significant systolic dysfunction. **A:** An E-point septal separation (EPSS) of 1.2 cm (normal, <6 mm). **B:** Recording in a patient with more significant left ventricular systolic dysfunction in which the EPSS is 3.0 cm. Also note the interrupted closure of the mitral valve with a B bump (top), indicating an increase in the left ventricular end-diastolic pressure.
FIGURE 5.6. M-mode echocardiogram recorded through the aortic valve in a patient with reduced cardiac function and decreased forward stroke volume. Note the rounded closure of the aortic valve, indicating decreasing forward flow at the end of systole. Normal and abnormal aortic valve opening patterns are noted in the schematic superimposed on the figure.

Inspection of the aortic valve opening pattern also provides indirect evidence regarding systolic function of the left ventricle. If left ventricular forward stroke volume is decreased, there may be a gradual reduction in forward flow in late systole. This results in a rounded appearance of aortic valve closure in late systole (Fig. 5.6). Reliance on these earlier observations and calculations has been supplanted by direct measures of ventricular size and performance available from modern ultrasound platforms.

MEASUREMENTS FROM STANDARD TWO-DIMENSIONAL IMAGING

Two-dimensional echocardiography provides inherently superior spatial resolution for determining left ventricular size and function. Its role in obtaining linear measurements has already been discussed. A number of two-dimensional echocardiographic views have been used to provide information
regarding ventricular systolic function, some of which rely exclusively on area measurements and others of which rely on calculation of ventricular volume. Table 5.2 outlines commonly used two-dimensional measurements and their derived calculations. Table 5.3 provides the American Society of Echocardiography–recommended normal ranges for commonly obtained measurements.

Most often, apical images are used to determine ventricular volumes in diastole and systole, from which stroke volume and ejection fraction are calculated. There are several geometric assumptions and formulas that have been used in the past for calculating ventricular volume. The advantage of the geometric assumption techniques, such as an area-length or truncated ellipse formula, is that they require only limited visualization for calculation of ventricular volume. These formulas work only in a symmetrically contracting ventricle and have been supplanted by more direct calculation of ventricular volumes.

The advent of high-resolution 90-degree digital two-dimensional scanners, as well as the computational capacity of quantitation packages incorporated in modern platforms and off-line analysis systems, has largely made these earlier methods for volume determination obsolete. Currently, the most common method for determining ventricular volumes is the Simpson rule, or the “rule of discs.” This technique requires recording an apical, four- and/or two-chamber view from which the endocardial border is outlined in end-diastole and end-systole. The ventricle is mathematically divided along its long axis into a series of discs of equal height. Individual disc volume is calculated as the product of height and disc area, where disc height is assumed to be the total length of the left ventricular long axis divided by the number of segments or discs. The surface area of each disc is determined from the diameter of the ventricle at that point (area = \( \pi r^2 \)). The ventricular volume is calculated as the sum of the volume of the discs. This methodology is illustrated in Figure 5.7.

If the ventricle is symmetrically contracting, either the four- or two-chamber view will reflect the ventricular volume. For accurate volume determination, the transducer must be at the true apex and the ultrasonic beam must be through the center of the left ventricle. These conditions are frequently not met, resulting in underestimation of true ventricular volumes. There are several clues that help determine whether the transducer is at the...
true apex. Normally, the true apex is the thinnest area of the left ventricle. If the visualized apex has the same or greater thickness as the surrounding walls, and appreciable motion in systole, it is likely to be a tangential cut through the left ventricle rather than a true on-axis view. In addition, a properly recorded apical view is defined as the one with the greatest long-axis (apex to base) dimension. In any view, foreshortening of the ventricular apex will result in underestimation of ventricular volume. In clinical practice, the apical two-chamber view is often imaged tangentially, and the volume derived from this view may underestimate the true left ventricular volume. Because of cardiac translational motion, tangential imaging (i.e., not through the midline of the ventricle) is more common in systole. This results in an artifactually small systolic left ventricular cavity and may result in overestimation of ejection fraction. It is common to encounter minor degrees of off-axis imaging in the apical view in which tangentially located myocardium appears to fill in the apex because of beam width imaging. Visually evaluating the location of the true apical myocardium in real time, before tracing the boundary, and purposefully placing the boundary within the vague tangential echoes can reduce the magnitude of this problem.

<table>
<thead>
<tr>
<th>Table 5.2</th>
<th>AREA/VOLUME-BASED MEASUREMENTS FOR VENTRICULAR SIZE AND FUNCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>parameter</td>
<td>Abbreviations</td>
</tr>
<tr>
<td>Short-axis diastolic area (at mid LV)</td>
<td>ASx_d</td>
</tr>
<tr>
<td>Short-axis systole area (at mid LV)</td>
<td>ASx_s</td>
</tr>
<tr>
<td>Fractional area change</td>
<td>FAC</td>
</tr>
<tr>
<td>Four-chamber LV area in diastole</td>
<td>ALV_4c–d</td>
</tr>
<tr>
<td>Four-chamber LV area in systole</td>
<td>ALV_4c–s</td>
</tr>
<tr>
<td>LV volume in diastole</td>
<td>LVV_d</td>
</tr>
<tr>
<td>LV volume in systole</td>
<td>LVV_s</td>
</tr>
<tr>
<td>Stroke volume</td>
<td>SV</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>EF</td>
</tr>
</tbody>
</table>
Determined by the Simpson rule, area length method, etc.

**Table 5.3**  NORMAL VALUES FOR 2D ECHOCARDIOGRAPHIC PARAMETERS OF LV SIZE AND FUNCTION ACCORDING TO GENDER

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>2-SD Range</td>
</tr>
<tr>
<td>LV internal dimension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic dimension (mm)</td>
<td>50.2 ± 4.1</td>
<td>42.0–58.4</td>
</tr>
<tr>
<td>Systolic dimension (mm)</td>
<td>32.4 ± 3.7</td>
<td>25.0–39.8</td>
</tr>
<tr>
<td>LV volumes (biplane)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV (mL)</td>
<td>106 ± 22</td>
<td>62–150</td>
</tr>
<tr>
<td>LFESV (mL)</td>
<td>41 ± 10</td>
<td>21–61</td>
</tr>
<tr>
<td>LV volumes normalized by BSA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV (mL/m²)</td>
<td>54 ± 10</td>
<td>34–74</td>
</tr>
<tr>
<td>LVESV (mL/m²)</td>
<td>21 ± 5</td>
<td>11–31</td>
</tr>
<tr>
<td>LVEF (biplane)</td>
<td>62 ± 5</td>
<td>52–72</td>
</tr>
</tbody>
</table>

FIGURE 5.7. Schematic illustration of Simpson rule or the rule of discs for calculating left ventricular volume. In the upper panel, a schematized left ventricular volume has been subdivided into 10 sections, each of which is presumed to represent a disc of equal diameter at its top and bottom margins. The volume of each disc is calculated as area \times height where height is defined as

\[
L \quad \text{Disc Volume} = \pi r_n^2 \times h
\]

Total Volume = \sum_{n=1}^{\text{max}} \pi r_n^2 \times h
the left ventricular length from apex to base divided by the number of discs. The total volume of the ventricle is calculated as the sum of each disc volume. The lower panel is an apical four-chamber view recorded in a normal individual in which this algorithm has been used to calculate left ventricular volume.

For determination of left ventricular volume, the endocardial border is traced with papillary muscles and trabeculae excluded from the cavity (Figs. 5.8 and 5.9). The well-recognized underestimation of left ventricular volume by echocardiography, compared to a standard such as cardiac magnetic resonance imaging, is in part due to failure to exclude trabeculae from the cavity tracing. If there is asymmetry of ventricular geometry or a systolic wall motion abnormality, a single-plane view will have reduced accuracy for the reasons previously alluded to. In this instance, averaging of volumes from multiple views or use of three-dimensional echocardiography will increase accuracy.

Once the diastolic and systolic volumes have been determined, stroke volume can be calculated as the difference between these two volumes. Assuming the absence of mitral or aortic insufficiency, forward cardiac output then equals the product of heart rate and stroke volume. Ejection fraction can be calculated from these volumes as: stroke volume ÷ end-diastolic volume. Because the difference between the diastolic and systolic left ventricular volume represents the total volume pumped by the ventricle, it represents the sum of forward-going stroke volume plus the volume of mitral and aortic regurgitation, if present.

**Automated Edge Detection**

Most currently available instrumentations incorporate algorithms to automatically identify and track the endocardial border of the left ventricle. The precise methodology by which endocardial borders are tracked varies from manufacturer to manufacturer. The basic principle is that the acoustic boundary between the blood pool and tissue is identified and then tracked throughout the cardiac cycle (Fig. 5.10). The degree to which this is automated is highly variable and ranges from fully automated systems in which there is no user interaction, to systems in which multiple points of the ventricular contour are manually defined, after which the boundary between points is extrapolated. One of the more common techniques is to identify the
apex of the left ventricle and then the lateral and medial mitral annulus after which automatic detection algorithms identify the endocardial boundary. After the initial approximation of the endocardial boundary, operator interaction is often required to adjust the boundary to fit the visually identified cavity (Fig. 5.11). Because the algorithms are detecting a blood pool–tissue boundary they often delineate the cavity of the left ventricle along the boundary of trabeculations and papillary muscles, which by convention should be excluded from the blood pool for calculation of ventricular volumes (Fig. 5.9). These same techniques (or speckle tracking which is discussed subsequently) for detecting the cavity boundary can also be applied to three dimensional datasets (Fig. 5.12).
FIGURE 5.8. Apical four-chamber view recorded in a patient with normal ventricular size and function. The upper panel is the apical four-chamber view from which volume can be calculated. Notice the vague echoes at the apical septal and apical lateral wall due to a combination of beam width imaging and
trabeculae (arrows) as well as the papillary muscle protruding into the left ventricular cavity (arrow). The lower panel outlines three separate contours which could be drawn from this view. The white line represents the true inner endocardial border of the left ventricle, excluding trabeculation, beam width imaging, and the papillary muscle from the cavity, and results in a left ventricle cavity volume of 97 mL. The yellow line excludes the papillary muscle tip but includes the apical trabeculations and tangential beam–related echoes and results in a left ventricular volume of 70 mL. The red line further excludes the papillary muscle tip from the left ventricular volume and would result in a left ventricular volume of 60 mL.

After the endocardial boundary has been identified, various algorithms are utilized to determine the volume. In earlier systems, Simpson rule was employed in a manner similar to that used for manually drawn contours. In its simplest form borders were outlined only in diastole and end-systole. Modern systems typically determine the left ventricular volume from an actual pixel count bounded by the endocardial boundary. Ventricular volume can then be calculated continuously through the cardiac cycle and graphically displayed over time. Stroke volume and ejection fraction can be calculated from the maximal and minimal volumes.
FIGURE 5.9. Apical four-chamber view recorded in a young patient with normal ventricular function and fairly prominent trabeculae along the lateral ventricular wall. The upper panel is an apical four-chamber view in which the papillary muscle and trabeculae can be seen on the lateral wall (arrows). The lower left panel is the initial, unaltered, automatically determined endocardial border from a commercially available platform. Note that the algorithm for identifying the endocardial border has included papillary muscles and the trabeculae within the
ventricular cavity which results in a calculated left ventricular volume of 99 mL. The lower right panel was recorded after manual adjustment of the previously automatically determined border. Only the lateral border has required adjustment. After adjustment, notice that the calculated left ventricular volume is 158 mL.

Video 5-9

FIGURE 5.10. Apical four-chamber view from which continuous volume determination has been made using an automated acoustic boundary detection system. The apical four-chamber view is presented. The dots represent the
automatically determined acoustic boundary after manual adjustment. See Figure 5.11 for the initially automatically detected acoustic boundary. At the upper right is a table with numeric values. Note the calculated ejection fraction of 58.1%.

**Video 5-10**

**FIGURE 5.11.** Apical four-chamber view recorded in the same patient depicted in Figure 5.10. This figure represents the first approximation of the ventricular boundary by the automated edge detection algorithm. Note that the automatically detected boundary (*dotted lines*) has foreshortened the left ventricle and located
the apex well short of its true location (arrows). At the lower right is an expanded view of the same image. The downward-pointing arrows denote the location of the epicardial boundary of the apex and the double-headed arrow the distance between the automatically detected boundary and the endocardium of the apex. In this instance the erroneous boundary detection was related to vague echoes in the apex due to beam width artifact. Also note that along the lateral wall the detection algorithm has identified the endocardial border at the tip of the papillary muscle. This has resulted in an overestimation of ejection fraction and an underestimation of ventricular volume as noted at the upper right. The image in Figure 5.10 was recorded after a single manual manipulation of the boundary.

Video 5-11

An additional methodology for tracking the myocardium is “speckle tracking.” This technique relies on creating an “acoustic signature” of multiple regions of interest within the myocardium (Fig. 5.13). The acoustic signature of any given region remains stable throughout the contraction sequence and therefore the region can be tracked over the cardiac cycle. Most currently available echocardiographic platforms provide speckle tracking in two dimensions. Accurate high frame rate three-dimensional speckle tracking is still in development. The speckle tracking technology provides a mechanism for tracking discreet myocardial segments from which deformation measurements of strain and strain rate can be calculated. It also provides definition of the myocardial boundary from which the myocardial blood pool and ventricular volumes can be extrapolated.
FIGURE 5.12. Composite image derived from a real-time three-dimensional full-volume dataset. The image at the far lower right is the cropped three-dimensional volume set in which the details of the left ventricular cavity are identified. The images at the left include apical four-chamber (4C), apical long-axis (ALx) and two-chamber (2C) views. At the lower right quadrant is a fitted model of the left ventricle and left atrium derived from acoustic boundary determination. The table at the upper right outlines end-diastolic and end-systolic volumes, ejection fraction, and left atrial area.
FIGURE 5.13. Illustration of the basic principles of speckle tracking. An apical four-chamber view is presented from which a section of the ventricular septum has been expanded (bordered area). Within the expanded area, two circular regions of interest are identified. Note the distinctly different acoustic signature within these regions. This illustration is a simplification of the speckle phenomenon and, in reality, substantially smaller regions of interest with more subtle variation in tissue signature based on more fundamental imaging characteristics are utilized.

With either tissue tracking or acoustic boundary detection, the utility of the automated edge detection is greatest in high-quality studies and rapidly drops off with lower-quality images. For patients with significant degradation of overall visual image quality automated edge detection systems may consistently fail and will provide erroneous information which should not be used.

It should be heavily emphasized that blind reliance on any of the available automated edge detection algorithms, whether utilizing two or three-dimensional echocardiography, acoustic boundary detection or speckle tracking, must be visually confirmed as accurate before the data are utilized. Visual analysis by a skilled echocardiographer incorporates endocardial motion and myocardial thickening into the assessment of ventricular function both regionally and globally. A skilled echocardiographer visually and
mentally filters out artifact and other vague extraneous echoes which can be confused for the true endocardial border. At all times the automatically detected border should be scrutinized for accuracy against the known location of the endocardial border when analyzed in real time and appropriately adjusted. Automated edge detection algorithms commonly track the papillary muscle or trabeculae as the endocardial border (Fig. 5.9) or foreshorten the apex by tracking vague echoes in the apical cavity which are related to beam width and do not represent the true endocardial border (Fig. 5.11).

Intravenous contrast for left ventricular opacification is also a valuable technique for enhancing endocardial border definition. It has been recommended that if two or more ventricular segments are poorly visualized, there is incremental yield of intravenous contrast for left ventricular opacification both for regional wall motion assessment and for reproducibility of volume determination. Intravenous contrast can be employed either with two-dimensional or with three-dimensional imaging and, as discussed in Chapter 3, requires attention to detail with respect to mechanical index and other technical factors of imaging.

**Assessment of Left Ventricular Function With Three-Dimensional Echocardiography**

A three-dimensional echocardiographic dataset which potentially includes all four cardiac chambers can be acquired through a number of methods. Acquisition methods include real-time three-dimensional full volumetric scanning and merging or “stitching” several three-dimensional subvolumes into a full-volume dataset. This dataset can be further analyzed with a number of techniques. Some state-of-the-art systems include an artificial intelligence algorithm which matches the left ventricular contour to a “library” of previously identified ventricular cavities of various sizes and geometries in an effort to ensure that the automated boundary detection is providing information within the realm of previously identified ventricular cavities. This then provides a second approach for determination of ventricular volume and ejection fraction. Compared to two-dimensional imaging, three-dimensional imaging provides more accurate information regarding left ventricular volume when compared to a standard such as cardiac magnetic resonance imaging. The advantage of three-dimensional volumetric
calculations appears greatest in irregularly shaped ventricles which do not conform to a predictable geometric shape. Three-dimensional datasets have been merged with a variety of edge detection algorithms allowing semiautomatic extraction of a three-dimensional volume after user identification of a limited number of points, or as a fully automatic analysis. This advancement has dramatically reduced the time required for derivation of accurate three-dimensional volumes (Figs. 5.14 and 5.15). As with automated algorithms for determination of left ventricular volume from two-dimensional echocardiography, manual adjustment of the automatically defined ventricular border is commonly necessary. Once generated, the three-dimensional volume can be further subdivided into a 16- or 17-segment model as done with two-dimensional echocardiography. A variety of sophisticated measures of global and regional ventricular functions can be extracted from the same three-dimensional volume (Figs. 5.16 and 5.17). The data that can be extracted is platform specific but includes regional volume change in 16 or 17 segments as well as parameters of volume change over time which have shown promise for evaluation of mechanical dyssynchrony. Numerous studies have demonstrated the superiority of three-dimensional echocardiography over two-dimensional echocardiography for determination of left ventricular volumes when compared to a standard such as cardiac magnetic resonance imaging. While the accuracy and inter- and intraobserver reproducibility of left ventricular volumes derived from three-dimensional datasets exceeds that of two-dimensional imaging, the magnitude of improvement in accuracy is not always at a level likely to result in a change in clinical decision making. Most studies have suggested that left ventricular volumes determined with real-time three-dimensional echocardiography underestimate both end-diastolic and end-systolic volume. As with two-dimensional imaging, this is apparently due to inclusion of left ventricular trabeculae and papillary muscles within the cavity and is a more prominent problem with less experienced operators.
FIGURE 5.14. Stylized shell rendering of a normal left ventricle recorded using full-volume three-dimensional echocardiography. Note the calculated end-diastolic and end-systolic volumes as well as ejection fraction and stroke volume. In the accompanying video note the normal motion of all visualized 17 segments.
After acquisition of the full-volume three-dimensional dataset customized two-dimensional imaging planes can be extracted. Commonly a semi-automated technique is used to extract an apical two- and four-chamber view or a series of left ventricular short-axis views (Figs. 5.18 and 5.19). While it is feasible to extract multiple two-dimensional views from the three-dimensional dataset, the image quality of the extracted views is below that obtained from a dedicated two-dimensional scan. This is demonstrated in Figure 5.18 which illustrates a three-dimensional dataset from which an apical four-chamber view has been extracted at the lower left. At the lower right is a superimposed two-dimensional apical four-chamber view from the same patient recorded on the same platform but utilizing dedicated two-dimensional scanning.
FIGURE 5.15. Full-volume three-dimensional imaging recorded in a patient with a left bundle branch block and mildly reduced left ventricular ejection fraction. Volumes, stroke volume, and ejection fraction are at the upper right. At the upper left a stylized shell has been fitted to the anatomically defined left ventricular cavity. In this instance selective volumes for only the septal and lateral walls are being graphed (lower panels). Note the heterogeneity of volume reduction in the septal and lateral walls related to the left bundle branch block.

Video 5-15
FIGURE 5.16. Parametric imaging derived from a patient with normal left ventricular systolic function. The bull's-eye plots depict timing of contraction (upper plot) and wall excursion (lower plot). Individual volumetric changes for each of the 17 segments are plotted in the lower graph. Detailed parameters of temporal heterogeneity are displayed on the right.
Strain and Strain Rate Imaging (Deformation Imaging)

The majority of analysis techniques discussed thus far analyze left ventricular wall motion from the frame of reference of the transducer. As such, rotation, translational motion, and tethering confound analysis. Doppler tissue imaging and speckle tracking allow for evaluation of a myocardial region with reference to an adjacent myocardial segment rather than to a fixed transducer position and theoretically provide more accurate data regarding ventricular function. Analysis of ventricular mechanics or shape during the cardiac cycle...
is referred to as deformation analysis. Deformation can be characterized by myocardial strain, strain rate, or torsion, each of which defines a different parameter of shape change with contraction and relaxation.

**FIGURE 5.18.** Comparison of the three-dimensionally and two-dimensionally derived apical four-chamber views in a patient with high image quality. The upper figure is the three-dimensional dataset from which apical four- and two-chamber views have been extracted. At the lower left is the three-dimensionally derived apical four-chamber view. At the lower right is a dedicated two-dimensional scan from the same patient which has been superimposed in the location where the two-chamber view was originally displayed. Note the substantially better resolution available from the dedicated two-dimensional probe compared to the extracted four-chamber view from the three-dimensional probe.
FIGURE 5.19. Multiple two-dimensional imaging planes have been extracted from a full-volume three-dimensional dataset. The upper panels show apical four- and two-chamber views and a single short-axis image which have been extracted from the full-volume dataset. At the lower right is a series of nine short-axis images which correspond to the horizontal lines in the apical views. The lower graph depicts the instantaneous volume change for multiple analyzed segments.
These parameters of function are derived from analysis of motion (strain) or velocity (strain rate) at two or more myocardial regions from which strain and other advanced parameters can be calculated. Strain may be calculated in any of three orthogonal planes, representing longitudinal, circumferential, and radial contraction (Fig. 5.20). Strain is defined as the normalized change in length between two points (Fig. 5.21). Negative strain implies shortening of a segment (contraction) and positive strain lengthening of a segment (relaxation). As such, normal contraction is defined by negative longitudinal systolic strain followed by biphasic diastolic strain related to early and late diastolic filling, respectively. Normal radial strain, reflecting wall thickening is positive in systole. Strain rate represents the change in velocity between two adjacent points.

Strain and strain rate can each be calculated either from Doppler tissue imaging or from speckle tracking techniques and displayed in a multitude of formats (Figs. 5.22 to 5.24). Because of poor signal to noise ratios and other factors most current platforms rely on speckle tracking rather than tissue Doppler techniques. It should be emphasized that for Doppler tissue imaging, the initial raw data represent myocardial velocity at a point in space within the interrogating beam. To calculate distance, this velocity is integrated over time. If two discrete points within a region of interest are compared for change in velocity over the cardiac cycle, strain rate is the primary parameter obtained. Strain, or the change in distance between the two points is, therefore, the derived variable. Conversely, with speckle tracking it is the actual location of discrete myocardial segments (rather than the velocity) that is calculated. As such, the primary calculation is of tissue displacement. If two points are simultaneously compared for their location, the primary parameter derived is strain rather than strain rate. With speckle tracking, strain rate can be derived from the original data by calculating the rate of change in location over time (velocity) for two adjacent points. With either technique, regions of interest can vary from 5 to 6 mm to 2 to 3 cm in length.

Using current generation platforms, the typical method by which longitudinal strain is calculated is to acquire an apical four-chamber, two-chamber and apical long-axis view of the left ventricle. Each of these views is then subjected to speckle tracking analysis to assess longitudinal strain in discrete segments. Assessment of end-systolic strain requires identification of end-systole. The current recommendation is that timing of end-systole be
determined from Doppler of the left ventricular overflow tract and defined as aortic valve closure (Fig. 5.22). The current recommendation for global longitudinal strain (GLS) is that it be calculated on an 18-segment model with part of the apex being represented in each of six segments representing the apical third of the left ventricle. Individual strain can be plotted over the cardiac cycle for each segment and GLS calculated as the average longitudinal strain in each of the 18 segments (Fig. 5.23).

While strain can be calculated either in the longitudinal, circumferential, or radial dimensions, most commercially available platforms provide only analysis of longitudinal strain. Because ultrasound platforms use proprietary algorithms for calculation of strain, initially there was substantial variability in normal ranges across platforms. More recently significant standardization has occurred and GLS appears to be fairly reproducible and equivalent across multiple ultrasound platforms. In addition, studies have demonstrated that GLS is more reproducible and provides a more reliable, reproducible parameter for following ventricular function in a broad spectrum of disease than does radial strain or strain rate.
FIGURE 5.20. Schematic demonstration of the three orthogonally directed strain calculations. Longitudinal strain ($\varepsilon_L$) is defined as along the long axis of the left ventricle. Radial strain ($\varepsilon_R$) is orthogonal to the longitudinal strain and oriented perpendicular to the endocardial border. Circumferential strain ($\varepsilon_C$), calculated in the short axis of the ventricle, is parallel to the radius of the ventricle. The curved arrows outside the schematic depict the normal clockwise basal and counterclockwise apical twisting of the left ventricle.
Strain, like most parameters of systolic function, is not uniform among all myocardial segments. Myocardial velocities and displacement have a gradation in magnitude from base to apex, with basal parameters being higher than apical values. Longitudinal strain, defined as motion parallel to the long axis has less variability apex to base but varies substantially around the circumference of the left ventricle, with higher strain in the anterior and lateral walls compared to the inferior and septal wall. Normal longitudinal strain averages \(-20\%\) and is numerically less than normal radial strain. There is a well-described base to apex variation in strain in normals which has varied in magnitude based on the ultrasound platform used and technique (tissue Doppler vs. speckle tracking). This lack of uniformity probably relates to a combination of factors, including angle dependency with tissue Doppler, length of segment analyzed, and incorporation of annular or pericardial tissue in the region of interest. If Doppler tissue imaging is used to calculate myocardial velocity, there will be angle dependency of the velocity determination which becomes more pronounced at the apical segments where ultrasound beam interrogates a wall curve. At the true apex, the beam
intersects the myocardium at 90 degrees and longitudinal strain precipitously declines if assessed with Doppler tissue techniques. For this and other reasons, including a more favorable signal to noise ratio, speckle tracking has largely replaced Doppler tissue imaging for determination of myocardial strain. While remaining preload dependent, both strain and strain rate imaging are more sensitive and earlier indicators of abnormal myocardial function than is assessment of wall thickening alone. This has been demonstrated experimentally as well as during spontaneous or induced myocardial ischemia.

A significant limitation to analysis of strain or strain rate is the heterogeneity of normal values within the myocardium as well as patient-to-patient variability resulting in a broad range of normal values. As such, subtle deviations from “normal” must be interpreted within clinical context and serial changes within a given patient may have more diagnostic value. Quantitation of myocardial strain is highly dependent on image quality, probably to a greater degree than less sophisticated quantitative techniques. While largely automated, significant user interaction is frequently necessary to ensure accurate myocardial tracking (Fig. 5.25). In studies with poor image quality, it may not be possible to obtain valid data.
FIGURE 5.22. Apical four-chamber view from which longitudinal strain has been obtained in seven segments. The image at the upper left is the apical four-chamber view. The mid myocardium is noted by the dotted line. Below the apical four-chamber view is a graphic representation of each of the seven segments as well as the global strain for the apical four-chamber view. The vertical line (AVC) denotes end-systole. At the lower right is a Doppler of the left ventricular outflow tract from which the time from onset of QRS to aortic valve closure has been calculated as 387 ms to define end-systole. At the upper right are the simultaneously obtained volumetric measurements of left ventricular volume from which the ejection fraction is calculated to be 62.2%.

FIGURE 5.23. A: Apical four-chamber view from which longitudinal strain has been recorded in 18 segments as per the current recommendation. The central figure is the apical four-chamber view with the myocardium automatically tracked
by speckle tracking technology. Individual strain data for the visualized six segments as presented in the graph. The table at the upper right presents volumes and ejection fraction. B: Bull's-eye plot of longitudinal strain obtained in the recommended 18 segments recorded in the same patient presented in panel A.

FIGURE 5.24. A: Longitudinal strain obtained in a patient with a dilated nonischemic cardiomyopathy. The apical long-axis view is presented. Individual graphs of the visualized seven segments are presented. B: Bull's-eye plot of the global longitudinal strain for 17 segments recorded in the same patient presented in A. Note the calculated ejection fraction of 25.6% and the markedly reduced global longitudinal strain of –6.9%. Video 5-24a
FIGURE 5.25. Apical four-chamber view recorded in a patient with less than ideal quality images. A represents an apical four-chamber view with superimposed tissue tracking for determination of longitudinal strain. This image is the first approximation created automatically by the ultrasound machine. Note that the lateral border is outside of the actual wall which is denoted by the arrow. Also note that the mid septum has been tracked in the right ventricular cavity. The inset at the lower right is the four-chamber view without tissue tracking. Note the prominent trabeculae on the right side of the septum (dual arrows) which were mistakenly tracked in the first approximation by the automatic system. The actual septum is delineated by the inward-pointing arrows. B is the same image after manual adjustment. Following this both the septal and lateral myocardium are more appropriately identified and tracked. Note the average strain of \(-21.6\) and ejection fraction of \(59.3\%\). Video 5-25a
The highly quantitative and detailed techniques of strain and strain rate analysis clearly detect abnormalities in myocardial contraction or deformation that are not apparent by visual analysis of wall motion characteristics. They remain limited by the technical and biologic factors discussed above but have shown promise as markers of preclinical disease in a number of conditions (Table 5.4). While reduced strain or strain rate may
be noted in many diseases, early in their course, and before abnormalities are otherwise detectable, a reduction in strain or strain rate remains nonspecific for any given disease, and in many instances the differential diagnosis includes two or more entities with similar early presentations. While technically feasible, calculation of radial and circumfractional strain has seen little clinical acceptance. Multiple studies have suggested that longitudinal strain and GLS are the more stable, reproducible, and clinically feasible calculations. For this reason, they have seen increasing clinical acceptance for evaluation of left ventricular function, determination of prognosis, and serial follow-up in a variety of clinical scenarios.

**Ventricular Torsion**

Normal contraction is a complex process involving contraction of circumferentially located myocardial fibers. In early systole, the left ventricle rotates clockwise (as viewed from the apex). Subsequently, the base of the heart continues with clockwise rotation and the apex develops counterclockwise rotation. This results in a “wringing” motion of the ventricle in systole. The degree of twisting of the heart varies with age and is altered in a variety of disease states. Loss of this normal wringing motion may be an early marker of preclinical cardiomyopathy. While recognition of this twisting motion of the heart allows more detailed recognition of myocardial mechanics in both diastole and systole, clinical application of this phenomenon has not yet been established. The twisting motion of the heart can be analyzed using either Doppler tissue imaging or speckle tracking and has likewise been confirmed with tagged, magnetic resonance imaging. **Figure 5.26** was recorded in a patient with normal left ventricular contractility using a hybrid speckle tracking system in which the clockwise rotation at the base of the heart and counterclockwise rotation at the apex are clearly demonstrated. This phenomenon can also be demonstrated using Doppler tissue techniques in which differential timing to peak velocity of subepicardial and subendocardial regions can be displayed as well as direction of motion in opposing walls from which the torsion can likewise be surmised. Rotation of the heart is described in degrees and when viewed as noted above, the normal myocardium has a positive rotation at the base and a negative rotation at the apex. The difference between the two represents the
total rotation which, when divided by the distance between the two analyzed segments, results in calculation of torsion defined as the twist in degrees divided by the distance (Fig. 5.27).
FIGURE 5.26. Parasternal short-axis view recorded at the base and apical levels in a patient with normal ventricular function. A modified speckle tracking algorithm has been used to track endocardial targets and displayed as a vector velocity map in which the length of an arrow represents magnitude of motion. The vector also demonstrates the direction of motion. Note in this normal example, the clockwise orientation of the velocity vectors at the base of the heart and the counterclockwise direction of the velocity vectors at the apex, consistent with normal “wringing” motion of the left ventricle (large curved arrows).

ASSESSMENT OF REGIONAL LEFT VENTRICULAR FUNCTION

Coronary artery disease, with its sequelae of myocardial ischemia, infarction, and chronic remodeling is the most common form of acquired heart disease encountered in adults. Coronary artery disease typically results in regional rather than global abnormalities, which requires a different approach to analysis from that used for assessment of global function (Table 5.5).

Normal ventricular contraction involves several simultaneous events. Myocardial fibers are oriented in a spiral fashion around the left ventricle. Contraction results in myocardial thickening and excursion of the endocardium toward the center of the ventricle. Simultaneous with this motion toward the center and cavity shrinkage is a twisting or wringing motion of the left ventricle. When viewed from the apex, there is initially a slight clockwise rotation of the entire heart after which the base of the left ventricle continues to rotate in a clockwise fashion and the apex rotates in a counterclockwise fashion. This systolic wringing motion of the left ventricle is an intrinsic component to myocardial contractility and efficiency. In diastole, the twisting motion of the heart is reversed and the early untwisting is largely responsible for early diastolic suction.
FIGURE 5.27. Graphic demonstration of angular rotation extracted from a normal patient in the upper panels and a patient with left ventricular hypertrophy (LVH) and reduced left ventricular ejection fraction (LVEF) in the lower panels. The left-hand panels are recorded at the apex and the right-hand panels at the base of the left ventricle. In the normal patient, note the positive 16.7-degree rotation at the apex and the –5.4-degree rotation at the base resulting in a total twist of 22.1 degrees and torsion of 2.6 degrees/cm compared to reduced values in the patient with reduced left ventricular function.

Using M-mode or standard two- or three-dimensional echocardiography, only myocardial thickening and endocardial motion toward the center of the ventricle are appreciated. Abnormalities of thickening and endocardial motion are reliable indicators of myocardial ischemia or infarction and their detection remains the mainstay of diagnosis of ischemic syndromes. There is regional and temporal heterogeneity of this motion, with the proximal inferoposterior and lateral walls contracting slightly later than the septum and
anterior walls. There is also normal heterogeneity of the degree of endocardial excursion and myocardial thickening, with greater absolute and percentage changes from diastole to systole at the base when compared with the apex.

Most commonly, abnormal regional wall motion is the result of coronary artery disease which interrupts perfusion to fairly well-defined territories and hence results in abnormal motion in those segments. There is a gradation of wall motion abnormality that consists progressively of hypokinesis, akinesis, and subsequently dyskinesis in which a wall moves away from the center of the ventricle. Because wall thickening and endocardial motion are intrinsically tied, virtually all regional wall motion abnormalities are initially associated with abnormalities of thickening as well as endocardial motion.

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<tr>
<th>Table 5.5</th>
<th>METHODS FOR EVALUATION OF REGIONAL WALL MOTION ABNORMALITIES</th>
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<td>Visual/subjective</td>
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<tr>
<td>Descriptive: normal, hypokinetic, akinetic, dyskinetic</td>
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<td>Myocardial strain</td>
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FIGURE 5.28. Schematic representation of the 17-segment model of the left ventricle. Parasternal and apical views are depicted. For the 16-segment model, each of the distal segments (13 to 16) incorporates its adjacent portion of the apical segment. For each segment, the coronary distribution most likely responsible for wall motion abnormality in that area is noted. When more than one coronary territory is listed, overlap between coronary distributions is anticipated in that segment. The true apex is most often perfused by the left anterior descending coronary artery; however, in the presence of a dominant right or circumflex
Regional wall motion abnormalities should be described in a standardized manner. Figure 5.28 schematizes the 17-segment model for description of regional wall motion currently recommended by the American Society of Echocardiography. Previous schemes used a 16-segment model, which includes a portion of the true apex in each of the four distal segments. A shortcoming of the 16-segment model is that if an abnormality is isolated to the apex, it is represented in each of four separate segments, thus resulting in a disproportionate contribution to the wall motion score, especially if the abnormality was limited to the “true” apex. The 17th segment represents the true apex. Addition of the 17th segment allows more precise comparisons with other imaging modalities, such as cardiac magnetic resonance imaging, computed tomography, or radionuclide perfusion techniques. Depending on the size of an apical wall motion abnormality, it may either enhance the accuracy of the wall motion score, if the abnormality is confined to the true apex, or result in overestimation if it involves portions of the four distal segments. When portions of the distal segments are involved, they will also be given an abnormal wall motion score, which again may result in disproportionate weighting of an apical wall motion abnormality.

The location of a wall motion abnormality is predictive of the location of the coronary “culprit” lesion in myocardial ischemia or infarction. Figure 5.28 also depicts the relationship of the predefined segments of the left ventricle to the traditional distributions of the left anterior descending, circumflex, and right coronary arteries. It should be emphasized that there can be substantial overlap in the more distal distributions of these arteries as well as in the posterior circulation in general. Following coronary artery bypass surgery, the location of wall motion abnormalities may be atypical, depending on the location of the myocardium perfused by the residual native arteries and by bypass grafts.

In clinical practice, the most common wall motion analysis is a segment-by-segment description of wall motion as either being normal, hypokinetic, akinetic, or dyskinetic. A numeric score (1, 2, 3, 4) is then ascribed to each segment, and a score index is calculated by summing the scores and dividing by the number of visualized segments. The techniques for calculating a wall motion score are discussed in Chapter 15.
Quantitative Techniques

There are a number of quantitative techniques for analyzing left ventricular regional function which have been used for investigational purposes but are rarely used in routine clinical practice. They are discussed here for historical purposes and because they often provide insight into the limitations of more advanced quantitative techniques. These include measurement of radian or area shrinkage in the short axis of the left ventricle. This is accomplished by describing a series of radians from the center of mass of the ventricle. The number of radians can range from 8 to 100, with each radian defined as the length from the center of mass to the endocardial border in diastole and subsequently in systole. Normal ventricular motion is represented by a reduction in the length of each of the constructed radians from diastole to systole (Fig. 5.29). In the presence of a regional wall motion abnormality, radians in the involved wall segment will lengthen rather than shorten (Fig. 5.30). In addition to radian length, sector area and myocardial thickness can be calculated using similar methods. Because of rotation of the heart in systole, there may not be exact correspondence of each radian position in diastole and systole, but rather the systolic length of a radian may be compared with the diastolic length of another. A more troublesome issue results from cardiac translation. Because there is motion of the center of the heart from diastole to systole, this results in motion and displacement of the systolic contour compared with the location of the diastolic contour. This has the effect of artificially shortening the radians that lie in the direction of translational motion and lengthening the radians in the opposite direction if the diastolic center of mass is used as a reference (Fig. 5.31). This can be corrected by realigning the center of mass of the contour before radian comparisons are made. When dealing with a normal, symmetrically contracting ventricle, this will correct for the errors attributable to cardiac translation. However, if a wall motion abnormality is present, the center of mass in diastole and systole will not be equivalent with respect to the distance from either the normal or abnormal walls. If one then corrects by using a separate center of mass, there will be predictable underestimation of the extent of wall motion abnormality.
FIGURE 5.29. Schematic diagram of normal endocardial wall motion without translational motion. **Top:** The *outer dark circle* represents the diastolic thickness of the left ventricle and the *inner lighter shaded circles* represent the extent of systolic contraction. Eight radians from the center of mass have been drawn for both the diastolic (*dotted line*) and systolic (*solid line*) endocardial boundaries. **Bottom:** The percentage of change in length from diastole to systole is schematized. The *dotted line* represents zero change in length and the *solid line* represents the actual percentage of change in length for the normally contracting ventricle, which in this example is a 20% reduction in length. This diagram is subsequently repeated for demonstration of wall motion abnormalities and
algorithms for correction of translation motion. In each subsequent similar figure, the darker outer ring represents the normal diastolic contour and the solid line represents the systolic endocardial contour.

FIGURE 5.30. Schematic demonstration of posterior dyskinesis with no translational or rotational motion using the diastolic center of mass for both systole and diastole. Top: The dark outer ring represents the contour of the ventricle in diastole and the inner circle represents the endocardial contour in systole. Note the maximal area of dyskinesis at segment five with less dyskinesis.
at segment four and akinesis at segment six. **Bottom:** The change in radian length from diastole to systole is graphed. Note the apparent hyperkinesis of the noninvolved segments with increased radian shortening compared with normal contraction in Figure 5.19.

Complicating any of the quantitative analyses of regional wall motion in ischemic disease is the phenomenon of tethering. This can occur on either a horizontal or vertical basis and occurs because the motion of a segment with intrinsically normal function may be altered by its proximity to an abnormal segment which “tethers” the adjacent normal segment and reduces its apparent function (**Fig. 5.32**). Regional wall motion abnormalities and the impact of coronary artery disease are discussed in more detail in **Chapter 15**.
FIGURE 5.31. Schematic representation of posterior dyskinesis with posterolateral translation, using separate diastolic and systolic centers of mass for determining radian length. Note that because there is posterior dyskinesis, the systolic center of mass moves toward the dyskinetic wall, resulting in an apparent reduction in the degree of dyskinesis when separate systolic and diastolic radian
lengths are then compared. This results in an artifactual underestimation of the severity of the wall motion abnormality and a simultaneous underestimation of function in the noninvolved zones.

**Determination of Left Ventricular Mass**

Echocardiography was one of the first imaging modalities used clinically for determination of left ventricular mass. It has seen widespread acceptance in epidemiologic studies of hypertension in which the presence of hypertrophy has been associated with worsened outcomes and its regression has been a goal of therapy. Left ventricular mass can be determined using a number of echocardiographic algorithms.

The earliest methodology for determining left ventricular mass was based on M-mode measurement of septal and posterior wall thickness and the left ventricular internal dimension. M-mode calculations assume a predefined ventricular geometry, and their accuracy will diminish in instances in which the left ventricular shape is abnormal. One of the methods for determining left ventricular mass is the cubed (Teichholz) formula, which assumes that the left ventricle is a sphere. The diameter of this sphere is the interior dimension of the left ventricle and the sphere wall thickness is that of ventricular myocardium. The formula calculates the outer dimensions of the sphere and then the inner dimension, the difference being the presumed left ventricular myocardial volume. The cubed formula is expressed as left ventricular mass = (interventricular septum + left ventricular interior dimension + posterior wall)$^3$ – left ventricular interior dimension$^3$ (Figs. 5.33 and 5.34). This then gives the volume of the stylized sphere of the myocardium, which, when multiplied by the specific gravity of muscle (1.05 g/cm$^3$), provides an estimate of left ventricular mass. Several investigators subsequently modified this approach using regression analysis. The cubed volume approach has the obvious limitation of determining ventricle size and wall thickness only along a single line and incorrectly assuming spherical ventricular shape. As it is common for the M-mode dimension to exceed the true minor-axis dimension, further error is introduced. Although the regression equations allow calculation of mass that correlates with autopsy specimens, there can be substantial error in the actual mass determination. The cubed methodology has been widely used, especially in serial evaluations, because for any given patient, the magnitude and direction of the
error is expected to remain constant.

In theory a more accurate determination of left ventricular mass can be obtained with two-dimensional echocardiography. When using two-dimensional echocardiography, geometric assumptions of the ventricular shape are typically still employed but the assumption is that of a bullet-shaped ventricle rather than a sphere. In addition, mean left ventricular wall thickness is determined rather than wall thickness at only one point on the septum and posterior wall. Mean wall thickness can be calculated by determining the epicardial and endocardial areas of the short axis of the left ventricle at the midcavity level. The difference between these two areas then represents myocardial area. Left ventricular mass can then be calculated either by an area length method or by assuming a truncated ellipse geometry (Fig. 5.35). More recently, three-dimensional echocardiography has been used to extract epicardial and endocardial borders from multiple orthogonal planes, from which left ventricular mass can also be determined. Limited studies have suggested excellent correlation of three-dimensional mass with anatomic and magnetic resonance imaging as standards. Determination of left ventricular mass by these techniques is problematic in less than ideal studies and is rarely used in clinical practice.
FIGURE 5.32. Schematic representation of horizontal tethering. This diagram represents posterior dyskinesis without translational motion. Note that the true extent of the infarct is as noted in the darkly shaded area, encompassing radian five and parts of radians six and four. Note that there is a border zone (lightly shaded area) adjacent to the infarct area that is anatomically normal but has abnormal motion due to the tethering effect of posterior dyskinesis. In the schematic, the true anatomic defect represents 20% of the circumference of the left ventricle with the tethered border zone giving an apparent total extent of 30%.

FIGURE 5.33. Schematic representation of the cubed formula for determining left ventricular mass. All measurements can be taken from either a two-dimensional
or an M-mode echocardiogram of the minor axis of the left ventricle. The formula for calculation of left ventricular mass is as noted. Based on comparison with anatomic specimens, several regression equations have been developed that are variations on the basic cubed formula. LVIDd, left ventricular internal dimension in diastole; PW, posterior wall.

**FIGURE 5.34.** Two-dimensionally guided M-mode echocardiogram recorded in a patient with mild hypertension. Note in the small inset, the tangential M-mode interrogation beam which is a result of beam orientation and slight angulation of the heart. The M-mode is as displayed from which a left ventricular internal dimension of 5.77 cm is measured. The true minor-axis dimension of the left ventricle is 4.7 cm. The bottom panel represents the calculated M-mode report from the measured values. The numbers in parentheses are the corresponding values from a true minor-axis dimension (4.7 cm) used rather than the off-axis 5.77 cm. Note the substantial overstatement of left ventricular mass using the dedicated M-mode measurement versus a true minor-axis dimension from the two-dimensional echocardiogram.

**Physiologic Versus Pathologic Hypertrophy**
Left ventricular hypertrophy can be characterized as concentric, eccentric, or physiologic (Fig. 5.36). It should be emphasized that calculation of left ventricular mass is a determination of the mass of the left ventricular muscle and may not relate to overall cardiac enlargement. Increases in left ventricular mass can occur with chamber enlargement and relatively normal wall thickness (eccentric hypertrophy), as is seen in regurgitant valvular lesions, or secondary to a predominant increase in wall thickness with normal chamber sizes, as is seen in the pressure overload of systemic hypertension. When evaluating patients for left ventricular hypertrophy, it is important to characterize the hypertrophy as being due to either chamber enlargement or increased wall thickness. One additional index of hypertrophy is relative wall thickness which is defined as (posterior wall thickness + interventricular septal thickness)/left ventricular internal dimension. Relative wall thickness of ≥0.42 has been used as a threshold of pathologic left ventricular hypertrophy.

**FIGURE 5.35.** Illustration of methodology for calculating left ventricular mass using two-dimensional imaging. In the central figure note that the epicardium and endocardium have been outlined in the short-axis view of the left ventricle from which mean wall thickness is calculated (dotted lines). At the lower right is an apical four-chamber view in which the length of the left ventricle has been
measured. At the upper left are the calculations of left ventricular mass using an area length (A/L) method.

**FIGURE 5.36.** Graphic demonstration of normal geometry, concentric remodeling, concentric hypertrophy, and eccentric hypertrophy. American Society of Echocardiography recommended thresholds for defining hypertrophy are as noted.

An additional form of hypertrophy is the physiologic hypertrophy seen in highly trained athletes. In general, this is a physiologic adaptation in which there is a slight increase in both wall thickness and chamber dimension. Wall thickness more than 13 mm is unusual in physiologic hypertrophy. Because the hypertrophy is a physiologic adaptation to physical training, wall stress tends to be normal. Physiologic hypertrophy seen in athletes regresses relatively quickly after cessation of vigorous training and, as such, can be differentiated from pathologic hypertrophy seen in hypertrophic
cardiomyopathy if imaging is repeated after a period of deconditioning.

**MISCELLANEOUS TECHNIQUES FOR EVALUATION OF LEFT VENTRICULAR FUNCTION**

There are a number of miscellaneous techniques for evaluating left ventricular regional or global function which have been developed over the years. Some remain valid for routine or selective use in a contemporary echocardiography laboratory, while others have only limited utility or are exclusively used for research purposes.

**Tissue Doppler Color M-Mode**

Using colorized Doppler tissue imaging an M-mode line can be directed through the ventricle and a color Doppler M-mode of tissue motion acquired (Fig. 5.37). This technique has shown utility in describing the timing of wall motion abnormalities. In the presence of a left bundle branch block, clear alternation in blue-red colorization of the septum is seen in patients with multiphasic septal motion related to conduction disturbances.

**Myocardial Performance Index**

A rapidly determined index of ventricular function has been derived by comparing the total systolic time from mitral valve closure to mitral valve opening with the systolic time involved in actual aortic flow (ejection time). Figures 5.38 and 5.39 illustrate the calculation of this index. The total systolic time is defined as isovolumic contraction time (IVCT) ± ejection time + isovolumic relaxation time (IVRT). The myocardial performance index (MPI) essentially divides the total isovolumic times (IVCT + IVRT) by the ejection time. This index, referred to as the MPI or “Tei index,” combines features of both systolic and diastolic function and has been shown to correlate with outcome in ischemic and nonischemic disease states. Normal MPI is less than 0.40 with progressively greater values implying progressively worse ventricular function.
FIGURE 5.37. Color tissue Doppler M-mode echocardiograms recorded in a normal patient in the upper panel and in a patient with anteroseptal dyskinesis in the lower panel. Notice in the upper panel, the abrupt blue colorization timed with the QRS (downward-pointing arrow) of the ventricular septum as it moves posteriorly followed by an abrupt change to red colorization representing anterior motion at the end of systole (upward-pointing arrow). Notice in the posterior wall the red colorization representing anterior motion of the normally moving posterior wall (double-headed arrow). The lower panel was recorded in a patient with an anteroseptal infarct and septal dyskinesis. Notice the similar appearance of the posterior wall colorization with normal red coloring encoding in systole (double-headed arrows) but the similar red encoding for the ventricular septum representing dyskinesis (small arrows). PW, posterior wall.
FIGURE 5.38. Schematic outlining calculation of the myocardial performance index (MPI). The myocardial performance index is the ratio of the sum of the isovolumic contraction and relaxation times (IVCT, IVRT) to ejection time (ET). It can be calculated by subtracting ET from total systolic time (TST) as noted in the two alternate formulas. Normal MPI is ≤0.40.
FIGURE 5.39. Composite illustration of myocardial performance index (MPI) calculated in three different patients. For each patient, the mitral inflow and left ventricular outflow tract velocities are provided from which the time from mitral closure to mitral opening and ejection time are calculated. The upper panels were recorded in a normal individual with mild hypertensive cardiovascular disease and an ejection fraction of 63% who has a normal MPI of 0.34. The middle panels were recorded in a patient with a mild dilated cardiomyopathy, ejection fraction of 30%, and more severe diastolic dysfunction. Note the MPI of 0.69. The bottom panels were recorded in a patient with a severe dilated cardiomyopathy, pseudonormal mitral filling related to Grade 2 diastolic dysfunction, and ejection fraction of 22%. Note the calculated MPI of 1.0.

Determination of Left Ventricular $dP/dt$

An additional parameter of left ventricular global function is left ventricular $dP/dt$ which has long been a standard calculation using a high-fidelity micrometer catheter in the catheterization laboratory. $dP/dt$ represents the rate of increase in pressure within the left ventricle. If confined to early systole, during isovolumic contraction, it is a relatively load-independent measure of ventricular contractility.

Using the spectral display of a mitral regurgitation jet, it is possible to derive similar information regarding the rate of pressure development within the left ventricle. If this measurement is undertaken in early systole while the
increasing ventricular pressure is less than the aortic pressure, it is relatively load independent. The method by which this is performed is to record the mitral regurgitation spectral profile at a high sweep speed (typically 100 mm/s), as shown in Figures 5.40 and 5.41. Examination of the early velocity curve can then be used to derive instantaneous pressure measurements. To determine the $dP/dt$, one calculates the time difference in milliseconds from the point at which the velocity is at 1 m/s and at 3 m/s. The time between these two points represents the time that it takes for a pressure change of 32 mm Hg to occur in the left ventricular cavity. $dP/dt$ is then calculated as $dP/dt = 32 \text{ mm Hg/ time (ms)}$. Determination of $dP/dt$ using this method has been validated against invasive hemodynamics. In addition to determining this parameter in early phases of systole, the negative $dP/dt$ over the analogous pressure change (36 to 4 mm Hg) in diastole can also be calculated and provides information regarding diastolic function. Either a reduced positive or negative $dP/dt$ carries significant prognostic implications. There are contributors to left ventricular $dP/dt$ in addition to intrinsic myocardial contractility. In the presence of marked mechanical dyssynchrony (as typified by left bundle branch block), $dP/dt$ may be reduced, not due to intrinsically decreased myocardial contractility but rather as a consequence of contractile dyssynchrony and overall pump inefficiency.
FIGURE 5.40. Schematic representation and example of calculating the left ventricular dP/dt from the continuous-wave Doppler mitral regurgitation spectral signal. **Left:** A continuous-wave spectral Doppler image recorded in a patient with severe left ventricular systolic dysfunction in which the online measurement of dP/dt is noted to be 482 mm Hg/s. **Right:** The methodology for this determination, which includes recording continuous-wave Doppler imaging of mitral regurgitation at a high sweep speed (150 mm/s in this example) and defining points for which the mitral regurgitation velocity has reached 1 and 3 m/s, is depicted. This represents a 32 mm Hg/s pressure increase in the left ventricle into a low-compliance left atrium, thus making this a relatively load-independent measure of contractility. The time between the two points required to reach 1 and 3 m/s (Δt) is then divided into the pressure difference (32 mm Hg) for calculation of dP/dt.
**Left Ventricular Wall Stress**

Most clinically used parameters of ventricular function including stroke volume and ejection fraction are afterload dependent, that is, they are dependent on the pressure developed and impedance against which the left ventricle must contract. Several methods have been proposed for correcting afterload or creating afterload-independent indices of left ventricular performance, including calculating ventricular wall stress and creation of pressure volume loops.

These calculations have been used as a measure of myocardial contractility in the investigation of cardiomyopathy and valvular heart disease. Because it accounts for wall thickness and pressure generation, wall stress is more afterload-independent than parameters such as fractional shortening or ejection fraction. Left ventricular stress can be calculated either globally or regionally. There are three different regional stress calculations: radial, circumferential, and meridional, each of which is mutually orthogonal. In its simplest form, meridional stress is defined by the formula: stress = (pressure × radius) ÷ h (where h = wall thickness) (Fig. 5.42). This formula assumes spherical geometry, which obviously is not the case in the left ventricle. As such, while correlating with other measures of left ventricular stress, it may not truly represent the actual value. Regional stress can be calculated along any of the ventricular segments using a similar equation for which the radius is independently determined for that segment rather than for the left ventricular cavity as a whole. Because of left ventricular–right ventricular interaction and changes in the radius of curvature of the ventricle, regional stress varies from apex to base and around the circumference of the left ventricle. Calculation of stress indexed to ventricular volume has been used as an index of ventricular performance in valvular heart disease and cardiomyopathy. In this instance, it is an additional refinement of the determination of left ventricular reserve and ventricular compensation in either pressure or volume overload states. A final highly detailed assessment
of left ventricular contractility involves creation of a pressure volume loop, which provides load-independent information regarding ventricular contractility. This can be accomplished by exporting instantaneous volume data from automatically determined borders and combing the continuous volume data with simultaneously determined pressure recordings.

**Doppler Evaluation of Global Left Ventricular Function**

Clinicians have used Doppler spectral profiles to evaluate global left ventricular function since the early 1970s. The earliest, conceptually simplest, and still probably one of the more clinically useful methods for following left ventricular function with Doppler is to evaluate the time velocity integral (TVI) of the left ventricular outflow tract or ascending aorta. Basically, the principle is that if the cross-sectional area of flow is known, then the product of that cross-sectional area and the mean velocity of flow equals the volumetric flow (Fig. 5.43). Typically, the area evaluated for determination of systolic flow, and hence global left ventricular performance, has been the left ventricular outflow tract. The calculated stroke volume can be multiplied by the heart rate to obtain cardiac output. The principles and limitations of this measurement are further discussed in Chapter 8.

Although measurement of the actual outflow tract area may be subject to significant error, there are no commonly encountered disease states in which the area of the outflow tract would be expected to change over a short period. With this in mind, the outflow tract area can be considered a constant over time in most patients. In this instance, the TVI is the only variable to change over time, and therefore calculation of this value alone can be used to track serial changes in forward flow. Figure 5.44 was recorded from patients with various disease states and shows the range in TVI values that can be encountered. Note in panels C and D the variation in TVI is due to rhythm disturbances.
FIGURE 5.42. Schematic representation of the simplified methods for determining left ventricular wall stress. Wall stress can be defined as radial, circumferential, or meridional, all of which are mutually orthogonal. Meridional wall stress is the simplest to calculate. Circumferential wall stress incorporates the length of the left ventricle and is best calculated from the two-dimensional echocardiogram. Bottom: The relationship of location to regional stress with respect to variation of wall thickness \((h)\) and local radius of wall curvature \((r)\) is depicted.

In theory, these same principles can be applied to any of the four cardiac valves or outflow or inflow tract dimensions. The right ventricular outflow tract, just below the pulmonic valve, provides information analogous to that for the left ventricular outflow tract. Comparison of the TVI-outflow tract area product at these two sites has been successfully used in congenital heart
disease to compare right and left ventricular stroke volume and hence
determine shunt ratios in patients with intracardiac shunts. In theory, similar
calculations can be performed using either the mitral valve annulus or an
average mitral valve area. In practice, determination of the cross-sectional
area of the annulus or of the mitral valve orifice is more problematic than
determination of an outflow tract area and quantitative flow calculations from
the mitral valve are rarely used in clinical practice.

FIGURE 5.43. Schematic representation of the method for determining
volumetric flow. This method is applicable for any laminar flow for which the
cross-sectional area (CSA) of the flow chamber can be determined. The product
of cross-sectional area and the time velocity integral (TVI) is stroke volume (SV).
Cardiac output (CO) can be calculated as the product of stroke volume and heart
rate. See text for further details.

NONISCHEMIC WALL MOTION ABNORMALITIES

There are several commonly encountered variations on wall motion
abnormalities that deserve comment and which may be confused for ischemia
or infarction (Tables 5.6 and 5.7). Tardokinesis refers to the delayed
contraction of a segment of the left ventricle, typically occurring in the final
50 to 100 ms of mechanical systole. Tardokinesis is most often noted in the
proximal inferior or posterior wall. It should be distinguished from
postsystolic contraction which can be seen in an ischemic segment and may
be most reliably detected with strain imaging. Isolated tardokinesis is rarely a
manifestation of myocardial ischemia and is most often seen at high heart rates in the stress phase of a stress echocardiogram. Another potentially confusing segmental wall motion finding is early relaxation, in which a segment relaxes or moves outward before the rest of the chamber. This finding is generally considered a normal variant. It is noted most often with stress echocardiography at high heart rates in individuals with preserved exercise tolerance. It is most commonly seen in the distal septum and apex and its location may not fit anticipated coronary anatomy. Typically one notes an abrupt relaxation of the ventricular wall occurring while the mitral valve is closed. The duration of early relaxation rarely exceeds 50 to 100 ms (Fig. 5.45).

Left bundle branch block alters the sequence of electrical activation and hence the sequence of contraction of the left ventricle. Normally, conduction down the left bundle precedes that down the right bundle by 10 to 20 ms, and hence the normal initial activation of the heart is in the proximal midseptum on the left ventricular side. In general, after this initial activation, there is relatively smooth progression of activation of contraction. In the presence of a complete left bundle branch block, the initial septal activation sequence is reversed and the right side of the ventricular septum is initially activated. This causes right septal activation before activation of the body of the left ventricle and results in initial right-to-left (anterior to posterior) movement of the ventricular septum.

**FIGURE 5.44.** Time velocity integral (TVI) in the left ventricular outflow tract recorded in four different patients. **A:** Note the TVI of 27 cm recorded in a patient
with normal cardiac function and a diminished TVI of 10 cm recorded in a patient with a cardiomyopathy and reduced stroke volume (B). C: The variation in TVI seen in a patient with severe left ventricular systolic dysfunction. The first beat to the left is a post–premature ventricular contraction (PVC) beat showing augmentation. Note the alternating TVIs after this beat, which is the corollary of pulsus alternans. D: Recorded in a patient with mild valvular aortic stenosis. Note the augmented peak velocity and TVI after the compensatory pause after a PVC (complex 3). Also note the marked reduction in both velocity and TVI for the PVC beat. In this instance, only the TVI and peak velocity associated with beat number 1 represent the true gradient.

The wall motion abnormality associated with left bundle branch block may be most easily appreciated with M-mode echocardiography (Fig. 5.46). It consists of initial downward motion of the ventricular septum followed by anterior or paradoxical septal motion and then subsequent thickening of the ventricular septum and posterior motion toward the center of the heart. The magnitude of this abnormal motion can be subtle and is occasionally noted only on detailed inspection of an M-mode sweep through the ventricular septum. On two-dimensional echocardiography, it may be noted as a “bounce” in the septum. In other instances, there will be a dramatic “paradoxical” motion of the ventricular septum. This range in activation abnormality is due to the variation in the degree to which left bundle branch block has delayed activation, the presence or absence of more distal His-Purkinje system disease, and the impact of concurrent disease that may either mask or exaggerate the bundle branch block pattern. Another characteristic of the left bundle branch block pattern is that the magnitude of the abnormality is often increased during pharmacologic stress with dobutamine. It is less often noted to be augmented during the physiologic stress of exercise. In a subset of patients, the mechanical dyssynchrony results in deterioration of ventricular function and a cardiomyopathic syndrome ensues. This can be reversed with biventricular pacing.

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<tr>
<td>Left bundle branch block</td>
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<td>Ventricular pacing</td>
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<tr>
<td>Premature ventricular contractions</td>
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<tr>
<td>Ventricular pre-excitation (Wolf–Parkinson–White syndrome)</td>
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<td>---------------------------------------------------------</td>
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<tr>
<td>Abnormal ventricular interaction</td>
<td></td>
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<tr>
<td>Right ventricular volume overload</td>
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<td>Right ventricular pressure overload</td>
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<tr>
<td>Pericardial constriction</td>
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<tr>
<td>Miscellaneous</td>
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<td>Tardokinesis</td>
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<td>Early relaxation</td>
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<td>After cardiac surgery</td>
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<td>Congenital absence of the pericardium</td>
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<td>Posterior compression</td>
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<td>Ascites</td>
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<td>Hiatal hernia</td>
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<td>Pregnancy</td>
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FIGURE 5.45. Apical four-chamber view recorded in a young, healthy individual immediately postexercise, demonstrating early relaxation of the apical septum. The upper panel (A) was recorded at end-systole and shows normal hyperdynamic motion of all visualized segments. The lower panel (B) was recorded 50 ms later and reveals abrupt outward motion of the apical septum (arrows) consistent with early relaxation. Note that the mitral valve remains
closed. In the subsequent frame, the remaining walls relax normally as well.

<table>
<thead>
<tr>
<th>Abnormality</th>
<th>Location</th>
<th>Onset</th>
<th>Duration</th>
<th>Thickening</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left bundle branch block</td>
<td>Anterior septum</td>
<td>Early systole</td>
<td>Multiphasic</td>
<td>Blunted</td>
</tr>
<tr>
<td>Paced rhythm</td>
<td>Distal septum</td>
<td>Early systole</td>
<td>Multiphasic</td>
<td>Blunted</td>
</tr>
<tr>
<td>Postoperative motion</td>
<td>Whole heart</td>
<td>Early systole</td>
<td>Whole cycle</td>
<td>Preserved</td>
</tr>
<tr>
<td>Ventricular pre-excitation (WPW)</td>
<td>Variable</td>
<td>Presystolic</td>
<td>Very brief (&lt;50 ms)</td>
<td>Preserved</td>
</tr>
<tr>
<td>Constriction</td>
<td>Septum/posterior wall</td>
<td>Diastole</td>
<td>Last 3/4</td>
<td>Preserved</td>
</tr>
<tr>
<td>Ischemia/infarction</td>
<td>Distal &gt; proximal</td>
<td>Early systole</td>
<td>All systole</td>
<td>Absent</td>
</tr>
</tbody>
</table>

A common scenario is for there to be a left bundle branch block in a patient for whom coronary artery disease is a diagnostic consideration. Separation of the wall motion abnormality due to the bundle branch block from the effects of coronary disease involving the left anterior descending coronary artery can be problematic, especially for the less experienced echocardiographer. Table 5.7 outlines a number of features that can help separate left bundle branch block and other nonischemic abnormalities from an ischemic wall motion abnormality. It should be emphasized that none of these features is absolute, and even experienced echocardiographers may have difficulty in separating a left bundle branch block wall motion abnormality from an ischemic wall motion abnormality. It should also be recognized that left bundle branch block may coexist with resting ischemia, myocardial infarction, or inducible ischemia at the time of cardiovascular stress. Perhaps the most valuable observation when attempting to separate left bundle branch block from ischemia is myocardial thickening. With left bundle branch block, myocardial thickening is typically preserved as is initial early ventricular contraction. By using M-mode echocardiography, or
confining wall motion analysis to the first half or third of systole, one can often appreciate that systolic thickening is preserved. Additional valuable clues include the fact that ischemia involving the proximal left anterior descending coronary artery, which would be required to result in a proximal septal abnormality, will usually result in distal abnormalities as well. In most instances, left bundle branch block does not result in abnormalities in the apex or distal anterior wall. This can be a valuable clue to the etiology of the wall motion abnormality. Right bundle branch block does not alter the initial sequence of activation of the left ventricle and, unless associated with intrinsic disease of the right heart, will not be associated with appreciable wall motion abnormalities.

**FIGURE 5.46.** M-mode echocardiogram recorded in a patient with a left bundle branch block. In the M-mode note the abrupt downward motion of the ventricular septum shortly after the onset of the QRS (arrows) and the relatively neutral position of the ventricular septum throughout the remainder of systole. In the accompanying parasternal long-axis two-dimensional image note the quivering of the septum in real time consistent with the left bundle branch block abnormality.
Premature Ventricular Contractions

A premature ventricular contraction (PVC) results in segmental wall motion abnormality for the beat in which the left ventricle is activated by the PVC. The most extreme example is a PVC arising in the lateral wall that is temporally and anatomically as remote from normal contraction as possible. In this instance, there will be immediate myocardial thickening and contraction of the lateral wall, occasionally resulting in dyskinesis of the relaxed septum, followed by asynchronous contraction of the left ventricle. High temporal resolution, two-dimensional echocardiography can be used to identify the site of earliest mechanical activation. In practice, a skilled echocardiographer should rarely be confused by wall motion abnormalities arising from PVCs. Scrutiny of the accompanying electrocardiogram is obviously informative, and the nature of the wall motion abnormality is frequently inconsistent with the known distribution of coronary or other forms of commonly encountered heart disease. Appreciation of the secondary effects of PVC is important. After a PVC, there is a “compensatory pause” and the subsequent left ventricular contraction is normally hyperdynamic (Fig. 5.47). It is important to appreciate this phenomenon so as not to then compare normal sinus beats and assume that the ventricle is hypokinetic. On occasion, an echocardiogram is performed in a patient with persistent bigeminy or trigeminy. This can result in confusion because each PVC will be accompanied by abnormal wall motion and frequently hypokinesis of the remaining walls, related to underfilling during the shortened preceding
diastole. The wall motion of the beat, following the compensator pause, will then be hyperdynamic. The third beat, representing normal contraction, provides the only assessment of true normal ventricular contractility. This issue may be especially problematic when viewing single digital cardiac cycle cine loops, where the relationship of systole function to rhythm may not be obvious. The augmented post-PVC beat will also be associated with increased flow velocity and TVI in the left ventricular outflow tract (Fig. 5.48).
FIGURE 5.47. M-mode echocardiogram recorded in a patient with ventricular bigeminy. The upper panel was recorded during bigeminy and reveals an abnormal contraction pattern of the ventricular septum (arrow) coincident with the PVC. The internal dimension (double-headed arrows) in diastole and systole for the post-PVC beat is noted from which a fractional shortening of 0.45 is calculated. The lower panel was recorded in the same patient during an arrhythmia-free period. Note the normal contractile pattern of the septum and posterior wall and the consistent fractional shortening of 0.33. The increased fractional shortening in the post-PVC beat is related to hyperkinetic motion following a post-PVC pause.

Video 5-47

Ventricular Pacing

The majority of ventricular-paced rhythms are done with apically located right ventricular endocardial leads. This results in a left bundle branch block pattern on the electrocardiogram, and a wall motion abnormality similar to that seen in native left bundle branch block. Many of the same rules regarding preservation of thickening and of late systolic endocardial motion discussed previously also pertain to evaluating wall motion in the presence of a paced rhythm. Because most endocardial pacing leads are placed apically, the location of maximal abnormality previously referred to is far less helpful. On occasion, a ventricular pacing lead can be in the more inferior portions of the distal septum and result in a distal inferior wall motion abnormality (Fig. 5.49). Separation of this wall motion abnormality from that due to true ischemia can occasionally be problematic. It has become standard therapy to use biventricular pacing for mechanical resynchronization in patients with underlying conduction system disease (typically left bundle branch block).
and systolic dysfunction. Resynchronization, via simultaneous biventricular pacing, results in more efficient mechanics of ejection and improved cardiovascular performance. The appearance of regional wall motion abnormalities in these patients will be highly variable and dependent on underlying conduction and the relative contributions of the two pacing sites. Caution is advised when attempting to diagnose an ischemic wall motion abnormality in this setting.

FIGURE 5.48. Continuous-wave Doppler in a patient with aortic stenosis and reduced left ventricular systolic function, demonstrating the impact of a PVC on left ventricular contractility. The central continuous-wave Doppler is a continuous recording. Note the PVC (upward-pointing arrow), after which there is a modest postcompensatory pause. The post-PVC beat has augmented contractility and stroke volume. This results in an increased aortic valve peak gradient of 71 mm Hg and a mean gradient of 40 mm Hg. The panel at the upper right was recorded during regular sinus rhythm at which time there was a stable peak gradient of 33 mm Hg and a mean gradient of 20 mm Hg. The numeric data at the upper left depict the gradient for the post-PVC beat in the upper portion and for the normal sinus rhythm in the lower portion.
FIGURE 5.49. Apical two-chamber view recorded in a patient with a right ventricular transvenous pacemaker. In the M-mode echocardiogram, notice the atypical pattern of septal motion consistent with a bundle branch block. In the apical two-chamber view, note the marked inferoapical wall motion abnormality in this patient, known to be free of coronary artery disease, related to pacing at the inferoapical aspect of the right ventricle.
Pericardial Constriction

Pericardial constriction results in a variety of wall motion abnormalities. The underlying reason for the abnormalities is exaggerated differential filling and contraction of the right and left ventricles. This alters the sequence and magnitude of septal position and motion. Superimposed on the beat-to-beat abnormality of septal motion can be exaggerated respiratory variation in septal position related to increased ventricular interdependence. Initial descriptions of abnormal wall motion in constrictive pericarditis were based on M-mode echocardiography, and one or two septal and posterior wall motion abnormalities were described as “typical” (Fig. 5.50). It quickly became apparent that there was a broad range of septal motion abnormalities, all of which resulted in an early downward deflection followed by varying degrees of “paradoxical” septal motion. Many of the septal motion patterns noted in constrictive pericarditis mimic right ventricular volume or pressure overload, septal pre-excitation, left bundle branch block, and, less commonly for the experienced observer, myocardial ischemia. This topic is discussed further in Chapter 9.
Ventricular Pre-Excitation

Ventricular pre-excitation, as typified by the Wolf–Parkinson–White syndrome, may result in segmental wall motion abnormalities which are more subtle than those seen with left bundle branch block or pacing. The abnormalities seen with pre-excitation are often in atypical locations that are not consistent with the anticipated location of coronary artery disease. The abnormalities associated with ventricular pre-excitation are highly localized and of very small magnitude and duration. They are often only appreciated with M-mode echocardiography, which has the ability to detect relatively small degrees of motion that occur over only a 10- or 20-ms period (Fig. 5.51). It should be emphasized that normal contraction typically begins after completion of the entire QRS. In most patients with pre-excitation, activation through the normal conduction system precedes in an orderly fashion and soon overtakes the wave of the pre-excited myocardium. Pre-excitation of the right ventricular myocardium is rarely detected with echocardiography, and it is more often the septal and posterolateral bypass pathways that are associated with visible wall motion abnormalities.

Postoperative Cardiac Motion

After any form of cardiac surgery in which the pericardium is opened, there is a characteristic abnormality of cardiac motion. This was initially appreciated
only as abnormal septal motion on M-mode echocardiography. Rather than being an isolated septal abnormality, this motion abnormality actually is a global phenomenon, involving exaggerated anterior motion of the entire heart within the thorax. The initial descriptions of this abnormality were in patients who had undergone valve replacement surgery. It soon became apparent that coronary artery bypass surgery also resulted in abnormal septal motion. Serial echocardiography during each sequential phase of cardiac surgery has demonstrated that the abnormality develops after any procedure in which the pericardium is opened and it may regress over 3 to 5 years.

The abnormal postoperative motion on M-mode echocardiography was noted as frank paradoxical motion of the ventricular septum with preserved myocardial thickening but without the initial downward deflection seen with a left bundle branch block. With two-dimensional echocardiography, it is easily appreciated that the center of the left ventricle moves anteriorly during contraction to an exaggerated degree. This has the effect of exaggerating apparent motion of the anteroposterior and posterolateral walls and of reducing the apparent motion of the anterior septum. Figure 5.52 was recorded in a patient with “paradoxical septal motion” after cardiac surgery. Note that septal thickening is preserved and that overall cardiac motion in the thorax is abnormal.
FIGURE 5.51. M-mode echocardiograms recorded in two patients with ventricular
pre-excitation due to the Wolff–Parkinson–White syndrome. A: A patient with a septal pathway is noted. Note the brief early downward systolic motion of the ventricular septum (arrow) slightly before the upstroke of the QRS. B: Note the very slight anterior motion of the posterior wall recorded in a patient with a posterolateral pathway due to Wolff–Parkinson–White syndrome (arrows). PW, posterior wall.

One early observation was that the absence of “paradoxical septal motion” after valve replacement surgery may be an indicator of prosthetic valve dysfunction. There were a number of case examples in which paradoxical septal motion failed to occur in the presence of prosthetic valve dysfunction, presumably due to the concurrent volume overload that mitigated against the development of abnormal motion. Reliance of this observation is obviously outmoded.

Evaluation of a postoperative, left bundle branch block or paced rhythm wall motion abnormality is often complicated by coexistence of any of these three entities plus concurrent myocardial ischemia or infarction. Combinations of these nonischemic wall motion abnormalities, each of which can result in a wall motion abnormality, obviously makes the interpretation problematic. Even experienced observers may have difficulty detecting a primary ischemic wall motion abnormality when two or more of these other situations are present. The single best tool for separating ischemic from nonischemic abnormalities is to rely heavily on the presence or absence of systolic wall thickening. Because many of these nonischemic abnormalities are confined to either the early or latter half of systole, evaluating a digitized two-dimensional echocardiogram only during the first half of systole may allow the echocardiographer to identify preserved thickening and normal endocardial motion. It is also important to have a firm understanding of the anticipated pathophysiology of underlying coronary artery disease. Many of the abnormalities discussed above result in an “anatomically incorrect” distribution of wall motion abnormalities, and a skilled clinician-echocardiographer should be in a position to recognize that a wall motion abnormality is a result of a nonischemic process based on its location, timing, and other characteristics. It should also be recognized that after successful coronary bypass surgery, the distribution of regional wall motion abnormalities might also be atypical.
FIGURE 5.52. Apical four-chamber view recorded in a patient after cardiac surgery demonstrates postoperative motion of the entire heart. A: Image was recorded in end-diastole. The vertical line marks the position of the right side of the ventricular septum. B: Image was recorded in end-systole. Note that, compared with the vertical reference line, there has been overall anterior (leftward) motion of the heart. Note the thickness of the ventricular septum (double-headed arrow).
**FIGURE 5.53.** Example of posterior wall pseudodyskinesis related to ascites. In the central image note that the ascites has compressed the posterior wall of the left ventricle (*arrows*) and that in diastole it has a “D-shaped” configuration. In early systole (upper left inset, note the ventricle has resumed normal circular geometry). In the real-time image note the apparent dyskinesis of the posterior wall in early systole followed by normal contraction.

Posterior Compression

Nonischemic abnormalities also include those occurring when there is extracardiac compression of the left ventricle. This can be seen when a structure such as an aneurysmal thoracic aorta or hiatal hernia compresses the heart or when there is compression from a subdiaphragmatic process such as ascites, abdominal masses, and pregnancy. In these instances, the inferior wall will be compressed superiorly resulting in a D-shaped distortion of left ventricular geometry when viewed in a short-axis view. The distortion is most prominent during diastole. With mechanical systole and myocardial contraction, the left ventricle reassumes normal circular geometry and the previously distorted wall appears to move paradoxically. **Figure 5.53** illustrates this phenomenon. Close attention to underlying coexisting pathology likely to result in this phenomenon, and to myocardial thickening, allows accurate identification of this artifactual wall motion abnormality. This phenomenon is quite similar to the “paradoxical” septal motion seen in a right ventricular volume overload in which there is diastolic deformation of the left ventricle with resumption of normal circular geometry in early
systole.

**Suggested Readings**

**GENERAL**


**BASIC QUANTITATION**


**THREE-DIMENSIONAL ECHOCARDIOGRAPHY**


**STRAIN AND STRAIN RATE IMAGING**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Chapter 6
Evaluation of Diastolic Function

Over the past three decades it has become apparent that diastolic dysfunction is a common contributor to symptoms of congestive heart failure and also carries significant prognostic importance which is additive to systolic dysfunction in patients with congestive heart failure. Both the definition of diastolic dysfunction and the echocardiographic parameters for its detection and characterization remain in evolution. Doppler evidence of delayed ventricular relaxation can be seen in over 40% of patients older than the age of 40 years and is often the consequence of the normal aging process, but is accentuated in patients with hypertension or diabetes. Diastolic dysfunction as the major contributor to congestive heart failure symptoms is also well-recognized and is an integral component to the diagnosis of heart failure with preserved ejection fraction.

The current recommendations for detection and grading of diastolic dysfunction stress the importance of integrating clinical with echocardiographic data. All patients presenting with symptoms suggestive of congestive heart failure, or who have underlying anatomy likely to result in congestive heart failure, should have a complete evaluation of diastolic function performed as part of a routine echocardiogram.

BASIC DIASTOLIC PHYSIOLOGY

Systole and diastole are intrinsically linked as the left ventricle alternately serves as pump and reservoir. For this reason, it is not appropriate to think of systole and diastole as separate and independent. Figure 6.1 illustrates some
of the differences between systolic and diastolic heart failure using pressure–volume loops. Although isolated systolic or diastolic dysfunction can occur, in most patients, elements of both contribute to the overall clinical status and symptom complex. Furthermore, many of the main causes of diastolic dysfunction are the same conditions that result in systolic dysfunction. Hypertension, coronary disease, and valvular heart disease are common causes of both conditions. In an individual patient with one or more of these diseases, detectable abnormalities of both systole and diastole frequently coexist although manifestations of one or the other may predominate.

![Pressure-volume loops](image)

**FIGURE 6.1.** Diastolic filling and systolic ejection can be demonstrated using pressure–volume loops. By tracing a loop counterclockwise, the entire cardiac cycle depicting the interplay between pressure and volume, is illustrated. In this example, the changes that occur with systolic versus diastolic heart failure (HF) are contrasted.

It is also important to recognize the contribution of both upstream and downstream factors, relative to the left ventricle, as contributors to diastolic function. Upstream, left atrial function has an important effect on left ventricular filling. Since the left atrium acts as both conduit and pump, its ability to transfer blood to the ventricle essentially defines left ventricular filling. This explains why left atrial volume is now established as a useful indicator of the presence, chronicity, and severity of left ventricular diastolic function.
dysfunction. Downstream, effective arterial elastance is related to both systolic and diastolic function of the left ventricle. Although afterload is more directly related to systolic function, it should be recognized that chronic elevation in arterial pressure will also affect left ventricular relaxation and chamber compliance.

Left ventricular diastole begins when the aortic valve closes and includes isovolumic relaxation, rapid early ventricular filling, diastasis, and left atrial contraction (see Fig. 6.2). The initial phase, prior to mitral valve opening, involves the rapid, energy-dependent relaxation of the left ventricular myocardium to its resting unstressed length. This process is associated with a brisk decline in left ventricular pressure. The interval between aortic valve closure and mitral opening is referred to as isovolumic relaxation. Once ventricular pressure falls below left atrial pressure (which is slowly rising as a result of pulmonary venous inflow), the mitral valve opens.

The next step involves filling the left ventricle as rapidly as possible without resulting in a significant increase in pressure. After the mitral valve opens, ventricular pressure continues to fall, creating a pressure gradient between the left atrium and left ventricle and blood is literally pulled through the mitral valve (Fig. 6.3). As the left ventricle begins to fill, the pressure within the chamber rises and the rate of inflow slows. Continued filling in mid diastole only occurs if the left ventricle is sufficiently compliant, or if left atrial pressure is sufficiently high, to allow the forward flow of blood. However, in most normal individuals, at physiologic heart rates, little additional filling occurs in mid diastole.

The final phase of left ventricular filling results from atrial contraction and ends with mitral valve closure. If diastolic pressure rises too quickly, left ventricular filling will be reduced and prematurely terminated. If a compensatory increase in left atrial pressure is required to maintain left ventricular filling, pulmonary venous pressure will rise as a result, leading to symptoms.

Conceptually, it is helpful to regard diastolic filling as a process of transporting blood through the mitral valve from one reservoir (the left atrium) to another (the left ventricle). This process depends on creating and maintaining a pressure gradient between the two chambers, the magnitude of which determines the rate of flow. Blood can either be pulled through the mitral valve, by rapidly lowering left ventricular pressure below left atrial
pressure (suction), or pushed through the valve by raising atrial pressure above ventricular pressure. Both occur in the normal heart. In early diastole, flow is initiated by the rapidly relaxing left ventricle resulting in a suction of blood from the left atrium, through the mitral valve. In late diastole, the continued forward flow of blood is accomplished by a pushing mechanism, the result of atrial contraction. The concept of pulling versus pushing blood through the mitral valve is fundamental to understanding some of the pathophysiologic principles of diastolic function, which are discussed below.
FIGURE 6.2. The four stages of diastole are illustrated in this schematic. The upper tracing illustrates the LV and LA pressure curves, while the lower tracing demonstrates the associated transmitral filling pattern, recorded with Doppler. Isovolumic relaxation begins with aortic valve closure (AVC) and ends with mitral valve opening (MVO), at which point, left ventricular filling begins. This is the result of a pressure gradient between the LA and LV, and is coincident with the mitral E wave. A period of diastasis, during mid diastole, is characterized by relatively little additional filling. In late diastole, atrial systole once again creates a
transmitral pressure gradient and results in the Doppler A wave, terminating with mitral valve closure (MVC). IVRT, isovolumic relaxation time.

**FIGURE 6.3.** The instantaneous pressure gradient across the mitral valve, between the left atrium and left ventricle creates flow which can be recorded using the Doppler technique. In early diastole, the rapid fall in left ventricular pressure (LVp) produces the E wave, while in late diastole, left atrial contraction produces the A wave. LAP, left atrial pressure.

**GRADES OF DIASTOLIC DYSFUNCTION**

The concept of grades to characterize the natural history and severity of diastolic dysfunction has both strengths and limitations. Because diastolic dysfunction is a progressive condition, it is helpful to think of it as
transitioning from mild to severe along a continuum. Although conceptually attractive, there are several problems with this approach. First, diseases rarely progress in discrete steps, but rather change gradually in continuous manner. Some patients will exist at the interface of two stages, with some features of both. Second, diastolic function is a constellation of hemodynamic and physiologic parameters, so no one variable fully defines “diastolic function” in a given patient, or at a given time. Third, none of the Doppler variables are accurate enough to be completely reliable in isolation. In both normal and abnormal groups, a range of values exists for each variable. Rarely do all the Doppler variables in an individual patient conform to one grade. Fourth, diastolic function is a complex, dynamic state that can improve or worsen rapidly. A Doppler study is taken at one point in time. In an unstable patient, that study will only be able to describe where the patient is currently, not where they were or which direction they are headed. Finally, while the severity of diastolic dysfunction tends to gradually worsen slowly over time, it can improve, usually as the result of treatment (e.g., diuresis, heart rate control, or blood pressure lowering). For these reasons, it is important to view the grades of diastolic dysfunction in contexts where precise labeling of all patients is not possible. Despite these considerable limitations, this approach is helpful in our understanding of the pathophysiology of diastolic dysfunction and, in many cases, to assist in the diagnosis and treatment of patients. The pathophysiology and typical clinical scenario expected with each grade of diastolic dysfunction are listed in Table 6.1.

**Normal Diastolic Function**

Diastolic function changes with age, so the Doppler criteria used to define normal and abnormal function must account for this factor. With aging, the left ventricle becomes stiffer and relaxes more slowly. Regardless of age, however, normal diastolic function can be characterized as the complete and efficient filling of the left ventricle at physiologic pressures. This implies that an abnormally high left atrial pressure is not required and that the left ventricle can fill completely without an associated abnormal increase in pressure during filling. Following isovolumic relaxation, the mitral valve opens and most filling occurs in the first third of diastole, the result of elastic recoil and active relaxation of the chamber. This phase corresponds to the
mitral E wave (Fig. 6.4A). This rapid early filling is associated with a similarly brisk motion of the mitral annulus as the chamber expands to accommodate the inflow of blood. This process can be recorded and quantified using tissue Doppler as the $e'$ (Fig. 6.4B). Little filling occurs in mid diastole, the diastasis, the duration of which is heart rate dependent; that is, it shortens or disappears with increasing heart rate. This is followed by atrial systole (the A wave), which contributes a relatively small amount of additional filling. As such, the A wave peak velocity and area under the curve (time velocity interval) are less than the E wave. As blood enters the ventricle through the mitral valve, it propagates rapidly toward the apex, a parameter that is evaluated using color Doppler M-mode, and termed the propagation velocity or Vp. Coincident with left ventricular filling, left atrial filling occurs via the pulmonary veins. Normal pulmonary venous flow consists of a systolic and diastolic component followed by a brief reversal of flow during atrial systole (Fig. 6.5). Finally, normal diastolic function is associated with a normal left atrial volume.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Name</th>
<th>Dominant Pathophysiology</th>
<th>Clinical Scenario</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Impaired relaxation</td>
<td>Delayed LV early active relaxation</td>
<td>Often asymptomatic at rest Dyspnea with exertion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Normal LA pressure</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Low opening LA-LV pressure gradient</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reduced LV suction force</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>Pseudonormalization</td>
<td>Delayed LV early active relaxation</td>
<td>Mild to moderate symptoms Exertional dyspnea common</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mildly elevated LA pressure</td>
<td>Symptoms variable, depending on volume status</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Low opening LA-LV pressure gradient</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reduced LV suction force</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>Restrictive filling</td>
<td>Noncompliant LV chamber (increased stiffness)</td>
<td>Moderate to severe symptoms, often at rest</td>
</tr>
<tr>
<td></td>
<td></td>
<td>May be HFpEF or HFrEF</td>
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</table>
**Impaired Relaxation (Grade I)**

For most patients who have diastolic dysfunction, the initial or earliest abnormality is termed *impaired relaxation*. This results from the loss of elastic recoil of the left ventricle in early diastole leading to a reduction in the force by which blood is sucked through the mitral valve. Hemodynamically, this leads to a delay or prolongation of the left ventricular pressure curve during isovolumic relaxation. This prolongation, in turn, causes a delay in mitral valve opening and a prolongation of the isovolumic relaxation time (IVRT). With the decrease in suction during early diastole, the left atrial to left ventricular (LA-LV) pressure gradient at the time of mitral valve opening is also decreased (Fig. 6.6). The rate of deceleration of early mitral inflow diminishes (i.e., deceleration time is prolonged, unless left ventricular stiffness is significantly increased) and the slope of the early diastolic flow propagation profile is also reduced. Antegrade flow across the mitral valve continues through mid diastole. In contrast, mitral flow velocity during atrial systole is increased. This occurs through a combination of increased atrial preload and a more forceful atrial contraction, a compensatory mechanism. The auscultatory equivalent of this is the S4 gallop. At this early stage, pulmonary venous flow and the E/e' ratio are usually normal and consistent with normal filling pressure at rest.

**Pseudonormalization (Grade II)**

With further deterioration of diastolic function, a decrease in chamber
compliance (increased stiffness) adds to the continued delay in relaxation. Transmitral flow is increasingly dependent on maintaining a high left atrial pressure rather than active relaxation (i.e., pushing as opposed to pulling blood into the left ventricle). This results in an increase in mean left atrial pressure which has two subsequent effects. First, it contributes to a shortening of IVRT. The reasons for this are illustrated graphically in Figure 6.7. Second, in contrast to impaired relaxation, the early mitral inflow velocity is restored back to the normal range. This increase is because the high left atrial pressure results in a larger LA-LV pressure gradient at the time of mitral valve opening. In most patients, left atrial contractility is maintained. As a result of these factors, the mitral inflow pattern appears similar to the normal state (Fig. 6.8). Thus, this phase is often referred to pseudonormalization. Pulmonary venous flow will usually show diastolic predominance (see Fig. 6.5B). A very small systolic wave (less than 50% of the diastolic wave) suggests elevated filling pressures. The important concept here is that the mitral inflow velocity pattern resembles the normal state due to the combined effects of high filling pressure and impaired relaxation.

**FIGURE 6.4.** In A, a normal mitral inflow velocity pattern is illustrated, demonstrating the E wave velocity greater than the A wave velocity. In B, the corresponding tissue Doppler recording of mitral annular velocity shows the e’ velocity greater than a’ velocity.
Restrictive Filling (Grade III)

With advanced diastolic dysfunction, left ventricular chamber compliance becomes increasingly abnormal. To maintain forward flow, left atrial filling pressure must continue to increase. This results in a further shortening of the IVRT and a marked increase in the early diastolic mitral inflow velocity (Fig. 6.9). Although the early mitral inflow velocity is very high, the rate of deceleration of flow is marked, the result of a noncompliant left ventricular chamber leading to a rapid equilibration of the LA-LV pressure gradient early in diastole. This pressure equilibration prevents the continuation of flow during mid diastole. Filling velocity during atrial contraction is also reduced through a combination of elevated left ventricular pressure and failing left atrial contractility. Pulmonary venous flow during systole is greatly reduced relative to diastolic flow and there is usually prominent flow reversal during atrial systole. The retrograde pulmonary venous A wave duration (Ar) is typically longer in duration than the mitral A wave (Ar – A >30 ms), indicating high filling pressures. This phase of diastolic dysfunction is called restrictive filling or restrictive physiology.

In some patients, this stage may be reversible. That is, with diuresis (or other forms of preload reduction), the restrictive filling pattern may revert one of the earlier stages of diastolic dysfunction, usually resembling pseudonormalization. This occurs because of an intervention that lowers left
atrial pressure and reduces the LA-LV pressure gradient.

**FIGURE 6.6.** Two examples of impaired relaxation are shown. In A, the mitral inflow pattern shows E/A reversal, with a ratio of 0.7. In B, the patient demonstrates low E wave velocity, prolonged E wave deceleration, and a large A wave. See text for details.

**FIGURE 6.7.** The effect of an increase in mean left atrial pressure on Doppler inflow velocity is demonstrated. On the left, in the setting of normal left atrial pressure, a typical mitral inflow velocity pattern is shown. On the right, when left atrial pressure is elevated, isovolumic relaxation time (IVRT) is reduced and an increased LA-LV pressure gradient results in a higher E wave. See text for details.

In the most severe forms, the pattern may be come irreversible. In such cases, preload manipulation no longer leads to an improvement in the filling pattern nor the clinical status. This late stage of irreversible restrictive
physiology is often associated with a marked intolerance to volume manipulation. These patients often survive within a very narrow range of volume tolerance. In such patients, maintaining the precarious balance between volume overload and hypoperfusion can be very difficult.

**ECHO DOPPLER PARAMETERS OF DIASTOLIC FUNCTION**

The stages of diastolic dysfunction can be characterized using various echocardiographic parameters which are summarized in Table 6.2. Note that each parameter reflects a specific component of diastolic function, but that no marker, by itself, completely captures all the information necessary to characterize an individual patient.

![FIGURE 6.8](image1.png)

**FIGURE 6.8.** An example of pseudonormal diastolic dysfunction is provided. In **A**, a “normal” E/A ratio is noted (1.25). In **B**, however, the annular velocity (e′) is markedly reduced at 5 cm/s, indicating delayed relaxation.
FIGURE 6.9. Restrictive physiology is demonstrated in this elderly patient with ischemic heart disease. In A, mitral inflow shows a high E wave velocity, short deceleration time, and an abnormal E/A ratio of 2.4. In B, a very low e’ velocity is noted.

Table 6.2  ECHO DOPPLER MODALITIES FOR EVALUATING DIASTOLIC FUNCTION

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Modality</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>IVRT</td>
<td>Pulsed Doppler</td>
<td>Information on LA pressure, rate of early active LV relaxation</td>
</tr>
<tr>
<td>Mitral inflow:</td>
<td>Pulsed Doppler</td>
<td></td>
</tr>
<tr>
<td>E/A ratio</td>
<td></td>
<td>Reflects LA-LV gradient in early and late diastole; helps define stages</td>
</tr>
<tr>
<td>Deceleration time</td>
<td></td>
<td>Information on LV chamber compliance</td>
</tr>
<tr>
<td>Response to Valsalva</td>
<td></td>
<td>Helps differentiate normal from pseudonormal stages</td>
</tr>
<tr>
<td>A wave duration</td>
<td></td>
<td>Combined with PVa wave, reflects LV filling pressure</td>
</tr>
<tr>
<td>Flow propagation velocity</td>
<td>Color M-mode</td>
<td>Reflects elastic recoil, rate of early diastolic LV relaxation; can be used to estimate pulmonary capillary wedge pressure</td>
</tr>
<tr>
<td>Annular velocity:</td>
<td>Tissue Doppler</td>
<td></td>
</tr>
<tr>
<td>e’</td>
<td></td>
<td>Correlates with early diastolic relaxation; distinguishes RCM from constrictive pericarditis</td>
</tr>
<tr>
<td>E/e’ ratio</td>
<td></td>
<td>Predicts LV filling pressure</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Pulsed</td>
<td></td>
</tr>
<tr>
<td>Flow:</td>
<td>Doppler</td>
<td></td>
</tr>
<tr>
<td>-------</td>
<td>---------</td>
<td></td>
</tr>
<tr>
<td>S/D ratio</td>
<td>Changes correlate with stages of diastolic dysfunction</td>
<td></td>
</tr>
<tr>
<td>A – Ar</td>
<td>Difference in duration of the two waves reflects LV filling pressure</td>
<td></td>
</tr>
<tr>
<td>LA volume</td>
<td>2D echo Information on presence and chronicity of diastolic dysfunction; prognostic value</td>
<td></td>
</tr>
</tbody>
</table>

PVa, pulmonary venous A wave; RCM, restrictive cardiomyopathy; S/D ratio, systolic to diastolic ratio; other abbreviations as in text.

**Isovolumic Relaxation Time**

IVRT measurement provides insight into the rate of early diastolic left ventricular relaxation. When relaxation is prolonged, mitral valve opening is delayed and IVRT is increased. Conversely, when left atrial pressure is elevated, mitral valve opening will occur earlier and IVRT will be shortened. These concepts are illustrated in Figure 6.10. IVRT does not directly measure the rate of relaxation, but rather the duration of relaxation prior to mitral valve opening. It is derived using pulsed Doppler from a modified apical four-chamber view. The goal is to adjust the image to allow simultaneous visualization of left ventricular inflow and outflow. Once this view is obtained the Doppler sample volume is placed midway between the inflow and outflow areas so that mitral and aortic flows are captured simultaneously (Fig. 6.11). The sample volume size can be adjusted to permit optimal recording, and generally a relatively large sample volume is best. IVRT is most easily obtained by measuring the time from middle of the aortic closure click to the onset of the E wave of mitral flow. Gain and wall filters should be adjusted to allow precise definition of aortic closure and mitral opening. In general, a fast sweep speed is used and measurements are performed during quiet respiration. At least three measurements of IVRT should be obtained and averaged.

IVRT is an indicator of the rate of myocardial relaxation. A major limitation is the fact that multiple factors influence the duration of the IVRT. For example, impaired relaxation lengthens IVRT while increases in left atrial pressure shorten IVRT. Furthermore, IVRT increases with age and is sensitive to changes in both heart rate and systolic function. All of these factors contribute to the nonspecificity of IVRT which should never be used in isolation as a predictor of diastolic function.
**Mitral Inflow**

An accurate recording of mitral inflow velocity is the single most important parameter for the assessment of diastolic function. The use of mitral inflow Doppler recordings to assess diastolic function is based on the premise that the velocity curve throughout the cardiac cycle reflects the instantaneous pressure gradient between the left atrium and ventricle (see Figs. 6.2 and 6.3). The greater the pressure difference, the higher the velocity at that point in time. If no gradient exists, then flow will cease. Thus, mitral inflow provides unique insight into left ventricular filling throughout the entire period of diastole.

![Diagram of Changes in relaxation rate and LA pressure](image)

**FIGURE 6.10.** This schematic demonstrates how left ventricular relaxation rate and changes in left atrial pressure affect isovolumic relaxation time (IVRT). See text for details. AVC, aortic valve closure; MVO, mitral valve opening.
Mitral inflow is recorded from the apical four-chamber view using pulsed-wave Doppler. Once the view is properly aligned, the sample volume is positioned at the tips of the mitral leaflets. Sample volume size should be small, about 2 mm. Care should be taken to avoid placing the sample volume too close to the mitral annulus which will result in lower velocities and an inaccurate E/A ratio. By moving the sample volume up and down relative to the mitral tips, the true peak velocity in early and late diastole can be recorded with confidence (Fig. 6.12). In addition, continuous-wave Doppler
can also be performed to confirm that maximal velocities are in fact recorded. Spectral gain and wall filter settings should be adjusted to ensure that a clean envelope is recorded and to facilitate the accurate timing of the beginning and end of mitral inflow. The Doppler recording should be performed at both a slow and a fast sweep speed. The slow speed is useful for evaluation of respiratory variation while the fast speed is used to obtain measurements. These measurements should be recorded at end-expiration and multiple beats should be averaged. Ideally, mitral inflow also should be recorded during the Valsalva maneuver. Doppler data are collected for approximately 10 seconds during the strain phase (see below).

Once the Doppler recording is optimized, a variety of measurements should be obtained. The primary measurements include the peak early filling velocity (E wave), peak filling velocity during atrial systole (A wave), the E/A ratio, and the deceleration time of the early filling velocity (Fig. 6.13). Deceleration time is defined as the time interval from early peak inflow velocity (the E wave) and the cessation of the early rapid filling phase (Fig. 6.14). It is inversely proportional to chamber stiffness and is obtained by tracing the deceleration curve from the maximal E wave velocity to the baseline which represents the time of pressure equalization between the two chambers (when inflow ends and velocity is zero). In many patients, the deceleration limb of the E wave does not reach the zero line. In these cases, the line should be extrapolated to the baseline in order to define the deceleration time (Fig. 6.15). However, with significant E-A wave fusion, and especially if atrial systole begins while the E velocity is still high, deceleration time should not be measured because of the likelihood of inaccuracy. In some cases, it is technically impossible to accurately measure the deceleration time. Figures 6.16 and 6.17 show examples of mitral inflow patterns in which deceleration time could not be determined. Factors that affect the mitral inflow pattern include sinus tachycardia and first degree AV block, which tend to fuse the E and A waves, atrial fibrillation, which eliminates the A wave, and mitral valve disease, which independently alters the velocity pattern.
FIGURE 6.12. The effect of sample volume location on the mitral inflow velocity pattern is demonstrated. The schematic at the lower left shows four sample volume locations. Each location results in a different pattern of mitral inflow. The proper location, used to record peak velocity in early and late diastole, usually requires placement of the sample volume at the tips of the mitral leaflets.

FIGURE 6.13. Once an optimal mitral inflow velocity recording has been obtained, a variety of measurements can be made. These are illustrated in the figure. See text for details.

**Color M-Mode Flow Propagation Velocity (Vp)**

When the mitral valve opens, flow accelerates from the valve orifice toward the apex of the left ventricle. Propagation velocity (Vp) throughout diastole can be measured with color Doppler M-mode. Although a variety of parameters can be obtained, by convention, the slope of the early diastolic valve-to-apex contour is used most often. From the four-chamber view, the M-mode cursor is placed in the center of the column of mitral inflow, as parallel as possible to flow direction (Fig. 6.18). Temporally, this is performed in early diastole, coincident with the E wave. By shifting the color baseline to a low Nyquist limit, an aliasing border (blue to red, representing the first aliasing velocity) near the center of the column is obtained. Although this border is not truly linear, a tangent is drawn from the mitral valve to a
point 4-cm distal, representing the early diastolic flow propagation velocity.

The slope of this line corresponds to the velocity gradient from left ventricular base to apex. The primary determinant is the rate of myocardial relaxation or elastic recoil of the chamber in early diastole. Thus, impaired relaxation will slow the propagation of blood and thereby reduce the slope of the line. However, several other factors affect this simple measurement. These include ventricular geometry, chamber volume, regional dyssynchrony, systolic function, and the complexity of flow vortex patterns once blood enters the chamber. It is recommended that propagation velocity should never be used in isolation and should only be assessed in the setting of a dilated left ventricle with reduced systolic function.

**FIGURE 6.14.** The method used to measure deceleration (decel) time is shown. It is defined as the time required for the peak E wave velocity to the cessation of early rapid filling as indicated by the arrows. See text for details.

**Tissue Doppler Mitral Annular Velocity**

The velocity of the mitral annulus can be recorded throughout the cardiac cycle using the tissue Doppler method (Fig. 6.19). From the four-chamber
view, the sample volume is positioned on the annulus, near the insertion site of the mitral valve. Both the septal (medial) and lateral sites should be recorded. Because of the high amplitude of the signal, spectral gain should be lowered to ensure a crisp, reproducible tracing. Because of the low velocity, the velocity scale should also be adjusted to maximize the size of the curve, thereby permitting accurate determination of velocity throughout the cardiac cycle. The sweep speed should be high, between 50 and 100 cm/s. Measurement of three or more consecutive cycles should be obtained. Using this approach, accurate, reproducible recordings are possible in the majority of patients.

Although several velocity measurements can be made, the most useful is the peak annular velocity in early diastole, called e'. The e' velocity primarily depends on left ventricular relaxation. When diastolic function is abnormal, e' is relatively independent of preload. However, when diastolic function is normal, e' increases with higher filling pressure. For this reason, the use of the e' has limitations in normal subjects. In patients with diastolic dysfunction, however, e' can be used to mitigate the effect of left ventricular relaxation on the E wave velocity. The practical importance of this observation will be discussed subsequently.

In assessing diastolic function, e' is reported separately and in combination with the E wave velocity into the ratio, E/e' (Fig. 6.20). A measure of e' should be made from both septal and lateral locations. In most patients, lateral e' will be higher than the septal value. Thus, the E/e' will be lower if the lateral position is used for e', and higher if the septal value is used (Fig. 6.21). Debate continues over which e' and E/e' should be reported. The range of normal and abnormal E/e' ratios published in the literature was initially generated using the septal value. Currently, it is recommended to measure both septal and lateral e'. Cutoff values for both are included in the most recent guidelines (Nagueh et al., 2016). For E/e', an average is recommended. That is, the mean of the septal E/e' and the lateral E/e' should be reported. In some cases, however, it may be appropriate to record only one of the e' values. This may be due to poor image quality, left bundle branch block, annular calcification or an adjacent wall motion abnormality. In such cases, cutoff values for both sepal and later E/e' are available.
FIGURE 6.15. The schematic demonstrates three types of mitral inflow velocity curves and shows how deceleration time should be determined in each case. Note in the middle panel that the velocity curve does not reach the baseline and the deceleration line must be extrapolated in order to determine the deceleration time.

FIGURE 6.16. An example of very abnormal mitral inflow are shown. The E wave is followed immediately by a wave of mid-diastolic filling (sometimes called an L wave, see arrowhead). Little or no filling occurs in late diastole (larger arrow).
The main use of the E/e’ ratio is to predict filling pressure in the setting of abnormal diastolic function (Figs. 6.22 to 6.24). A considerable amount of data has emerged validating this approach for estimating pulmonary capillary wedge pressure. A limitation of this approach is that the two measurements, E and e’, are obtained from different cardiac cycles and at different times. To minimize variability, the recording of the mitral inflow and annular velocities should be performed in close temporal proximity. Additional limitations exist. Age, preload, and systolic function can affect these parameters. The ratio may not be predictive in normal subjects, presumably because of the sensitivity of e’ to preload in the normal heart. Finally, prosthetic mitral valves, annular rings, and significant annular calcifications can create technical problems in measuring e’.

FIGURE 6.17. Two examples of E-A fusion are shown. In both, a combination of heart rate, very delayed left ventricular relaxation, and high left atrial pressure contribute to this pattern.
FIGURE 6.18. A color Doppler M-mode image recorded from the apical four-chamber view. In A, normal flow propagation velocity ($V_p = 77 \text{ cm/s}$) is demonstrated as evidenced by the steep slope of the early diastolic valve-to-apex contour. In B, the reduced slope and lower velocity ($V_p = 35 \text{ cm/s}$) is consistent with decreased chamber compliance.
Pulmonary Venous Flow Patterns

Pulmonary venous flow velocity can be recorded at the junction of the veins and left atrium, providing insight into the factors that affect left atrial filling. To obtain pulmonary venous flow, the apical four chamber should be used. Some superior angulation of the view is often required and color Doppler is helpful to identify the entrance of the veins into the chamber. Then, a pulsed Doppler sample volume should be positioned within the vein approximately 5 mm from its junction with the atrium (Fig. 6.25). To optimize the recording, wall filter settings should be lowered and a fast sweep speed should be employed. Measurements should be obtained over three consecutive cycles.
Of all the Doppler parameters described above, this is the most difficult to obtain, but is still feasible in most patients.

FIGURE 6.20. This schematic demonstrates the relationship between mitral annular velocity (top) and mitral inflow velocity (bottom). As filling pressure increases, the annular e’ velocity decreases while the mitral inflow E velocity increases, as shown in the right panel. This results in an increase in the E/e’ ratio. LVDP, left ventricular diastolic pressure.
Pulmonary venous flow consists of three main components: an antegrade systolic wave (which often has two peaks, S1 and S2), a diastolic wave (D), and a retrograde wave (Ar) corresponding to atrial systole (Fig. 6.26). Both the time velocity integral (TVI) and the peak velocity of each component can be measured. In addition, the duration and peak velocity of the retrograde atrial wave can be quantified. The systolic fraction is defined as the ratio of systolic to the diastolic TVI (i.e., the ratio of areas under the velocity curves). The most commonly reported value is the ratio of the peak antegrade velocities in systole and diastole, the S/D ratio. If two separate systolic velocities (S1 and S2) are present, as in the presence of bradycardia and first degree block, it is recommended that the second (or S2) value be used. The pulmonary venous flow pattern is affected by several factors. Young normal subjects have a predominant diastolic wave. With increasing age, the S/D ratio increases (Figs. 6.27 and 6.28). As left atrial compliance decreases and pressure rises, the S/D ratio decreases and the systolic fraction is usually less than 40%.

\[
E/e' = \frac{84}{10} = 8.4
\]
FIGURE 6.23. This example, taken from a patient with elevated left ventricular filling pressure, demonstrates an abnormally high E/e’ ratio of approximately 18. In (A), mitral inflow demonstrates an E wave velocity of 110 cm/s. Annular e’ is recorded in B and C. Note that a different E/e’ ratio is obtained depending on whether the septal (B) or lateral (C) e’ value is utilized.
The duration of the retrograde atrial wave, Ar, also increases with increased filling pressure. Furthermore, differences in duration of Ar and the mitral A wave (Ar – A) have been shown to correlate with left ventricular end-diastolic pressure. As left atrial pressure rises, Ar duration lengthens and Ar – A difference increases. Although technically challenging to measure, the Ar – A may be the most sensitive and earliest indicator of elevated left atrial pressure. A value of >30 ms indicates elevated left ventricular end-diastolic pressure and will be present before mean left atrial pressure becomes abnormal. This may be useful in patients with abnormal relaxation to separate those with normal from those with elevated filling pressures.
FIGURE 6.25. An example of normal pulmonary venous flow is illustrated. Normal flow consists of a systolic wave (S), a diastolic wave (D), and a small wave of flow reversal (Ar) that occurs during atrial systole.
FIGURE 6.26. In A, the schematic demonstrates the relationship between the three pulmonary venous flow components, recorded using pulsed Doppler, and the electrocardiogram (ECG). The drawing shows how the duration of retrograde flow during atrial systole (Ar) is measured. An example is provided in B, showing a very prominent atrial reversal wave (Ar). See text for details.
FIGURE 6.27. Abnormal pulmonary venous flow patterns are demonstrated. In A, diastolic predominance (D > S) is present. In B, the systolic wave is absent and forward flow occurs exclusively during diastole.

There are significant limitations to the routine use of pulmonary venous patterns in hemodynamic studies. In addition to the technical challenges in obtaining the recordings, age, heart rate, PR interval, mitral regurgitation, and systolic function also affect pulmonary venous flow. It has been shown that these parameters have limited accuracy in the setting of normal systolic function. For all these reasons, these parameters have been subjugated to a minor role in the practical assessment of diastolic function.

**Left Atrial Volume**

Although not a hemodynamic parameter, left atrial volume determination is an essential part of the diastolic function assessment. An increase in left atrial size is the morphologic expression of chronic diastolic dysfunction. Although admittedly nonspecific, it reflects both the duration and severity of disease. Chamber volume should be obtained using the biplane approach, from the apical four- and two-chamber views. The left atrial area should be measured at end-systole, just prior to mitral valve opening, when volume is greatest.

Two approaches to volume calculation have been reported ([Fig. 6.29](#)). The area-length method requires planimetry of the chamber and measurement of the distance from the annular plane to the superior border of the chamber. The length and area are obtained in both orthogonal views and then combined to derive volume. The second approach uses the Simpson method for volume determination and only requires planimetry of the chamber from the two views (i.e., linear dimensions are not involved). The echocardiographic planes should be adjusted to ensure that maximal area of the left atrium is captured. When performing the planimetry, care must be taken to exclude the pulmonary veins. Also, by convention, the mitral annulus is used as the inferior border when tracing left atrial area. Because of the relationship between atrial size and body size, it is recommended that volume be corrected for body surface area, and reported in mL/m².
The superiority of volume over simple linear dimensions for assessing left atrial size is now well established. With careful attention to technique, accurate determination of volume is feasible in most patients. However, the limitations of deriving volume from tomographic images should be apparent. For this reason, three-dimensional imaging will likely play an increasing role for this purpose in the future. Left atrial volume has both diagnostic and
prognostic value in the assessment of diastolic function. However, left atrial enlargement may also result from other factors, thereby reducing its specificity. In particular, mitral valve disease will often lead to left atrial dilation. This possibility should be considered whenever left atrial volume is increased in the setting of normal Doppler markers of diastolic function.

**The Valsalva Maneuver**

Preload manipulation is an integral part of the comprehensive diastolic function examination. This is most often accomplished using the Valsalva maneuver. This involves forced expiration against a closed nose and mouth. During the strain phrase, left ventricular preload is reduced. The most important and practical application of this maneuver is in conjunction with mitral inflow velocity assessment. For example, in the setting of a normal appearing mitral inflow pattern, preload reduction can unmask a pseudonormal state (Fig. 6.30). In normal subjects, Valsalva maneuver leads to a general reduction in velocity, affecting the E and A wave to a similar degree. Thus, the E/A ratio is unchanged. In the pseudonormal stage of diastolic dysfunction, the Valsalva maneuver will change the pattern to one resembling impaired relaxation. This is because pseudonormalization causes a moderate increase in filling pressure superimposed on delayed relaxation. By lowering preload, the delayed relaxation pattern is unmasked. Thus, during the Valsalva strain phrase, a decrease in the E/A ratio of >50% is a useful indicator of elevated filling pressure. However, in the setting of irreversibly elevated filling pressure (the restrictive filling pattern), this decrease in E/A may not occur.
Other Markers of Diastolic Dysfunction

Strain and strain rate can be measured using Doppler or speckle tracking methods. Although strain can be recorded during diastole and may provide unique information on diastolic function, its value for this purpose has yet to be established. Because *regional* strain (and strain rate) are typically assessed, it may be possible to use this approach to assess diastolic function locally. This may have relevance in the setting of acute ischemia, dyssynchrony, or viability assessment. Currently, however, there is no evidence to support the routine use of this technique for diastolic function...
Twisting and untwisting (or torsion) have been recognized as important factors in ventricular function. This type of motion occurs due to the presence of obliquely oriented subepicardial fibers and contributes importantly to the efficiency of contractility and relaxation. Speckle tracking now provides a unique noninvasive method to evaluate this component of myocardial mechanics. Diastolic untwisting is the result of elastic recoil as forces seek to restore ventricular shape to the resting, unstressed state. Both the rate and extent of untwisting can be quantified. This aspect of diastolic function may be an important factor in early diastolic suction generation. Evidence now suggests that relaxation abnormalities alter diastolic untwisting. Although experience with this technique is limited, it seems likely that this approach will become an increasingly important part of diastolic function assessment in the future.

**FIGURE 6.30.** Preload reduction using the Valsalva maneuver can be used to unmask the pseudonormal state. On the left, a normal mitral inflow velocity pattern is shown. On the right, following Valsalva, the E/A ratio decreases significantly, demonstrating impaired relaxation. This is consistent with the pseudonormal stage of diastolic dysfunction.

**A COMPREHENSIVE APPROACH TO DIASTOLIC DYSFUNCTION**

The assessment of diastolic function is a complex, inexact science in which
multiple factors must be assessed and integrated with clinical information. One of the most challenging aspects of diastology is the recognition that even in otherwise normal subjects, age-related changes in diastolic function are common. As the ventricle ages, increased myocardial stiffness and delayed relaxation are considered normal consequences of aging. For each parameter, a range of values exists with significant overlap between normal subjects and patients (Table 6.3). Furthermore, multiple factors, including blood pressure, heart rate, systolic function, pulmonary hypertension, right heart function, and pericardial constraint, also affect these same parameters. This means that no one parameter can be relied upon as “specific” for determining diastolic function and therefore should never be used in isolation. Instead, a number of markers must be evaluated, including the clinical scenario. An example of this is the finding of a high E/A ratio. This may indicate restrictive filling and elevated left atrial pressure. It may also be seen in a healthy young athlete. Distinguishing between the two can and should be made on clinical grounds and the inclusion of other Doppler variables, such as e’.

Thus, the diagnosis of diastolic dysfunction is most helpful when viewed in clinical context and in the setting of a plausible anatomic substrate. In some respects, the sheer number of potential measurements has created confusion and even frustration for clinicians. One of the problems is the lack of a gold standard and the challenge of validating each individual noninvasive marker against an appropriate benchmark. In one study (Kasner et al., 2007), a group of patients with heart failure and normal ejection fraction and a group of control patients were studied with sophisticated invasive techniques, including pressure–volume loop recordings and derivation of Tau (τ, the time constant of relaxation), to define the presence and severity of diastolic dysfunction. Then these findings were compared to Doppler and tissue Doppler parameters. Most Doppler markers, including E/A ratio, IVRT, and deceleration time, correlated modestly with the various invasive measures. The parameter that correlated best was E/e’ (using the lateral annulus). Using a cut point of 8, E/e’ (lateral) had a specificity of 92% and a sensitivity of 83% for the detection of diastolic dysfunction. This study underscores the complexity of diastolic function and reminds us that no single parameter, neither invasive nor Doppler, can completely characterize diastolic function. Instead, a comprehensive and systematic approach is recommended to fully address this important clinical problem.
### Table 6.3
#### DEFINING THE STAGES OF DIASTOLIC DYSFUNCTION: NORMAL AND ABNORMAL VALUES IN ADULTS

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Units</th>
<th>Normal</th>
<th>Stage I</th>
<th>Stage II</th>
<th>Stage III</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Impaired</td>
<td>Pseudonormal</td>
<td>Restrictive Filling</td>
</tr>
<tr>
<td>IVRT</td>
<td>ms</td>
<td>&lt;70</td>
<td>&gt;90</td>
<td>60–90</td>
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<td>E/A ratio</td>
<td>Unitless</td>
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<td>0.8–1.5</td>
<td>≥2</td>
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<tr>
<td>Δ with Valsalva</td>
<td>%</td>
<td>Both E &amp; A decrease, ratio unchanged</td>
<td>Both E &amp; A decrease, ratio unchanged</td>
<td>E decreases, A increases, ratio reverses</td>
<td>Variable</td>
</tr>
<tr>
<td>Deceleration time</td>
<td>ms</td>
<td>140–240</td>
<td>&gt;240</td>
<td>140—200</td>
<td>&lt;140</td>
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<tr>
<td>e’ (septum)</td>
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<td>&gt;10</td>
<td>&lt;7</td>
<td>&lt;7</td>
<td>&lt;5</td>
</tr>
<tr>
<td>e’ (lateral)</td>
<td>cm/s</td>
<td>&gt;12</td>
<td>&lt;10</td>
<td>&lt;10</td>
<td>&lt;8</td>
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<tr>
<td>E/e’ ratio (averaged)</td>
<td>cm/s</td>
<td>&lt;8</td>
<td>&lt;8</td>
<td>Variable</td>
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<td>Pulmonary venous flow</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>S/D ratio</td>
<td>Unitless</td>
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<td>S &gt; D</td>
<td>S ≤ or = D</td>
<td>S &lt;&lt; D</td>
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<td>Ar – A</td>
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<td>Varies</td>
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<td>&lt;50</td>
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<td>&gt;28</td>
<td>&gt;34</td>
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</table>

See text for abbreviations.

In another clinical study (Mullens et al., 2009), acutely ill patients in the intensive care unit with severe systolic dysfunction were examined. The Doppler-derived E/e’ was compared to invasively determined pulmonary capillary wedge pressure. Doppler and invasive data were gathered simultaneously. The correlation between the two methods was poor and the E/e’ did not reliably differentiate between those patients with a pulmonary capillary wedge pressure of <18 mm Hg from those >18 mm Hg. The poor predictive value of Doppler in this study may be partly explained by the
severity of heart failure and the acutely unstable condition of the patients. All of these patients had experienced marked clinical deterioration just prior to the study and were ill enough to warrant ICU admission and right heart catheterization.

For all these reasons, an integrated approach to diastolic function assessment is warranted. As a first step, some basic issues should be considered prior to undertaking an assessment of diastolic function (Table 6.4). Among these, left ventricular systolic function is a useful starting point. Some of the Doppler variables of diastolic function perform very differently in patients with reduced versus normal ejection fraction. For that reason, it makes sense to begin with an assessment of left ventricular ejection fraction. If ejection fraction is abnormal (usually defined as <50%), diastolic function is invariably abnormal, and goal of the evaluation should be to determine the severity of dysfunction and whether or not left ventricular filling pressure is elevated. The steps needed to make these determinations are covered below.

<table>
<thead>
<tr>
<th>Table 6.4</th>
<th>THE INITIAL APPROACH TO DIASTOLIC FUNCTION ASSESSMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor</td>
<td>Implications for Diastolic Function</td>
</tr>
<tr>
<td>Patient age</td>
<td>Diastolic function changes with age</td>
</tr>
<tr>
<td></td>
<td>Most Doppler parameters are age dependent</td>
</tr>
<tr>
<td></td>
<td>Overlap between normal and abnormal findings becomes increasingly prevalent in elderly patients</td>
</tr>
<tr>
<td>Presence/absence of Sx</td>
<td>Because of the overlap between normal and abnormal results, caution is urged in diagnosing DD in asymptomatic individuals</td>
</tr>
<tr>
<td></td>
<td>Diastolic function assessment is most relevant in the presence of Sx consistent with DD, e.g., dyspnea</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Both tachycardia and bradycardia will affect diastolic function assessment</td>
</tr>
<tr>
<td></td>
<td>Arrhythmias such as atrial fibrillation and complete heart block make the determination of DD challenging</td>
</tr>
<tr>
<td>LVH</td>
<td>The presence of LVH is strongly correlated with the likelihood of DD</td>
</tr>
<tr>
<td></td>
<td>In athletes, diastolic function assessment can be useful to distinguish physiologic vs. pathologic hypertrophy</td>
</tr>
<tr>
<td>Left atrial volume</td>
<td>Significant DD is unlikely in the absence of a dilated left atrium</td>
</tr>
<tr>
<td>LV ejection fraction</td>
<td>If LVEF is reduced, some degree of DD is invariably present</td>
</tr>
<tr>
<td></td>
<td>In the setting of a normal LVEF, an integrated approach to diastolic</td>
</tr>
</tbody>
</table>
If ejection fraction is preserved, but the patient has signs or symptoms that could be secondary to diastolic dysfunction, defining its presence and severity requires a careful assessment of both two-dimensional echocardiographic and Doppler parameters. Because of the overlap that exists in this population between normal and abnormal diastolic measures, caution should be exercised in diagnosing diastolic dysfunction in the asymptomatic individual with normal systolic function.

GUIDELINES FOR THE ASSESSMENT OF DIASTOLIC DYSFUNCTION

Over the past decade, guidelines for the echocardiographic assessment of diastolic function assessment have been formulated and updated. The most recent iteration, published jointly by the American Society of Echocardiography and the European Association of Cardiovascular Imaging (Nagueh et al., 2016), was released in 2016. Recognizing the complexity of diastolic function, the number of different parameters that can be measured, and the range of normal values for each, the guidelines emphasize a step-wise, integrated approach that addresses several issues. The goals of the algorithms are to distinguish normal from abnormal diastolic function, determine the severity or grade of dysfunction, detect evidence of elevated left ventricular filling pressure, and predict risk. Different approaches for patients with normal versus abnormal systolic function are also provided.

The guidelines provide a useful framework to systematically evaluate diastolic function and filling pressure in patients with suspected dysfunction. Validating their accuracy is difficult. As discussed below, precise, accurate classification of all patients will never be achievable. Applying guidelines to individual patients should always be done with caution and clinical judgment is critical for an accurate diagnosis.
When systolic function is abnormal, diastolic function is also abnormal. This implies that, in patients with reduced ejection fraction, the possibility of mistaking normal from pseudonormal (Grade II) diastolic dysfunction is avoided and, in such cases, a careful recording of mitral inflow will suffice to permit the severity of diastolic dysfunction to be determined (Fig. 6.31). If the E/A ratio is ≤0.8, filling pressure is likely normal and this would correspond to Grade I diastolic dysfunction (impaired relaxation). In most cases, the peak E wave velocity will be <50 cm/s. Since E wave velocity reflects the early diastolic pressure gradient between the left atrium and the left ventricle, a higher E velocity could suggest higher left atrial pressure. If the E/A ratio is ≥2.0, filling pressure is probably elevated and the patient will have Grade III or IV diastolic dysfunction. In most patients, the mitral E deceleration time will be <140 ms.

Unfortunately, many patients with reduced ejection fraction will have an E/A ratio between 0.8 and 2.0. For this group, other parameters should be measured. These include the average E/e’, left atrial volume index, and tricuspid regurgitation jet velocity. In particular, an average E/e’ >14, or a lateral E/e’ >12 suggests increased filling pressure. Elevated pulmonary artery pressure (an increased tricuspid regurgitant jet velocity) and a dilated left atrium also increase the likelihood of elevated filling pressure. A detailed summary of this approach is provided in Figure 6.32.
When systolic function is preserved, the evaluation of diastolic function is more complex and a different approach is necessary. Overlap in values of Doppler variables, the confounding influence of age, and the difficulty in distinguishing normal from pseudonormal create considerable challenges in this population. The optimal approach to these patients begins with the two-dimensional echocardiogram to determine if an anatomic substrate exists that would make diastolic dysfunction likely. These include left ventricular hypertrophy and increased left atrial volume. In addition, global longitudinal strain has shown promise for this assessment. Although absence of hypertrophy and left atrial enlargement does not exclude the possibility of
diastolic dysfunction, this does reduce its likelihood and raises the possibility of an alternative diagnosis.

The next step in patients with normal ejection fraction is to examine and integrate multiple echocardiographic measures. The guidelines (described above) emphasize four dichotomous variables (Table 6.5). These four variables and the cutoff value used to define abnormal are: (1) e' (<7 for septal e', <10 cm/s for lateral); (2) average E/e' >14; (3) left atrial volume index >34 mL/m$^2$; and (4) tricuspid regurgitation jet velocity >2.8 m/s. To establish a diagnosis of diastolic dysfunction requires three or all four of these abnormal values to be present. Once the diagnosis of diastolic dysfunction is made, grading of severity is as described previously, focusing on the mitral E/A ratio and the e' velocity (see Figs. 6.32, 6.33, and Table 6.3). Elevated filling pressure is suggested by an average E/e' >14, or a lateral E/e' >12. Other indirect markers of increased filling pressure include a high tricuspid regurgitation jet velocity, or a dilated left atrium. A low mitral E velocity (<50 cm/s), however, argues against a high filling pressure.

**FIGURE 6.32.** A flow diagram for grading diastolic dysfunction and estimating left ventricular filling pressure in patients with reduced ejection fraction and myocardial diseases (but normal ejection fraction) is presented. See text for details. DD, diastolic dysfunction; inc, increased; LAP, left atrial pressure; LAVI, left atrial volume index; neg, negative; pos, positive; TR, tricuspid regurgitation jet. (Modified with permission from Nagueh S, Smiseth OA, Appleton CP, et al.)
Table 6.5

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Cutoff Value</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Annular e’ velocity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Septal</td>
<td>&lt;7 cm/s</td>
<td>Correlates with the time constant of relaxation (τ)</td>
</tr>
<tr>
<td>Lateral</td>
<td>&lt;10 cm/s</td>
<td>Reflects rate of LV relaxation prior to MV opening</td>
</tr>
<tr>
<td>2 E/e’</td>
<td>&gt;14</td>
<td>Correlates with LV filling pressures</td>
</tr>
<tr>
<td>Average</td>
<td>&gt;13</td>
<td>Relatively load independent</td>
</tr>
<tr>
<td>Lateral</td>
<td>&gt;13</td>
<td></td>
</tr>
<tr>
<td>3 LA volume index</td>
<td>&gt;34 mL/m²</td>
<td>Reflects duration and severity of diastolic dysfunction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prognostically important</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sensitive, but not specific</td>
</tr>
<tr>
<td>4 TR peak velocity</td>
<td>&gt;2.8 m/s</td>
<td>Correlates with LA pressure in the absence of precapillary causes of pulmonary hypertension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Relatively nonspecific finding</td>
</tr>
</tbody>
</table>

LA, left atrial; TR, tricuspid regurgitation.


While attractive in its simplicity, the sensitivity and specificity of this guidelines-based approach are yet to be determined. In one multicenter study (Anderson et al., 2017), patients with a variety of cardiac diseases, including patients with normal and reduced ejection fraction, were evaluated using the algorithm from the ASE guidelines to predict left ventricular filling pressure. The echocardiographic findings were then compared to invasive data obtained during cardiac catheterization. The overall accuracy of
Echocardiography to detect elevated filling pressure was 87%. Accuracy was higher (91%) for patients with reduced ejection fraction compared to those with normal ejection fraction (84%).

**APPLYING THE ALGORITHMS TO PATIENTS**

**Impaired Relaxation (Grade I)**

As discussed previously, the earliest and usually least severe form of diastolic dysfunction is impaired relaxation, the result of delayed left ventricular pressure decline following aortic valve closure. This is associated with a reversal of the E/A ratio (usually <1) and a prolonged deceleration time (>240 ms). In most cases, and especially when the E wave velocity is low (<50 cm/s), filling pressures will not be elevated, although it should be noted that this mitral inflow pattern does not preclude the possibility of a modest increase in preload. Impaired relaxation is usually associated with a prolonged IVRT, although the multiple factors that affect IVRT limit the specificity of this finding. At this stage, E/e’ is often normal (indicating normal filling pressure) and left atrial volume is mildly increased. An example of impaired relaxation is presented in Figure 6.34. This case involved a 59-year-old woman with untreated chronic renal disease. Left ventricular systolic function was normal and the left atrium was moderately dilated. The case illustrates the E/A reversal and IVRT prolongation typical of impaired relaxation.
FIGURE 6.33. When ejection fraction is preserved, diastolic function may be normal, so four possible conditions exist. The schematic demonstrates typical mitral inflow velocity, pulmonary venous flow, and mitral annular velocity patterns in the setting of normal diastolic function, impaired relaxation, pseudonormal filling, and restrictive physiology. A high E/A can be either good (normal) or bad (restrict). E/E’ helps sort it out. See text for details.
Another example of impaired relaxation is shown in Figure 6.35, from a 47-year-old man with untreated hypertension. Two-dimensional imaging demonstrated left ventricular hypertrophy, but preserved ejection fraction. The mitral inflow pattern showed E/A wave reversal, the left atrium was moderately dilated, and e’ was reduced. However, the pulmonary venous flow pattern was normal. Overall, this combination of findings points to impaired relaxation with normal filling pressure, but, as is sometimes the case, some inconsistent findings are present.

**Pseudonormal (Grade II)**

With progression of disease, filling pressure rises, leading to the pseudonormal phase. Here, the E/A ratio and deceleration time are within the normal range (hence the name). Table 6.6 lists some of the markers that can be used to differentiate between normal and pseudonormal. Among the most helpful are the e’ velocity and the Valsalva maneuver. With pseudonormal filling, annular velocity is usually reduced (lateral e’ <8 or 10 cm/s, depending on age) and the E/e’ is often >14, suggesting elevated filling pressure. The Valsalva maneuver also can unmask the underlying relaxation abnormality. A decrease in E/A >50% during the strain phase is indicative of increased filling pressure and serves to distinguish normal from pseudonormal function. At this stage, the IVRT may fall within the normal range, due to the combined and offsetting effects of increased left atrial pressure and delayed relaxation. Furthermore, the E/e’ will be increased for the same reason. In almost patients at this stage of chronic diastolic dysfunction, left atrial volume will be significantly increased. Additional clues to the pseudonormal state include a reduced propagation velocity slope and a pulmonary venous systolic-to-diastolic ratio <1. Figure 6.36 is an example of pseudonormal diastolic dysfunction in a patient with end-stage renal disease and severe hypertension. Although the E/A ratio is normal at baseline, impaired relaxation is unmasked with the Valsalva maneuver. In
addition, the left atrium is significantly enlarged. A low septal e' (6 cm/s) and a high E/e' (18) indicate elevated filling pressure. This is further suggested by the prolonged pulmonary venous A wave (Ar) relative to the mitral inflow A wave.

FIGURE 6.35. An example of a patient demonstrating impaired relaxation. Overall left ventricular systolic function is preserved (A). Mitral inflow (B) reveals an E wave velocity < A velocity. However, the E is >50 cm/s and the mitral annular e' velocity is <7 cm/s (C). Pulmonary venous flow (D) shows systolic predominance. These findings suggest a more advanced form of impaired relaxation and possibly increased left ventricular filling pressure. See text for details. D, diastolic filling wave; S, systolic filling wave.
Video 6-35a

coming soon
LAVI = 40 ml/m²

IVRT = 90 ms

E/A = 1.5

E/A = 0.8

E/e' = 18

e' = 6 cm/s
FIGURE 6.36. A case study from a patient with pseudonormal diastolic dysfunction is presented. The patient had end-stage renal disease and severe hypertension. The left atrium is severely dilated and Doppler indices are consistent with the pseudonormal stage of diastolic dysfunction. See text for details.

<table>
<thead>
<tr>
<th>Table 6.6</th>
<th>DISTINGUISHING NORMAL FROM PSEUDONORMAL USING ECHO DOPPLER MARKERS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parameter</td>
<td>Normal</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>0.8–1.5</td>
</tr>
<tr>
<td>∆ with Valsalva</td>
<td>Both decrease</td>
</tr>
<tr>
<td></td>
<td>No change in ratio</td>
</tr>
<tr>
<td>e’ septum (cm/s)</td>
<td>&gt;10</td>
</tr>
<tr>
<td>e’ lateral (cm/s)</td>
<td>&gt;12</td>
</tr>
<tr>
<td>E/e’ (average)</td>
<td>&lt;8</td>
</tr>
<tr>
<td>LA volume index (mL/m&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>&lt;28</td>
</tr>
<tr>
<td>Pulmonary vein S/D</td>
<td>S ≥ D&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

D, diastolic; S, systolic.

<sup>a</sup>Variable depending on LV filling pressure.

<sup>b</sup>S may be less than D in young healthy people.

Another example of pseudonormal diastolic dysfunction is illustrated in Figure 6.37. Although the Valsalva maneuver failed to show a significant decrease in the E/A ratio, several other indicators of diastolic dysfunction, including left atrial dilation, increased tricuspid regurgitation velocity, a very low e’ velocity, and an abnormal pulmonary vein flow pattern, were present, making normal diastolic function very unlikely.

**Restrictive Filling (Grade III)**

With the development of restrictive filling, the E/A ratio increases (usually >2, an indication of a high LA-LV pressure gradient at the time of mitral opening) and the deceleration time becomes very short (<160 ms, due to a noncompliant left ventricle). This results from the loss of elastic recoil and
Increased reliance on pushing, rather than suction of blood into the left ventricle. The left atrium is invariably enlarged and an E/e' ratio greater than 14 confirms elevated filling pressure. If this stage of dysfunction is reversible, a decrease in the E/A ratio may occur with the Valsalva maneuver, or as a result of medical therapy to reduce preload, for example, diuresis. An additional clue to this stage is a small or absent pulmonary venous systolic wave, that is, a predominant diastolic wave.

As this phase of restrictive filling progresses to irreversibility, the E/A ratio becomes fixed and unresponsive to Valsalva (as well as other preload reducing strategies, including diuresis). An example of restrictive physiology is presented in Figure 6.38. These images are from a patient with ischemic cardiomyopathy and pulmonary edema. The left atrium is severely enlarged and the mitral inflow pattern is consistent with restrictive filling. The lack of responsiveness of the mitral pattern to Valsalva indicates irreversible restrictive filling. The E/e' ratio of 22 indicates elevated filling pressure. The pulmonary venous inflow pattern is also strikingly abnormal. Figure 6.39 demonstrates restrictive filling in a patient with ischemic cardiomyopathy.

SERIAL CHANGES IN DIASTOLIC FUNCTION

Changes over time in diastolic function are common and result from a variety of factors. Improvement can occur and can be demonstrated using echocardiography. This is usually the result of successful treatment of the underlying cause of the diastolic dysfunction. Figure 6.40 is from a patient with hypertension and coronary artery disease who presented with worsening symptoms of exertional dyspnea. At the time of his initial study, his heart rate was 90/min and blood pressure was 150/95 mm Hg. An ECG demonstrated first degree AV block. The echocardiogram shows left atrial dilation, left ventricular hypertrophy, and preserved ejection fraction. Mitral inflow revealed high E and A wave velocities and fusion of E and A. Note that atrial systole occurs early due to the high heart rate and first degree block. As a result, atrial filling (the A wave) begins while the mitral inflow velocity is still >50 cm/s. This pattern, along with a low lateral e' (8 cm/s) and high E/e' (20), is consistent with markedly abnormal relaxation and elevated filling pressure, explaining his symptoms of exertional dyspnea.
After treatment with a beta blocker and ACE inhibitor, symptoms improved significantly and he underwent repeat echocardiography. His heart rate had decreased to 68/min and his blood pressure was 130/80 mm Hg. At the lower heart rate, his left ventricular filling had improved. His E wave velocity was able to decrease to <20 cm/s before atrial systole began and both e' and E/e' had improved. Patients with fusion of the E and A waves often complain of exertional dyspnea, usually due to a combination of impaired relaxation and high filling pressures. The exertional symptoms are due in part to the abbreviated diastolic filling time that becomes even shorter at high heart rate (see Fig. 6.41). Blood pressure treatment and heart rate lowering frequently lead to symptom improvement. An extreme example of E and A wave fusion, from a patient with severe systolic and diastolic dysfunction, is shown in Figure 6.42.

With progression of disease, diastolic function can worsen. In such cases, echocardiography can be helpful to confirm the change in diastolic function and, in some patients, to determine whether left ventricular filling pressure is elevated so that appropriate therapy can be undertaken. Figure 6.43 illustrates such a case. At the time of the initial echocardiogram, the patient was feeling well and normally active, despite severe left ventricular systolic dysfunction, the result of ischemic cardiomyopathy. Doppler reveals evidence of impaired relaxation, with no evidence of high filling pressure. Similar findings are recorded 1 year later at a time when the patient remained clinically stable and active. However, 9 months later, the patient presented to the Emergency Department with unstable angina and pulmonary edema. Although his systolic function was unchanged, Grade III diastolic dysfunction, with elevated filling pressure, is now present. Note the marked change in the mitral inflow pattern, without any measurable decrease in systolic function.

### STRESS TESTING TO ASSESS DIASTOLIC FUNCTION

The diastolic stress test has several applications. It is useful in patients who report exertional dyspnea in the setting of normal pulmonary function and preserved ejection fraction. It is also helpful to evaluate filling pressures in patients with known diastolic dysfunction with no or mild symptoms. Often
patients in the early stages of diastolic dysfunction only have symptoms or limitations with exertion. Finally, the addition of exercise measures of diastolic function may increase the sensitivity of echocardiography for the diagnosis of diastolic heart failure.

Among the various parameters that can be assessed during exercise, the E/e′ ratio is most practical. In normal subjects, with exercise, both E and e′ increase and the E/e′ ratio remains unchanged or decreases slightly. In patients with impaired relaxation, mitral E velocity increases during exercise, while e′ increases minimally if at all. Thus, the ratio will increase significantly, an indicator of a rise in left atrial pressure. Because the changes in mitral E velocity usually persist for several minutes after termination of exercise, they can be detected postexercise, even after wall motion assessment has been completed. A brief delay in recording mitral inflow also avoids the problem of fused E and A waves that occurs at high heart rates. Thus, combining an assessment of diastolic function with routine exercise echocardiography is feasible and may be of particular value in those patients with exertional dyspnea.
Although significant increase in E/e’ during exercise is a helpful finding, the correlation between changes in E/e’ and invasive measures of diastolic dysfunction, such as pulmonary capillary wedge pressure, is modest and no clear cut point can be relied upon to distinguish normal from abnormal responses. Diastolic stress testing is most helpful in symptomatic patients when a clinical suspicion of diastolic heart failure exists, but resting Doppler findings are inconclusive. In such cases, the added sensitivity of the diastolic
stress test may demonstrate a link between symptoms and diastolic dysfunction so that a firm diagnosis can be made and appropriate therapy can be undertaken.

Finally, whenever stress testing is done for the purpose of dyspnea assessment, it is also prudent to record the tricuspid regurgitation velocity, before and after exercise. Like the diastolic parameters, determination of pulmonary pressures during stress in the group of patients can be very useful to answer clinical questions. If either pulmonary systolic pressure or left ventricular filling pressure increases significantly during exercise, the etiology of the patient’s symptoms is usually established.
FIGURE 6.38. These images were recorded from a patient with ischemic
cardiomyopathy, moderate systolic dysfunction and a significantly enlarged left atrium. Doppler indices are remarkable for restrictive physiology that does not respond to preload reduction. These findings suggest elevated left ventricular filling pressure and an irreversible stage of restrictive filling. See text for details.

**FIGURE 6.39.** A restrictive filling pattern from a patient with ischemic cardiomyopathy and reduced ejection fraction is provided. In **A**, mitral inflow shows an abnormally high E/A ratio and a short E wave deceleration time (<160 ms). In **B**, the e’ velocity is 5 cm/s, yielding an E/e’ ratio of 24, indicating elevated filling pressure.
FIGURE 6.40. An example of improvement in diastolic function in a patient with impaired relaxation is shown. Images were obtained before (A to C) and after (D and E) treatment. The benefits of heart rate slowing and blood pressure control are demonstrated. See text for details.

Video 6-40a

THE DIFFERENTIAL DIAGNOSIS OF HEART FAILURE
Diastolic dysfunction is an important component of heart failure with preserved ejection fraction. Among patients with heart failure symptoms, the demonstration of diastolic dysfunction is often cited as evidence for a cause-and-effect relationship. However, several other conditions may also lead to symptoms of fatigue and exertional dyspnea and must therefore be considered in the differential diagnosis. Pericardial disease, particularly constrictive pericarditis, should be considered when heart failure and normal systolic function coexist. It is appropriate to consider constriction as having an element of diastolic dysfunction, since filling pressures are elevated and the mitral inflow velocity usually demonstrates a restrictive filling pattern. The distinction is important, however, because the treatment is markedly different. Findings that suggest constrictive pericarditis include a normal or high $e'$ velocity, which is very unusual in other causes of restrictive filling, and abnormal hepatic vein flow, which usually show marked, respiratory-dependent flow reversal (Figs. 6.44 and 6.45). This is not seen in most other causes of diastolic dysfunction. Table 6.7 lists several features that can be used to differentiate restrictive cardiomyopathy, a form of severe diastolic dysfunction, from constrictive pericarditis.
FIGURE 6.41. This schematic demonstrates the effect of delayed left ventricular relaxation on the diastolic filling period (DFP). In a normal individual (left panels), the increased heart rate due to exercise leads to a modest shortening of DFP (see double-headed arrows). In patients with impaired relaxation (right panels), the same degree to exercise-induced tachycardia results in a delay in the rate of left ventricular pressure fall (red arrows) and a shortening of DFP, so that inadequate filling of the left ventricle occurs.
Among patients with normal systolic function and heart failure symptoms, several other conditions should be considered. In most cases, these are diseases in which diastolic dysfunction is the primary cause—or a major contributor—to symptoms. They are important, however, because specific treatment, sometimes curative, is available. These include mitral valve disease (both stenosis and regurgitation), restrictive cardiomyopathy, anemia, hypertrophic cardiomyopathy, and transient ischemia.

### EVALUATION OF DIASTOLIC DYSFUNCTION IN SPECIFIC PATIENT GROUPS

#### Sinus Tachycardia

Most Doppler parameters perform less well in the setting of sinus tachycardia, especially in patients with normal systolic function. For example, fusion of the E and A waves of the mitral inflow pattern makes it difficult to measure the E/A ratio and deceleration time. In addition, fusion of
the E and A waves will tend to increase A wave velocity and reduce the E/A ratio. The parameter that is most useful in sinus tachycardia is the E/e', which retains its ability to predict filling pressures at higher heart rates. This is true whether or not ejection fraction is reduced.

**Atrial Fibrillation**

Atrial fibrillation creates two distinct problems, absence of the mitral A wave and beat-to-beat variability. In patients with atrial fibrillation and systolic dysfunction, the deceleration time correlates modestly with filling pressures. A deceleration time <160 ms not only predicts an elevated filling pressure but also a poor prognosis. In addition, the E/e’ ratio retains its value in patients with atrial fibrillation. A ratio >11 corresponds to a left ventricular end-diastolic pressure ≥15 mm Hg. To ensure accuracy, several beats must be measured, due to the heart rate variability.

**Mitral Valve Disease**

Most patients with mitral stenosis have normal or low left ventricular diastolic pressure and elevated left atrial pressure. The mitral inflow pattern reflects the valvular disease rendering the usual Doppler markers of no value in assessing diastolic function. However, left atrial pressure is often a clinically important question. In these patients, shortening of IVRT and increased mitral E wave velocity correspond to an elevated early left atrial pressure. A more complex parameter, IVRT / (T_E – T_e') has been reported to correlate reasonably well with mean left atrial pressure. This is the ratio of IVRT to the time difference between the mitral E peak velocity and the annular e’. A ratio of less than 2 suggests elevated left atrial pressure. In patients with mitral stenosis, E/e’ has not been useful to predict left atrial pressure.
FIGURE 6.43. This case demonstrates worsening of diastolic function over time in a patient with ischemic cardiomyopathy and severe systolic dysfunction. The two studies were recorded 9 months apart. On the first study (A to D), there is impaired relaxation but no signs of elevated diastolic filling pressure. On the second study (E to H), note the marked change in diastolic function without a significant change in heart rate or ejection fraction. It shows restrictive filling with elevated left ventricular filling pressure and right ventricular systolic pressure.
Mitral regurgitation is usually associated with increased compliance of both the left atrium and ventricle. When severe, it is associated with a high E wave velocity, reflecting the high LA-LV pressure gradient in early diastole and the increased antegrade diastolic flow. The pulmonary venous systolic wave is often blunted. In these patients, the E/e’ may be useful to predict filling pressures, but only in the presence of a depressed ejection fraction. As in mitral stenosis, the ratio of IVRT / (T_E – T_e’) correlates reasonably well with pulmonary capillary wedge pressure.
FIGURE 6.44. An example of constrictive pericarditis is provided. **A:** Mitral inflow shows an E/A ratio >2 with a short E wave deceleration time. **B:** Exaggerated respiratory variation in mitral inflow E wave velocity is present. **C** and **D:** As is typical of constriction, the septal e’ (19 cm/s) is greater than the lateral e’ (17 cm/s). All of these findings are suggestive of constrictive pericarditis.
FIGURE 6.45. Pulsed Doppler recording of mitral inflow and hepatic vein flow recorded in a patient with constrictive pericarditis. **A:** Note the marked respiratory variation in mitral E-wave velocity. **B:** This is associated with exaggerated early expiratory (E) hepatic vein flow reversal.

**Hypertrophic Cardiomyopathy**

Neither the E/A ratio nor mitral deceleration time are helpful in hypertrophic cardiomyopathy. E/e′ is of value, but seems to exhibit greater variability (and less predictability) in this population. Of the parameters that have been
studied, the time difference between mitral A wave duration and pulmonary venous A wave duration (Ar – A), may correlate best with filling pressure. Other parameters that may prove of some value include pulmonary artery pressure, pulmonary vein atrial reversal velocity, and left atrial volume. Clearly, this represents a challenging area for the noninvasive prediction of diastolic function and filling pressures.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Constrictive Pericarditis</th>
<th>Restrictive Cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>LA volume</td>
<td>Dilated</td>
<td>Dilated</td>
</tr>
<tr>
<td>LV contractility</td>
<td>Usually normal</td>
<td>Normal to mildly reduced</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>&gt;1.5</td>
<td>&gt;1.5</td>
</tr>
<tr>
<td>Response to Valsalva</td>
<td>E variation &gt;25%</td>
<td>Minimal respiratory change</td>
</tr>
<tr>
<td>Deceleration time (ms)</td>
<td>&lt;160</td>
<td>&lt;160</td>
</tr>
<tr>
<td>e’ (septal, cm/s)</td>
<td>&gt;8</td>
<td>&lt;8</td>
</tr>
<tr>
<td>e’, septal vs. lateral</td>
<td>Septal &gt; lateral</td>
<td>Lateral &gt; septal</td>
</tr>
<tr>
<td>Hepatic vein flow</td>
<td>Expiratory diastolic reversal</td>
<td>Inspiratory diastolic reversal</td>
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</table>

<table>
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<th>Study</th>
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<th>Population</th>
<th>Cutoff Value</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Giannuzzi et al., 1996</td>
<td>DT</td>
<td>508 pts, low EF</td>
<td>125 ms</td>
<td>Event-free survival 77% if DT &gt;125 ms, 18% if DT &lt;125 ms</td>
</tr>
<tr>
<td>Pozzoli et al., 1997</td>
<td>Mitral inflow pattern</td>
<td>173 pts, CHF, low EF</td>
<td>Response to Δ loading</td>
<td>Event rate 51% with unresponsive RF, 19% responsive RF, 6% without RF</td>
</tr>
<tr>
<td>Hansen et al., 2001</td>
<td>Mitral inflow pattern</td>
<td>311 pts, CM</td>
<td>RF pattern vs. all others</td>
<td>2-yr survival 52% with RF, 80% without RF</td>
</tr>
<tr>
<td>Bella et al., 2002</td>
<td>E/A</td>
<td>3,008 American</td>
<td>Abnormal defined as</td>
<td>3-yr all-cause mortality 12% if abnormal, 6% if normal</td>
</tr>
</tbody>
</table>

Table 6.7  DISTINGUISHING CONSTRICITVE PERICARDITIS FROM RESTRICTIVE CARDIOMYOPATHY

Table 6.8  PROGNOSTIC SIGNIFICANCE OF ECHO DOPPLER PARAMETERS IN DIASTOLIC DYSFUNCTION
### PROGNOSIS IN PATIENTS WITH DIASTOLIC DYSFUNCTION

Several of the Doppler parameters described above also yield prognostic information. Most of these studies have focused on patients with systolic dysfunction (i.e., reduced ejection fraction) or acute myocardial infarction. These are summarized in Table 6.8. For example, in patients with acute myocardial infarction, a mitral deceleration time <140 ms predicts a poor short- and intermediate-term prognosis. The prognostic value of this finding

<table>
<thead>
<tr>
<th>Study (Year)</th>
<th>Parameter</th>
<th>Value</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hillis et al., 2004</td>
<td>E/e’</td>
<td>250 pts, acute MI</td>
<td>&lt;0.6 or &gt;1.5</td>
</tr>
<tr>
<td>Wang et al., 2005</td>
<td>e’</td>
<td>182 pts, EF &lt;50%</td>
<td>3 cm/s</td>
</tr>
<tr>
<td>Dini et al., 2000</td>
<td>DT and Ar – A</td>
<td>145 pts, CM</td>
<td>DT &lt;130 ms, Ar – A &gt;30 ms</td>
</tr>
<tr>
<td>Okura et al., 2006</td>
<td>E/e’</td>
<td>230 pts, nonvalvular AF</td>
<td>15</td>
</tr>
<tr>
<td>Bruch et al., 2007</td>
<td>E/e’</td>
<td>370 pts, CM and MR</td>
<td>13.5</td>
</tr>
<tr>
<td>Takemoto et al., 2005</td>
<td>LA volume index</td>
<td>1,375 elderly pts, normal EF</td>
<td>&lt;28, 28–37, &gt;37 mL/m²</td>
</tr>
<tr>
<td>Solomon et al., 2012</td>
<td>LA volume index, E/A, E/e’</td>
<td>300 pts with HFpEF</td>
<td>Over a 36-wk study, Δ in LA volume correlated with improved diastolic function and lower LVFP</td>
</tr>
<tr>
<td>Shah et al., 2015</td>
<td>LA volume index, E/A, E/e’</td>
<td>239 pts with HFpEF</td>
<td>Increases in LV volume and E/A ratio were predictive of a poor outcome</td>
</tr>
</tbody>
</table>

AF, atrial fibrillation; Ar, atrial reversal of pulmonary venous flow; CM, cardiomyopathy; DT, deceleration time; EF, ejection fraction; HF, heart failure; HFpEF, heart failure with preserved ejection fraction; LA, left atrial; LVFP, left ventricular filling pressure; MI, myocardial infarction; MR, mitral regurgitation; RF, restrictive filling pattern. Other abbreviations as in text.

appears to be incremental, or in some cases even more powerful, than the degree of systolic dysfunction. The E/e′ ratio has been studied in a variety of conditions and appears to provide similar prognostic data. Using mitral inflow pattern to predict outcome, several studies have shown that a restrictive filling pattern conveys a poor prognosis in heart failure patients. In most studies, irreversibility of the pattern carries a much poorer prognosis than if the pattern is reversible.

More recently, left atrial volume has also been examined for its prognostic value. Like other parameters of diastolic function, increasing left atrial volume is generally associated with increasing risk. Whether it provides incremental prognostic information or is superior to other markers has not yet been established. Finally, although preliminary, abnormal untwisting, or torsion, derived from the speckle tracking technique may prove useful for predicting risk.

**Suggested Readings**

**GENERAL CONCEPTS**


**HEMODYNAMICS**


**Prognosis**


**Stress Testing**

Burgess MI, Jenkins C, Sharman JE, Marwick TH. Diastolic stress echocardiography: hemodynamic


**TECHNIQUES AND METHODOLOGY**


To view video clips associated with the chapter, please access the eBook bundled with
this text whenever this icon is present.
Left Atrium

In the early history of echocardiography, the left atrium was one of the first cardiac structures to be identified, recorded, and analyzed. A relatively oval-shaped chamber with thin, muscular walls, the left atrium is easily visualized posterior to the aortic root and superior to the left ventricle. With the advent of two-dimensional echocardiography and Doppler, the shape, size, and function of the chamber could be assessed. More recently, using three-dimensional and strain imaging, the ability to thoroughly interrogate left atrial structure and function has become possible.

**Left Atrial Dimensions and Volume**

The left atrium serves as a reservoir for blood draining the pulmonary veins during ventricular systole and as a conduit for that blood during early diastole. In late diastole, the left atrium becomes a muscular pump to complete the process of left ventricular filling before ventricular contraction and mitral valve closure. Thus, changes in left atrial dimensions and volumes mirror this continuous process of filling and emptying and have been a topic of intense study using echocardiographic techniques.

The left atrium can be visualized in a number of echocardiographic views including the parasternal long- and short-axis and the apical four- and two-chamber views (Fig. 7.1). The major and minor dimensions, area, and volumes have been measured from each of these perspectives. Because no single tomographic view conveys complete information about a three-
dimensional structure, it is recommended that a combination of two or more imaging planes be used for these purposes.

In each plane, one or more linear dimensions can be measured and the area of the left atrium can be traced. Historically, left atrial size was determined using M-mode echocardiography from the parasternal window (Fig. 7.2A). Because the position of the left atrium relative to the scan plane could not be determined with M-mode echocardiography, the assumption that this dimension corresponded to a true anteroposterior measurement represented a significant limitation. For example, if the recording was made from a lower interspace, an oblique dimension was obtained, and the left atrial size was overestimated. This problem can be avoided using two-dimensional echocardiography, ensuring that the measurement plane is properly oriented relative to the chamber (Fig. 7.2B). An important difference between these two approaches to left atrial measurement is provided in Figure 7.3. In Figure 7.3A, dimension X (7.0 cm) is correctly aligned relative to the left atrial chamber. If a dimension along a raster line had been used, as would occur with M-mode echocardiography, dimension Y (7.8 cm) would be the result. Figure 7.3B is another example of proper alignment from a patient with a dilated left atrium.
FIGURE 7.1. The left atrium can be visualized from several different echocardiographic views.

FIGURE 7.2. In A, an M-mode echocardiogram through the base of the heart
offers one approach to measuring left atrial size. In B, using two-dimensional echocardiography, a properly oriented linear dimension is obtained. By convention, the measurement is performed at end-systole when left atrial volume is greatest.

An additional challenge in measuring the left atrial size involves the precise definition of the posterior left atrial wall. In many patients, hazy, amorphous echoes can often be seen lining the posterior wall. These may be due to artifact or stagnant blood and can sometimes be eliminated by changing the gain or adjusting the angle of the transducer. Side lobes from a calcified annulus or highly reflective atrioventricular groove can also obscure the location of the posterior left atrial wall.

Although a relationship between this measurement and left atrial volume clearly exists, linear dimensions provide an incomplete picture of true left atrial size. This is because the pattern of left atrial dilation is variable, and in part depends on constraint by adjacent structures, such as the aorta. Dilation of the ascending aorta can distort the anteroposterior dimension, whereas dilation of the descending aorta can encroach on the left atrium posteriorly (Fig. 7.4). In addition, other mediastinal masses can alter left atrial shape and geometry. Figure 7.5 is an example of left atrial compression from a mediastinal lymphoma. The left atrium is completely distorted. The left atrial size cannot be assessed and its function is obviously impaired. Thus, an accurate assessment of left atrial size requires visualization of the chamber from multiple views and an appreciation of the limitations of relying on any single plane.

FIGURE 7.3. A limitation of M-mode echocardiography is the lack of spatial orientation. This can result in inaccurate measurement of true left atrial
dimension. **A:** Measurement $Y$ (7.8 cm) represents a measurement that would have been recorded using the M-mode approach. The true left atrial dimension is better approximated by measurement $X$ (7.0 cm). Two-dimensional echocardiography provides spatial orientation and avoids the problem of oblique measurements. **B:** Correct orientation of a left atrial minor-axis measurement is demonstrated.

**FIGURE 7.4.** An apical four-chamber view is shown in a patient with a thoracic aortic aneurysm. The descending aorta (arrows) distorts the left atrial shape and creates the appearance of a mass within the chamber.
coming soon

Video 7-4
FIGURE 7.5. A subcostal four-chamber view in a patient with a mediastinal lymphoma is shown. External compression of the left atrium by the tumor creates the appearance of a mass (arrows).
Because of the many limitations described above, it is recommended that left atrial size be assessed based on a measure of chamber volume. This is typically performed at end-systole, just before mitral valve opening. A common approach involves the area-length technique from the apical four- and two-chamber views. Using this approach, the area of the left atrium is measured by planimetry of both apical views (Fig. 7.6). Then, a linear dimension, or length, is measured from the center of the mitral annulus to the superior border of the chamber. When length is measured in both four- and two-chamber views, the shorter dimension should be used. Left atrial volume is then calculated as

\[
\text{Volume} = \frac{8}{3\pi} \left[ (A_1 \times A_2) + L \right], \quad \text{or} \quad [\text{Eq. 7.1}]
\]

\[
\text{Volume} = 0.85 \left[ (A_1 \times A_2) + L \right]
\]

where \(A_1\) is the area in one plane and \(A_2\) is the area in the orthogonal plane, \(L\) is the major-axis linear dimension, and 0.85 is derived from \(8/3\pi\). Other formulae have been proposed and most yield similar results. Another approach uses the biplane disc summation method. The formula is

\[
\text{Volume} = \frac{\pi}{4} (h) \sum (D_1) (D_2) \quad [\text{Eq. 7.2}]
\]

For this approach, the areas and long-axis dimensions are obtained from the four- and two-chamber views. By tracing the area, the transverse dimension of each disc is obtained automatically, from which the volume is derived.
Once calculated, volume is then indexed to body surface area, with 34 mL/m$^2$ used as the upper limit of normal. Three-dimensional echocardiography is also being utilized with increasing frequency for this purpose and will likely become the technique of choice in the future. Recent studies using these methods confirm the powerful prognostic value of left atrial volume in a variety of situations.

Other indirect measures of left atrial size are also available. For example, the ratio of the aortic root diameter to the left atrial short-axis dimension provides a qualitative estimate that is often used in practice. In normal subjects, the ratio of these two dimensions is approximately 1:1. A significant change in this ratio is a useful visual indicator of an abnormal left atrial size. Similarly, bowing of the atrial septum into the right atrial cavity indicates left atrial dilation and/or elevated left atrial pressure (Fig. 7.7). This is most easily appreciated using the apical four-chamber view. Finally, isolated dilation of the left atrial appendage has been reported. Although transesophageal echocardiography is most useful for detecting left atrial appendage aneurysms, this abnormality can also be seen from a transthoracic approach.

In summary, some measure of left atrial size should be a part of most echocardiographic examinations. Linear measurements provide limited and potentially misleading data on chamber size but are easy to perform and have traditionally been reported in clinical studies. If the normally spherical left atrium is distorted, for example, linear dimensions may not accurately reflect chamber size. As a result, measuring and reporting left atrial volume is now considered a more accurate and clinically relevant approach. While this is currently performed using two-dimensional imaging, it is quite likely that three-dimensional echocardiography ultimately will prove superior for this purpose.

**Left Atrial Function**

Although not routinely reported, left atrial function has relevance in several disease states and can be assessed using two-dimensional imaging, strain, and Doppler techniques. Contraction of the left atrium, represented by the P wave on the electrocardiogram, occurs in late diastole and corresponds to the final phase of left ventricular filling before mitral valve closure. This is recorded using Doppler as the A wave of mitral flow. Both the maximal A-wave
velocity and the A-wave time velocity integral correlate with the degree of contractility. Loss of coordinated left atrial contractility, as occurs in atrial fibrillation, is associated with the absence of the mitral A wave and sometimes the presence of small F waves. Thus, the Doppler A wave and the P wave of the surface electrocardiogram represent, respectively, the mechanical and electric manifestations of atrial systole. In most cases, their presence or absence is correlated; both are present in sinus rhythm and both are absent in atrial fibrillation. Figure 7.8 includes three examples of mitral flow from patients with atrial fibrillation before (bottom) and after (top) cardioversion. Note the prominent A waves in patient C once sinus rhythm is restored. This correlation is not always present, however. For example, immediately after cardioversion, electric activity may return, producing P waves on the electrocardiogram, before coordinated mechanical function recovers. This results in diminutive or absent Doppler A waves, as is illustrated by patient B.
FIGURE 7.6. Left atrial volume can be measured in various ways, with the area-length method being one of the most common. A: From the four-chamber view, left atrial area and long-axis dimension are obtained. B: Similar measurements are taken from the two-chamber view. C: Volume is then determined using a biplane area-length technique as shown in the schematic.

More recently, strain imaging with speckle tracking has been applied to left atrial mechanics. As with the left ventricle, longitudinal strain and strain rate curves can be generated throughout the cardiac cycle and both regional and average values can be quantified. Normal values and information on reproducibility have now been published. Although technically challenging, it appears that strain is a sensitive but nonspecific indicator of left atrial dysfunction. It has been correlated with other markers of chronic dysfunction,
such as left atrial volume and pressure, and appears to be a relatively early indicator of dysfunction. In terms of specific uses, it is still being studied in a variety of areas such as atrial fibrillation, where strain values may be a predictor of response to therapy or the success of ablation. One promising area of investigation is heart failure with preserved ejection fraction (HFpEF), in which abnormal left atrial strain may help differentiate patients with abnormal diastolic function from those with true HFpEF.

With transesophageal echocardiography, left atrial appendage function can also be assessed. Using pulsed Doppler imaging, with the sample volume positioned at the mouth of the appendage, the maximal velocity during atrial contraction can be measured (Fig. 7.9). This velocity corresponds to the force of atrial appendage contraction or emptying. In normal individuals, the left atrial appendage emptying velocity is greater than 50 cm/s. Significantly, lower velocities occur in patients with atrial fibrillation, and this finding has been associated with a predisposition for the development of left atrial thrombus and the risk of thromboembolism.
The interatrial septum reflects the relative pressure difference between the atria. In this example, the septum bows (arrows) into the right atrial cavity indicating elevated left atrial pressure.

The most important pathologic condition affecting the left atrial appendage is the development of a thrombus. This is a common complication of mitral stenosis or atrial fibrillation and is associated with an increased risk of systemic embolic events, especially strokes. Detecting left atrial appendage thrombi is, therefore, of critical importance and is one of the most common reasons to perform an echocardiogram. Transthoracic echocardiography is suboptimal for this purpose and not should be relied on
to detect or exclude a thrombus in the left atrium. Transesophageal echocardiography, however, is a very accurate technique to interrogate the left atrium for thrombi. From a variety of planes, the appendage can be easily visualized. It lies just below the left upper pulmonary vein and is separated from the vein by a ridge of tissue. This ridge is sometimes quite prominent and may be confused with abnormal masses or thrombi. Color Doppler is often helpful to distinguish the appendage from the pulmonary vein (Figs. 7.10 and 7.11). To reliably exclude the presence of a thrombus, a thorough inspection of the appendage is required. It also contains small pectinate muscles along its surface that must be differentiated from thrombi. Because the appendage is multilobed in most patients, multiple views are needed for a complete evaluation (Fig. 7.12).

**ATRIAL SEPTUM**

Abnormalities of the atrial septum are relatively common and usually congenital in origin. These include a patent foramen ovale (PFO), atrial septal defect (discussed in Chapter 19), and aneurysms of the atrial septum. PFO is very common, occurring in 25% to 30% of all adults. Unlike atrial septal defect, a PFO represents a failure of the primum and secundum septa to fuse, allowing intermittent blood flow in a bidirectional fashion between the atria. Thus, the septum appears structurally intact, but shunting can be demonstrated by either contrast or color flow imaging. Occasionally, a tunnel-like gap between the two portions of the septa can be intermittently visualized because the transatrial pressure gradient changes with respiration. A PFO is frequently associated with exaggerated mobility of the atrial septum and, in the extreme form, an atrial septal aneurysm. Although a PFO can be seen from transthoracic imaging (Fig. 7.13), transesophageal echocardiography is more sensitive and provides a more complete assessment. To reliably characterize a PFO, the septum must first be thoroughly examined to exclude an atrial septal defect. Then contrast echocardiography, color Doppler imaging, or both techniques should be performed. Evidence of right-to-left shunting is respiratory cycle dependent and will, therefore, be intermittent. Once contrast appears in the right atrium, shunting should occur within three or four cardiac cycles. The appearance of
bubbles in the left atrium is typically phasic and respiratory cycle dependent. A large shunt through a PFO is shown in Figure 7.14. Figure 7.15 is an example of a PFO detected with transesophageal echocardiography. Dense contrast obscures the right atrium, but the bubbles are clearly seen crossing the septum and appearing in the left atrium. Although precise quantification of shunting is not possible, contrast echocardiography can provide a rough estimate of the magnitude, based on the number of bubbles that appear in the left atrium within three or four beats. Figure 7.16 shows a PFO with exaggerated septal mobility and a clearly defined tunnel through which shunting is easily demonstrated. Additional information on the use of contrast techniques for the evaluation of shunts is found in Chapter 3.

**FIGURE 7.8.** Three patients with atrial fibrillation are shown before (bottom) and after (top) cardioversion. Mitral inflow in each case demonstrates the absence of atrial contraction (A wave) while in atrial fibrillation, but a variable degree of recovery of atrial function after sinus rhythm is restored.
FIGURE 7.9. The left atrial appendage emptying velocity can be recorded using pulsed Doppler imaging. **A:** In a patient in sinus rhythm, the emptying velocity during atrial systole is approximately 60 cm/s. In atrial fibrillation (B), the emptying velocity is variable and much lower, indicating a lack of coordinated contractility. **C:** Another patient during atrial fibrillation. In this case, the velocity is higher.
FIGURE 7.10. A close-up view from a patient with left atrial appendage thrombus (arrow), the position of the appendage is demonstrated relative to the left upper pulmonary vein (asterisk). In B, color Doppler reveals blood flow in the vein (asterisk), just above the appendage.

Figure 7.11. A left atrial appendage thrombus is readily demonstrated (arrow) with transesophageal echocardiography.

Appearance of contrast in the left atrium after more than four beats
suggests the possibility of transpulmonary shunting, usually through an arteriovenous malformation. Although distinguishing this entity from a PFO is difficult, in most cases a pulmonary shunt is characterized by a continuous, even flow of contrast into the posterior left atrium several cycles after it first appeared in the right atrium (Fig. 7.17). The distinction is more easily made using transesophageal echocardiography where, in the presence of a PFO, bubbles can be directly visualized crossing the interatrial septum (as in Fig. 7.15). Figure 7.18 was recorded from a patient with both a PFO and known pulmonary arteriovenous malformations. With real-time imaging, an immediate appearance of bubbles in the right atrium was demonstrated followed several cycles later by a bolus of contrast-containing blood via the pulmonary veins.
**FIGURE 7.12.** Five views of the left atrial appendage are recorded at various angles of rotation of the scan plane. Note how the appearance of the appendage varies in the different views. The relationship of the appendage to the left upper pulmonary vein (asterisk) is also demonstrated.

**FIGURE 7.13.** Sequential frames recorded during injection of agitated saline, from a patient with a small PFO. As the contrast enters the right heart (A), bubbles gradually appear within the left atrium (B) and then, left ventricle (C).
An atrial septal aneurysm is a redundancy of the midportion of the atrial septum that results in excess mobility and billowing of the tissue in this region (Fig. 7.19). Because some motion of the atrial septum is normal, a standardized definition of atrial septal aneurysm requires maximal deviation of the aneurysmal tissue of at least 10 mm from the plane of the septum. The motion of the aneurysm reflects the relative pressure gradient between left and right atria, and thus the outpouching will usually occur in both directions over the course of the cardiac cycle (Fig. 7.20). In the example, redundant tissue in the area of the fossa ovalis billows from left to right, reflecting the changes in relative pressure between the two atria. Figures 7.21 and 7.22 are examples of septal aneurysms protruding into the right atrium. In Figure 7.22, the redundant aneurysmal tissue nearly extends through the tricuspid valve during diastole.

An atrial septal aneurysm can be identified by transthoracic echocardiography from the basal parasternal short-axis view or the apical four-chamber view. However, these aneurysms are more readily visualized using transesophageal echocardiography in the four-chamber view. The total excursion of the aneurysmal tissue can be assessed (Fig. 7.23) and the presence or absence of an associated shunt can be detected with either using color flow imaging or, more accurately, using agitated saline. Atrial septal aneurysms are associated with either a PFO or an atrial septal defect in as many as 75% of cases. The combination of an atrial septal aneurysm and a PFO has recently been associated with substantial risk of thromboembolism.
When an atrial septal aneurysm is detected, it is often appropriate to perform a venous saline contrast injection to search for an associated PFO because its presence may alter management. Both the left and right sides of the pouch should be carefully examined for the presence of thrombus.

**FIGURE 7.14.** From a patient with a PFO and large shunt, a bolus of contrast-containing blood crosses the PFO into the left atrium (A, arrow). Within a few cardiac cycles (B), the bubbles fill the left heart. [Video 7-14](coming soon)
FIGURE 7.15. An example of a PFO demonstrated during transesophageal echocardiography by injection of agitated saline into a peripheral vein. The 3 frames, separated by a few seconds, show the gradual appearance of contrast crossing from the right to left atrium through the PFO (arrows).
FIGURE 7.16. Contrast injection demonstrates a patent foramen ovale on transesophageal echocardiography. In this case, increased mobility of the atrial septum is present. The tunnel-like gap within the interatrial septum is evident (arrow), and bubbles can be seen traversing the patent foramen ovale from right to left. Video 7-16

coming soon
FIGURE 7.17. An example of a transpulmonary shunt due to a pulmonary arteriovenous malformation is shown. In A, following venous injection, contrast-containing blood is seen filling the right heart. In B, several cardiac cycles elapse before a few bubbles are seen in the left heart. In C, 2 cardiac cycles later, a bolus of contrast fills the left atrium and ventricle. It can be seen entering the left atrium via the pulmonary veins (arrows).
FIGURE 7.18. Both a small PFO and a transpulmonary shunt are present in this patient. In real time, the entrance of contrast into the left atrium appears in two phases, with a small number of bubbles crossing the septum early (arrow), followed by a larger bolus several cycles later. The late arriving contrast could be seen arising from the pulmonary veins rather than the fossa ovalis.
FIGURE 7.19. A subcostal four-chamber view demonstrates an atrial septal aneurysm (arrow) bowing into the right atrium.
Video 7-19
FIGURE 7.20. An atrial septal aneurysm (arrow) billows into the right atrium (A) and left atrium (B). With contrast (C), phasic flow through a PFO is demonstrated.
FIGURE 7.21. A large atrial septal aneurysm extends into the right atrium, outlined by contrast-containing blood. No evidence of a PFO was demonstrated.
FIGURE 7.22. A: An apical four-chamber view demonstrates an extreme form of an atrial septal aneurysm with a “windsock” appearance of the aneurysmal tissue into the right atrium and partially through the tricuspid valve (arrows). B: After contrast agent injection, the windsock is outlined by the contrast that flows around it from the right atrium to the right ventricle. In addition, the presence of a patent foramen ovale allows some contrast agent to cross into the left heart.
coming soon

Video 7-22
An acquired abnormality of the atrial septum is lipomatous hypertrophy. This involves fatty infiltration of the superior and inferior portions of the septum, typically sparing the fossa ovalis. Such a distribution creates a “dumbbell-shaped” appearance which, when present, allows a diagnosis of lipomatous hypertrophy to be made with confidence (Fig 7.24). Less commonly, diffuse septal infiltration of fatty tissue occurs. In these cases, the condition may be confused with malignancy or thrombus, and imaging with MRI may be helpful to distinguish fatty tissue from tumor and/or thrombus.

PULMONARY VEINS

In most normal individuals, four distinct pulmonary veins drain blood from the lungs to the left atrium. These four veins enter the left atrium relatively close together along the superior portion of the posterior wall. The veins from the left lung enter laterally, whereas the veins from the right lung enter more medially. It is often possible to visualize the entrance of one or two pulmonary veins into the left atrium using transthoracic echocardiography from the four-chamber view. An example of this is provided in Figure 7.25. From this same view, a recording of pulmonary venous inflow is possible. This is best accomplished by first using color flow imaging to identify one or
more veins and then positioning the pulsed Doppler sample volume within the mouth of the vein as it enters the left atrium. Using this approach, pulmonary venous flow patterns can be recorded routinely, and several examples are provided in Figure 7.26. A unique view to record the pulmonary veins is the “crab view,” which is recorded from the suprasternal notch with some posterior angulation. Directly below the right pulmonary artery, the posterior wall of the left atrium is visualized and the pulmonary veins occasionally can be recorded.

The entrance of the pulmonary veins into the left atrium is more completely recorded using transesophageal echocardiography. In most patients, all four veins can be visualized. To record the left pulmonary veins, a vertical imaging plane is used and the transducer is rotated to the patient’s far left (Fig. 7.27). The atrial appendage may be useful as a landmark to identify the left upper vein. Then, by gradually advancing the probe, the lower vein is seen. To record the right veins, set the imaging plane at 45 to 60 degrees and rotate the shaft of the probe clockwise to the patient’s far right. The right veins are usually seen together, forming a V shape as they drain into the left atrium (Fig. 7.28).
FIGURE 7.24. An example of an extreme form of lipomatous hypertrophy is shown from a young woman with palpitations. Transesophageal echocardiography demonstrates extensive hypertrophy of the septum, with typical sparing of the fossa ovalis (A). In addition, mobile round structures were seen (arrows) attached to the lipomatous material (A and B). On MRI, the appearance confirmed that the atrial masses were globular lipomatous materials extending from the interatrial septum (C).

FIGURE 7.25. Apical four-chamber (A) and two-chamber (B) views demonstrate the entrance of the pulmonary veins (arrows) into the superior portion of the left atrium.
FIGURE 7.26. Pulmonary venous inflow can be recorded from transthoracic imaging. A: Three distinct waves are demonstrated: an antegrade wave during systole (PV$_S$) and diastole (PV$_d$) and a retrograde wave coincident with atrial systole (PV$_a$). B: Two distinct velocity components during systole are present. C: A relative increase in the proportion of flow during diastole is noted.
FIGURE 7.27. A transesophageal echocardiogram demonstrates the entrance of the left upper pulmonary vein (LUPV) (A) and left lower pulmonary vein (LLPV) (B) into the left atrium.

FIGURE 7.28. A transesophageal echocardiogram shows the entrance of the right lower pulmonary vein (RLPV) and right upper pulmonary vein (RUPV) into the left atrium.

Normal pulmonary venous flow has three phases: antegrade flow occurs in systole and early diastole and some retrograde flow occurs after atrial contraction in late diastole (Fig. 7.29A). The ratio of peak flow velocity in systole and diastole and the duration of the retrograde pulmonary venous A wave are useful parameters in the assessment of diastolic dysfunction. This topic is covered in Chapter 6. In addition, retrograde flow into the pulmonary
veins in late systole can be observed in patients with severe mitral regurgitation. A variety of pathologic states are also associated with abnormal pulmonary venous flow, including mitral stenosis, constrictive pericarditis, and restrictive cardiomyopathy. **Figure 7.29B** is an example of abnormal pulmonary venous flow in a patient with ischemic cardiomyopathy and elevated filling pressure. Note that the inflow occurs almost exclusively during diastole, indicating high left ventricular filling pressure and restrictive physiology. Stenosis of the pulmonary veins can be either congenital or acquired. An example of pulmonary vein stenosis occurring as a result of an atrial fibrillation ablation procedure is shown in **Figure 7.30**. In this example, color Doppler demonstrates increased, turbulent flow and the stenosis is confirmed on the spectral Doppler tracing. **Figure 7.31** demonstrates increased pulmonary venous flow velocity from a patient with hyperdynamic cardiac function. Finally, transesophageal echocardiography is very useful to demonstrate anomalous pulmonary venous connections, either in isolation or in association with atrial septal defects.
FIGURE 7.29. Pulmonary venous flow is recorded using transesophageal echocardiography. **A:** Normal pulmonary venous flow is demonstrated. **B:** Antegrade systolic flow (PV$_S$) is blunted and diastolic flow (PV$_d$) is increased in a patient with elevated left atrial pressure due to left heart failure.
FIGURE 7.30. Pulmonary vein stenosis caused by an atrial fibrillation ablation procedure is shown. In A and B, normal color and spectral Doppler flow patterns from an unaffected pulmonary vein are demonstrated. In C, from the same patient, color Doppler through the affected vein shows increased, turbulent flow (arrows). The presence of stenosis is confirmed with spectral Doppler (D), which shows increased antegrade velocity during both systole and diastole.
RIGHT ATRIUM

The right atrium is a thin-walled ovoid structure that receives inflow from the superior and inferior vena cavae and the coronary sinus. It can be visualized in several views and contains several distinct anatomic structures. Right atrial size and function have not been as well studied as the other chambers, although dilation of the right atrium frequently accompanies right ventricular volume and pressure overload as well as right ventricular failure. Measurement of the right atrium is usually performed from the apical four-chamber or subcostal view. Linear dimensions can be determined, and the normal range of right atrial size has been reported. Planimetry of right atrial area should also be performed to more directly assess chamber area and volume. These measurements are performed at end-systole, prior to tricuspid valve opening. This method is similar to that described earlier for the left atrium and is illustrated in Figures 7.32 and 7.33. The formula used to estimate right atrial volume from the four-chamber view is the single plane area-length method:
Flow in the left upper pulmonary vein is recorded from transesophageal echocardiography. In this example, moderately increased flow velocity is the result of a hyperdynamic state.

On a clinical basis, visual comparison of left and right atrial size from the apical four-chamber view is routinely performed. A right atrium that appears larger than the left atrium is qualitative evidence of chamber enlargement. Similar to the left atrium, the right atrium is at risk of compression by extracardiac structures within the liver or mediastinum. Distinguishing extracardiac compression from an intracardiac mass can be difficult. Figure 7.34 is an example of compression of the right atrium by a liver mass. Note how the mass causes distortion of right heart structures and bowing of the atrial septum toward the left. When viewed in real time, the mass was immobile and independent of cardiac motion. Figure 7.35 is an example of right atrial myxoma. Although less common than left atrial myxomas, the appearance and characteristics are similar. Metastatic melanoma, occupying a portion of the right atrium and extending into the interatrial septum, is shown in Figure 7.36.

\[
\text{Volume} = \frac{8}{3\pi} \times \left(\frac{A^2}{L}\right) \quad \text{[Eq. 7.3]}
\]
FIGURE 7.32. The schematic illustrates different approaches to assessing right atrial size. On the left, linear dimensions of the right atrium, the major (M) and minor (m) axes, are shown. In the middle, right atrial area can be obtained by planimetry, using the apical four-chamber view. On the right, chamber volume is determined using a single-plane area-length method, which can then be indexed to body surface area. In each case, normal values are provided below.
The right atrium is the site of several anatomic variants, which are occasionally mistaken for pathologic structures. These include the eustachian valve and the Chiari network. The eustachian valve is a remnant of the embryologic valve responsible for directing inferior vena caval blood across the atrial septum to the left atrium. Referred to as the right sinus valve or the valve of the inferior vena cava, this structure normally regresses during embryonic development. Lack of normal regression results in a variety of anomalies that range from a prominent (but physiologically insignificant) eustachian valve to partial or complete septation of the right atrium, a condition inappropriately referred to as cor triatriatum dexter. The eustachian valve is a rigid and protuberant structure that arises along the posterior margin of the inferior vena cava to the border of the fossa ovalis. It is most
easily visualized from a medially angulated parasternal long-axis view at the junction of the inferior vena cava and right atrium (Fig. 7.37). The eustachian valve varies considerably in size, from inconspicuous to quite prominent. Although usually immobile, it may occasionally demonstrate independent motion within the right atrium and can be confused with tumors, vegetations, or thrombi (Fig. 7.38). When large, the eustachian valve can divert the flow of blood within the right atrium. An example of nearly complete septation of the right atrium by a very prominent eustachian valve is shown in Figure 7.39. During contrast injection, this can result in both false-negative and false-positive evidence of an atrial septal defect. For example, the streaming effect can result in the appearance of non–contrast-containing blood in the area of the septum, incorrectly suggesting a left-to-right shunt.

**FIGURE 7.34.** Compression of the right atrium by a hepatoma (arrows) creates
the impression of a right atrial mass.

**FIGURE 7.35.** A large right atrial myxoma is seen on transesophageal echocardiography. In the four-chamber view (A), it can be seen in the superior portion of the chamber. In the short axis (B), it appears to fill the right atrium, but an attachment site is not visualized. Using MRI, the stalk attaching the tumor to the right atrial superior wall is indicated by the *arrow* (C).
FIGURE 7.36. A large mobile mass invading the interatrial septum and extending into the right atrium is shown. In the four-chamber view (A), the mass could be confused with a myxoma or a straddling thrombus. In the short-axis view (B), the bulky, irregular shape of the mass (arrow) and its distortion of the normal interatrial septal shape suggest malignancy. The mass is further characterized with MRI (C, arrow). At surgery, this was found to be metastatic melanoma.
FIGURE 7.37. A medially angulated parasternal view demonstrates the right ventricular inflow tract. In the inferior portion of the right atrium, a eustachian valve at the entrance of the inferior vena cava is shown (arrow).
FIGURE 7.38. A prominent eustachian valve is demonstrated (arrow).
FIGURE 7.39. An extreme form of a eustachian valve is demonstrated in this four-chamber view. The prominent ridge of tissue (arrow) results in almost
complete septation of the right atrium.

A Chiari network is a delicate-appearing, membranous structure arising near the orifice of the inferior vena cava and serving as the valve of the coronary sinus. It is highly mobile and usually fenestrated, and its site of attachment varies within the chamber (Fig. 7.40). Although sometimes confused with the eustachian valve, a Chiari network is more delicate and more mobile. Like the eustachian valve, it has little clinical significance but may be confused with pathologic structures, such as vegetations or thrombi.
FIGURE 7.40. A subcostal four-chamber view illustrates a Chiari network (arrows) within the right atrium. In real time, the Chiari network is a highly mobile structure.
FIGURE 7.41. The bicaval transesophageal echocardiographic view can be used to record the right atrial appendage (RAA). From a vertical plane, the probe must be rotated to the right to view this structure.
Right Atrial Thrombi

Thrombi can occur either in the body of the right atrium or within the atrial appendage, usually as a consequence of atrial fibrillation. The right atrial appendage is difficult to visualize on transthoracic imaging but can be recorded during transesophageal echocardiography (Fig. 7.41). Because the right atrial appendage is more trabeculated than its left-sided counterpart, distinguishing muscles from thrombi can be challenging. In patients with atrial fibrillation studied before elective cardioversion, an assessment of the right atrial appendage to exclude thrombi should be performed routinely. In most instances, however, when a thrombus develops in the right atrium, it occurs in the main body of the chamber, a consequence of low flow, atrial arrhythmia, or the presence of foreign bodies (such as catheters or pacemaker leads). Thrombi in the right atrium are relatively common. Thromboemboli that arise in lower extremity or pelvic veins may occasionally be seen within the right atrium as a pulmonary embolus in transit (Figs. 7.42 and 7.43). Such masses usually have a multilobulated appearance and are freely mobile. They often have a worm-like shape, a reflection of the lower extremity veins in which they were formed. A thrombus that formed in the lower extremities and was recorded during transit through the right heart is shown in Figure 7.44. In this example, the thrombus is seen straddling the tricuspid valve. Distinguishing a thrombus from a tumor, especially renal cell carcinoma, may be difficult. Both can extend from the inferior vena cava into the right atrium and have a lobulated, mobile appearance. Other imaging techniques, such as an abdominal computed tomography, may be necessary to differentiate these entities.

Thrombi attached to indwelling catheters may be visualized with transthoracic echocardiography but are much more readily detected with transesophageal techniques. The ability to interrogate the entire right atrium as well as a portion of the superior vena cava is essential to detect such thrombi. Distinguishing a thrombus from vegetation is particularly difficult and may be impossible on echocardiographic grounds alone.
FIGURE 7.42. A lobulated thrombus is visualized within the right atrium using multiple transthoracic views from a patient with deep vein thrombosis and pancreatic cancer. The patient had recently suffered a pulmonary embolus. In real time, the tubular-shaped coil of thrombus (arrows) could be seen swirling within the chamber. The right ventricle is dilated and hypokinetic.

FIGURE 7.43. A thrombus can be seen within the IVC, just below the entrance into the right atrium, from the subcostal window (A, arrows). In B, the extension of the thrombus into the right atrium is indicated by the arrows.
coming soon

Video 7-43
FIGURE 7.44. A multilobed thrombus (arrows) is recorded straddling the tricuspid valve from the four-chamber view. The thrombus could be traced to the entrance of the inferior vena cava.

Right Atrial Blood Flow

Blood enters the right atrium via the inferior vena cava, the superior vena cava, and the coronary sinus. The location and orientation of the inferior vena cava facilitate its visualization from the subcostal views (Fig. 7.45). A highly
compliant vessel, the inferior vena cava changes shape and dimensions with changes in central venous pressure and the respiratory cycle. The size and respiratory variation of the inferior vena cava have been used to predict right atrial pressure. Inferior vena cava diameter should be measured at end-expiration just proximal to the junction with the right atrium. Dilation of the inferior vena cava suggests increased central venous pressure and may accompany volume overload states. The diameter of the inferior vena cava normally decreases more than 50% during inspiration. A blunted or absent inspiratory decrease in the inferior vena cava diameter suggests increased right atrial pressure. Since estimation of right atrial pressure is an essential component of the hemodynamic assessment of the right heart and the measurement of pulmonary artery systolic pressure, guidelines for doing so have been established. Although several schemes for estimating right atrial pressure based on inferior vena cava dimensions have been proposed, an algorithm, endorsed by the American Society of Echocardiography, is provided in Table 7.1. This simple approach addresses the two extremes of normal and severely elevated right atrial pressures. For in between situations, or when the findings do not fit the paradigm, an intermediate value of 5 to 10 mm Hg should be used. It is also recommended to use caution applying this approach to athletes, in whom a dilated inferior vena cava may be normal, and in patients on ventilators, whose inferior vena cava may not collapse.

<table>
<thead>
<tr>
<th>IVC Diameter (cm)</th>
<th>Response to Sniff</th>
<th>RA Pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤2.1</td>
<td>&gt;50% collapse</td>
<td>3</td>
</tr>
<tr>
<td>≤2.1</td>
<td>&lt;50% collapse</td>
<td>8</td>
</tr>
<tr>
<td>&gt;2.1</td>
<td>&lt;50% collapse</td>
<td>15</td>
</tr>
</tbody>
</table>


Both pulsed and color Doppler imaging can be used to record flow within
the inferior vena cava. Vena caval flow is occasionally visualized using color Doppler as a streaming effect from the vessel into the right atrium, extending along the septum. An example of this is shown in Figure 7.46. From the right ventricular inflow view (Fig. 7.46B), flow is seen emerging from the inferior vena cava, passing around the eustachian valve, and coursing into the right atrium. Such a pattern can occasionally be confused with flow through an atrial septal defect.

**FIGURE 7.45.** From the subcostal window, the inferior vena cava can be recorded as it passes through the diaphragm and enters the right atrium. A: Hepatic veins can be seen entering the inferior vena cava (arrow). B: The inferior vena cava is dilated and does not collapse with inspiration.

Video 7-45
FIGURE 7.46. Inferior vena caval flow can sometimes be recorded with color flow imaging as a streaming effect within the right atrium. The streaming effect is often confused with important pathology such as an atrial septal defect. This is demonstrated from the four-chamber view (A) and right ventricular inflow view (B).

Video 7-46

Doppler assessment of right atrial filling has relevance in several clinical situations. From the subcostal transducer location, alignment of the Doppler beam with inferior vena caval flow is difficult, and it has become customary to substitute hepatic vein flow for this purpose. Because hepatic vein flow and inferior vena caval flow are similar and because it is generally easier to align the Doppler signal with a hepatic vein, this is both useful and practical. An example of normal hepatic vein flow is shown in Figure 7.47. Antegrade flow (toward the right atrium) has two main components: a larger systolic wave and a slightly smaller diastolic wave. Between these two antegrade flow patterns, at end-systole, a small retrograde flow pattern may be recorded.
Likewise, during atrial systole, some retrograde flow is also present. Hepatic vein flow is respiratory cycle dependent with increased flow velocity during inspiration and decreased flow velocity (and a greater degree of retrograde flow) during expiration.

Several disease states result in characteristic abnormalities of hepatic vein flow (Fig. 7.48). As a surrogate for inferior vena caval flow, any condition that affects either right atrial pressure or filling will alter hepatic vein flow velocity. For example, increased right atrial pressure has been associated with a decrease in the systolic filling fraction of hepatic vein flow. Thus, as right atrial pressure increases, antegrade systolic hepatic vein flow decreases. In patients with severe tricuspid regurgitation, flow reversal during ventricular systole is characteristic. As the tricuspid regurgitant jet is transmitted retrograde into the right atrium, the normal antegrade systolic flow is replaced by a prominent retrograde wave. In the setting of atrial fibrillation, retrograde flow during atrial systole and the velocity of systolic antegrade flow are diminished. In contrast, pulmonary hypertension typically results in prominent flow reversal during atrial systole. Analysis of right atrial filling plays an important role in the evaluation of patients with restrictive physiology and constrictive pericarditis. These topics are discussed in Chapters 8 and 18.
The superior vena cava can be visualized from the suprasternal notch as a vertical structure just to the right of the aortic arch (Fig. 7.49) but is more readily evaluated using transesophageal echocardiography. Both long- and short-axis views of the vessel are possible (Fig. 7.50). Occlusion or external compression of the superior vena cava is a common clinical problem that can be assessed using echocardiography. The diagnosis can often be established using transthoracic imaging combined with color flow Doppler imaging. Because the underlying pathologic process may result in distorted anatomy, a precise diagnosis may be difficult using the transthoracic approach, and transesophageal echocardiography is often necessary.

**RIGHT VENTRICLE**

Echocardiographic evaluation of the right ventricle is hampered by its
unusual crescent shape, irregular endocardial surface, and complex contraction mechanism. These factors, coupled with the location of the right ventricle almost directly behind the sternum, combine to create formidable problems for the echocardiographer. A dependable anatomic feature of the right ventricle is the moderator band within its apex (Fig. 7.51). This structure helps identify the morphologic right ventricle and is best appreciated from the apical four-chamber view. A more trabeculated right ventricular apex is shown in Figure 7.52. This echocardiogram, recorded from a healthy, asymptomatic 24-year-old male, illustrates the range of apical anatomy that can be considered normal.

The normal right ventricle defies simplified assumptions regarding shape. Along the minor axis, the right ventricle has a characteristic crescent shape. Along the orthogonal long axis, however, the shape is more complex and variable. For this reason, no simple geometric three-dimensional figure accurately represents this chamber. Some simplifications that have been used include a parallelepiped (or three-dimensional parallelogram), a prism, and a pyramid with a triangular base. Contraction of the right ventricle is also complex. The pattern has been compared with the action of a bellows, in which minor-axis shortening is combined with significant long-axis shortening to draw the tricuspid annulus toward the apex. The low resistance of the pulmonary vascular circuit permits the right ventricle to eject a large volume of blood while performing a lesser degree of myocardial shortening. Relatively small movements of the walls, therefore, produce large ejection volumes, similar to a bellows.
Right Ventricular Dimensions and Volumes

A qualitative assessment of the right ventricle is a routine part of echocardiography. In the apical four-chamber view, for example, a visual comparison of right and left ventricular area permits a rough estimate of right ventricular volume to be made (Fig. 7.53). Normally, right ventricular size is approximately two-thirds that of the left ventricle. This estimate is based on a comparison of the relative sizes of the two ventricles from multiple views. More quantitative approaches, using two-dimensional echocardiography, are also available. Unlike the left ventricle, however, whose shape lends itself to simple geometric assumptions, the complex shape of the right ventricle greatly complicates volume quantification. This is particularly true of the normally shaped right ventricle. It is fortuitous that in patients with right
ventricular enlargement, the chamber’s shape becomes more ellipsoid, thereby facilitating the application of these quantitative approaches. In technically difficult studies, saline contrast can be helpful to delineate the right ventricular endocardial borders and help assess the chamber size and function.

Right ventricular dimensions should be obtained from both the inflow (apical four-chamber) and outflow (parasternal) views. Three linear dimensions can be obtained of the right ventricular outflow area from the parasternal long- and short-axis views (Fig. 7.54). From the apical four-chamber view, through proper alignment of the imaging plane, a long axis of the right ventricle is recorded. Care must be taken to avoid foreshortening and the image should be rotated to record the maximum right ventricular size. From this view, long-axis and transverse dimensions at the base and mid-chamber level should be obtained (Fig. 7.55). In addition, the right ventricular area from the four-chamber view should also be obtained by planimetry in systole and diastole (Fig. 7.56). From these measurements, fractional area change can be determined. A summary of these measurements is provided in the schematic (Fig. 7.57). From the subcostal four-chamber view, at end-diastole, the right ventricular wall thickness can be measured (Fig. 7.58). Normal and abnormal values for right ventricular size are provided in Table 7.2. By carefully recording the various measures of right ventricular size, both diagnostic and prognostic information about the right heart is obtained. Figure 7.59 is taken from a patient with recurrent pulmonary emboli. Note the increased right ventricular dimensions, from both the parasternal and the apical four-chamber views. The chamber was both enlarged and severely hypokinetic. Doppler imaging demonstrated evidence of severe pulmonary hypertension.
FIGURE 7.49. The superior vena cava can be visualized from the suprasternal notch as a vertical structure just to the right of the aortic arch (AA). RPA, right pulmonary artery.
FIGURE 7.50. The superior vena cava is best recorded with transesophageal
echocardiography. **A:** The bicaval view demonstrates both the superior vena cava and the inferior vena cava. **B:** A transverse plane at the base of the heart demonstrates the relationship between the aorta and superior vena cava. Posterior to the superior vena cava is the superior portion of the left atrium.

**FIGURE 7.51.** An apical four-chamber view demonstrates a moderator band (arrow) within the right ventricular apex.
FIGURE 7.52. Muscle trabeculations are commonly seen within the right ventricular apex, as indicated by the arrow in this healthy young individual.
FIGURE 7.53. A dilated right heart is clearly demonstrated from the apical four-chamber view in this patient with pulmonary hypertension secondary to recurrent pulmonary emboli. Note the enlarged right atrium and bowing of the interatrial septum into the left atrium. The right ventricle is dilated and hypokinetic.
FIGURE 7.54. Right ventricular linear dimensions are obtained from the outflow track area using both the parasternal long-axis (A) and short-axis (B) views. See text for details.
Linear dimensions, however, only serve as a surrogate for ventricular volume. To measure right ventricular volume, simplifying assumptions about shape are necessary. Both area-length and Simpson rule approaches have been undertaken. Because of the geometry of the right ventricle, accurate volume measurements from two-dimensional echocardiography are difficult and current guidelines do not recommend this approach. Alternatively, three-dimensional echocardiographic techniques have been applied to this problem. A major advantage of three-dimensional echocardiography is that assumptions about shape are no longer necessary and a complete echocardiographic rendering of the right ventricular cavity can be recorded and analyzed (Fig. 7.60). Multiple studies have confirmed the advantages of this approach to both volume and function of the right ventricle. In addition, both age- and sex-specific reference values for volumes and ejection fraction have been published. Right ventricular volumes derived from three-
dimensional echocardiography correlate well with cardiac MRI although these tend to underestimate true volume.

An additional approach to right ventricular systolic function involves the quantitative assessment of tricuspid valve annular motion during systole, or TAPSE (tricuspid annular plane systolic excursion). This can be recorded from the apical four-chamber view using M-mode or Doppler tissue imaging techniques (Fig. 7.61). Tricuspid annular motion during systole is normally between 17 and 28 mm. A value less than 17 mm is suggestive of right ventricular systolic dysfunction. Reduced TAPSE has been reported in a variety of conditions affecting the right heart and has been associated with a poor prognosis. Normal values for TAPSE, however, may be seen in the setting of right ventricular systolic dysfunction, if the left ventricle is hyperdynamic, resulting in a tethering effect on the right ventricle and tricuspid annulus.

A related method employs tissue Doppler to measure the tricuspid annular velocity throughout the cardiac cycle (Fig. 7.62). This is done from the four-chamber view using the lateral annulus for sample volume placement. From this recording, the peak velocity ($S'$) of the systolic annular motion is derived. The mean value in normal populations is approximately 15 cm/s and velocities less than 10 cm/s suggest systolic dysfunction. Peak systolic annular velocity is a surrogate for global right ventricular systolic function and has been shown to be reduced in several conditions. For example, this value is lower in patients with inferior myocardial infarction, especially if there is evidence of right ventricular involvement.
FIGURE 7.56. From the apical four-chamber view, right ventricular area during diastole (A) and systole (B) are obtained by planimetry so that fractional area change (FAC) can be derived.
FIGURE 7.57. This schematic summarizes the various linear and area measures recommended to assess right ventricular size and function.

FIGURE 7.58. Right ventricular free wall thickness is measured at end-diastole from the mid portion of the wall in the subcostal view. This places the endocardium and epicardium perpendicular to the ultrasound beam to ensure optimal definition of the two surfaces.

Table 7.2  RANGE AND LIMITS OF NORMAL FOR MEASURES OF RIGHT VENTRICULAR SIZE AND FUNCTION

<table>
<thead>
<tr>
<th>Parameter</th>
<th>View</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Size</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parameter</td>
<td>View</td>
<td>Range</td>
</tr>
<tr>
<td>----------------------------------------------</td>
<td>---------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Diameter at the base (mm)</td>
<td>4-chamber</td>
<td>25–41</td>
</tr>
<tr>
<td>Diameter at mid RV (mm)</td>
<td>4-chamber</td>
<td>19–35</td>
</tr>
<tr>
<td>RV length (mm)</td>
<td>4-chamber</td>
<td>59–83</td>
</tr>
<tr>
<td>Diameter proximal RVOT (mm)</td>
<td>PLAX</td>
<td>21–35</td>
</tr>
<tr>
<td>Diameter distal RVOT (mm)</td>
<td>PLAX</td>
<td>17–27</td>
</tr>
<tr>
<td>End-diastolic area (cm²)</td>
<td>4-chamber</td>
<td>11–28</td>
</tr>
<tr>
<td>End-systolic area (cm²)</td>
<td>4-chamber</td>
<td>5–15</td>
</tr>
<tr>
<td>Wall thickness (mm)</td>
<td>PLAX or subcostal</td>
<td>3–5</td>
</tr>
</tbody>
</table>

**Function**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>View</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fractional area change (%)</td>
<td>4-chamber</td>
<td>&gt;35</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>3D volumetric</td>
<td>&gt;45</td>
</tr>
<tr>
<td>TAPSE (mm)</td>
<td>4-chamber</td>
<td>≥17</td>
</tr>
<tr>
<td>S’ (cm/s)</td>
<td>4-chamber</td>
<td>&gt;10</td>
</tr>
</tbody>
</table>

PLAX, parasternal long axis; TAPSE, tricuspid annular systolic plane excursion.
FIGURE 7.59. In this patient, recurrent pulmonary emboli resulted in right ventricular enlargement and pulmonary hypertension. The increase in right ventricular size is apparent in the parasternal long-axis (A) and four-chamber (B) views. C: Doppler recording of tricuspid regurgitation velocity confirms significant pulmonary hypertension.
FIGURE 7.60. Three-dimensional echocardiography is well suited for quantitative evaluation of right ventricular volume and function. Using multiple planes, planimetry of right ventricular area is performed at end-diastole (left) and end-systole (right). From these areas, a more accurate assessment of chamber...
volume, stroke volume and ejection fraction is possible. EDV, end-diastolic volume; ESV, end-systolic volume; EF, ejection fraction; SV, stroke volume.

**FIGURE 7.61.** The motion of the lateral portion of the tricuspid annulus can be recorded with M-mode scanning from the apex. The systolic excursion of the plane (2.4 cm), referred to as TAPSE, is indicated by the *arrow*.
More recently, there has been considerable interest in using longitudinal strain as a quantitative approach to global right ventricular function. Similar to the left ventricle, a strain map is generated using the apical four-chamber view (Fig. 7.63). The magnitude and homogeneity of contractility can then be assessed. Preliminary studies suggest that right ventricular longitudinal strain may be a sensitive and quantifiable marker for a variety of pathologies that affect the right heart, such as pulmonary hypertension, pulmonary embolus, and myocardial infarction. However, the challenges of this approach are considerable. For example, the fact that the right ventricular free wall is thinner than the left ventricle creates technical difficulties in ensuring accurate and reproducible values. More work is needed before this approach to right ventricular systolic function is standardized.

A subjective assessment of right ventricular contractility can be made from multiple views. Abnormal right ventricular wall motion occurs in several disease states, including inferior myocardial infarction, pulmonary hypertension, and arrhythmogenic right ventricular cardiomyopathy (ARVC).
Figure 7.64 is an example of right ventricular dysfunction due to acute pulmonary embolus. Note the typical pattern of abnormal wall motion that involves the mid portion of the right ventricular free wall but spares the apex (McConnell sign). The patient’s heart rate at the time of this study was 120/min. In Figure 7.65, right ventricular enlargement and dysfunction are apparent in the setting of acute pulmonary embolus. The patient underwent emergent surgical thrombectomy. On follow-up study, improvement in both size and function of the right ventricle are demonstrated.

Abnormal right ventricular wall motion can also occur as a complication of inferior myocardial infarction (Fig. 7.66). As with the left ventricle, regional wall motion can be graded for the extent and severity of dysfunction. Both the free wall and the interventricular septum should be evaluated for thickening and endocardial excursion. By assessing regional right ventricular wall motion, a qualitative evaluation of overall right ventricular systolic function can be made. A more quantitative approach involves determination of right ventricular volume at end-diastole and end-systole. From these two volume measurements, the ejection fraction can be derived.

Right Ventricular Overload

Echocardiographic findings characteristic of both right ventricular volume and pressure overload have been described. Pressure overload of the right ventricle results in hypertrophy of both the free wall and the interventricular septum. This is often associated with an increase in the trabeculations of the right ventricular walls. By causing septal hypertrophy that is out of proportion to posterior left ventricular free wall hypertrophy, this combination of findings can be misinterpreted as evidence of asymmetric septal hypertrophy, suggesting hypertrophic cardiomyopathy. Because the right ventricle is trabeculated, measurement of right ventricular free wall thickness can be difficult and all available views should be used. End-diastolic wall thickness is best obtained from parasternal long-axis or subcostal views, where the ultrasound beam is aligned perpendicular to the wall and adequate endocardial and epicardial definition is achieved. An effort should be made to exclude epicardial fat and endocardial trabeculations from this measurement (Fig. 7.67). In adults, the normal right ventricular wall thickness has been reported to be $3.4 \pm 0.8$ mm and a value greater than 5 mm
is considered abnormal. A rough correlation exists between the degree of right ventricular hypertrophy and the severity of pulmonary hypertension, although this relationship has obvious limitations (Fig. 7.68).

**FIGURE 7.63.** Similar to the left ventricle, longitudinal strain of the right ventricular free wall can be assessed with speckle tracking technology. Regional variation in strain values and peak systolic strain can be recorded and displayed. See text for details.
FIGURE 7.64. A large acute pulmonary embolus results in right ventricular systolic dysfunction. In A, dilation of the right atrium and ventricle are demonstrated. In B and C, diastolic and systolic frames from the apical four-chamber view are provided. In real time, there is akinesia of the right ventricular free wall with sparing of the apex, the so-called McConnell sign.
FIGURE 7.65. The acute ventricular dysfunction that results from a massive pulmonary embolus can resolve with appropriate therapy. In A, the right ventricle is dilated and severely hypokinetic in the setting of an acute pulmonary embolus. Following surgical thrombectomy, a follow-up study is performed 6 days later (B). Significant improvement in both right ventricular size and function are apparent.

Right ventricular pressure overload also results in distortion of the shape and motion of the interventricular septum. “Flattening” of the interventricular septum in systole is the result of an abnormal pressure gradient between the left and the right ventricles (Fig. 7.69). In the normal heart, the round shape of the left ventricle is maintained throughout the cardiac cycle, a reflection of the higher pressure within the left ventricular cavity (and the instantaneous
transseptal pressure gradient). When right ventricular pressure is increased, this normal septal curvature is altered and the septum appears flattened and displaced toward the left ventricle. The greater the increase in right ventricular systolic pressure (RVSP), the greater the shift in septal position toward the left ventricular cavity. A characteristic feature of right ventricular pressure overload is the persistence of this septal distortion throughout the cardiac cycle, that is, in both systole and diastole. As is discussed below, this is in contrast to right ventricular volume overload, which leads to septal flattening predominantly during diastole.

![FIGURE 7.66. A segmental wall motion abnormality of the right ventricular free wall (arrows) is the result of a right ventricular infarction, complicating an acute inferior myocardial infarction.](image)

Doppler imaging is very useful to assess right ventricular pressure overload. Both pulmonary valve flow and tricuspid regurgitation velocity should be evaluated (Fig. 7.70). In normal individuals, pulmonary flow has a
symmetric contour with a peak velocity occurring in mid systole. As pulmonary pressure increases, peak velocity occurs earlier in systole and late systolic notching is often present (Fig. 7.70, lower panels). The acceleration time (time from onset to peak flow velocity) can be measured and provides a rough estimate of the degree of increase in pulmonary artery pressure. The shorter the acceleration time, the higher the pulmonary artery pressure.

A more direct measure of right ventricular pressure is possible by quantifying the tricuspid regurgitation jet velocity. Using the Bernoulli equation to measure the systolic gradient between the right ventricle and the atrium, RVSP is then determined from the following equation:

![Figure 7.67](image)

**FIGURE 7.67.** The assessment of right ventricular hypertrophy can be hampered by the presence of muscular trabeculations (arrows) along the right ventricular free wall. When measuring wall thickness, such muscle bundles should be avoided.
FIGURE 7.68. From a patient with pulmonary hypertension, the apical four-chamber view (A) demonstrates a dilated right heart with evidence of right ventricular hypertrophy (arrows). Using the tricuspid regurgitation velocity (B), the right ventricular systolic pressure is estimated to be 75 mm Hg.

\[
RVSP = 4(\text{TR}_\text{velocity})^2 + P_{RA}
\]  

[Eq. 7.4]

where \( \text{TR}_\text{velocity} \) is the maximal velocity of the tricuspid regurgitation jet (in meters per second) and \( P_{RA} \) is an estimate of right atrial pressure. When measuring peak TR velocity, care should be taken to identify the maximal modal frequency of the jet, not the diffuse, ill-defined frequencies that are often recorded when the Doppler gain is set too high. Because RVSP and pulmonary artery systolic pressure are similar (in the absence of pulmonary stenosis), this approach provides a simple and accurate means of quantifying the presence and severity of pulmonary hypertension. This topic is also covered in Chapters 8 and 12.

Pulmonary artery diastolic pressure can be estimated using a similar approach applied to the pulmonary regurgitation flow. In this case, the flow velocity of the regurgitant jet at end-diastole is used in the Bernoulli equation to quantify the pulmonary artery-to-right ventricular gradient. In normal individuals, pulmonary artery diastolic pressure exceeds right ventricular diastolic pressure by only a few millimeters of mercury, so the regurgitant jet velocity is low. With pulmonary hypertension, pulmonary artery diastolic pressure increases disproportionately, creating a higher pressure gradient and,
hence, an increased end-diastolic regurgitant velocity (Fig. 7.71). Thus, in patients with significant pulmonary hypertension, pulmonary regurgitant velocity at end-diastole is often higher than 2 m/s. These concepts are illustrated in Figure 7.72. In this patient with severe pulmonary hypertension, the right ventricle is dilated and hypokinetic, with septal flattening evident in the short-axis view. Doppler imaging reveals increased tricuspid regurgitation velocity (RVSP = 105 mm Hg). Elevated pulmonary regurgitation velocity (>2 m/s) is consistent with increased pulmonary artery diastolic pressure.

FIGURE 7.69. From a young woman with primary pulmonary hypertension, diastolic (A) and systolic (B) short-axis frames show systolic flattening due to elevated right ventricular systolic pressure. During diastole (A), partial restoration of the normal rounded shape of the chamber is noted. Also note the extensive trabeculations within the right ventricular cavity.
Right ventricular volume overload typically produces dilation of the right ventricle. In normal subjects, viewed from the apical four-chamber view, right ventricular diastolic area is approximately two-thirds that of the left ventricle. A subjective criterion for right ventricular dilation is a right ventricular diastolic area that appears equal to or greater than that of the left ventricle (Fig. 7.73). Volume overload of the right ventricle also affects septal motion. During diastole, the increase in right ventricular volume displaces the interventricular septum toward the left ventricular cavity, resulting in flattening of the septum (Fig. 7.74). The normal crescent shape of the right ventricle is replaced by a more spherical appearance. Such abnormalities can be appreciated using both M-mode and two-dimensional...
imaging techniques. In contrast to right ventricular pressure overload, volume overload of the right ventricle results in septal displacement only during diastole. During systole, because the normal transseptal pressure gradient is maintained, normal septal shape and position are also maintained. Thus, the degree of septal flattening during systole and diastole can be useful to distinguish volume from pressure overload. Patients with pure right ventricular volume overload have septal flattening confined to diastole. Patients with right ventricular pressure overload maintain septal flattening throughout the cardiac cycle. The degree of septal flattening also roughly correlates with the severity of pulmonary hypertension.

**FIGURE 7.71.** From an elderly patient with pulmonary hypertension, Doppler recordings of the tricuspid regurgitation jet (A) and pulmonary regurgitation jet (B) are shown. From the TR jet, a 54 mm Hg systolic gradient between the right ventricle and atrium is demonstrated, suggesting a RVSP of approximately 64 mm Hg. In B, the *arrow* indicates the end-diastolic velocity (*arrow*) of the regurgitant jet is 2.4 m/s, consistent with significantly elevated pulmonary artery diastolic pressure.
FIGURE 7.72. An example of right ventricular and right atrial dilation is shown from the parasternal short-axis (A) and the apical four-chamber (B) views. C: Pulsed Doppler imaging records pulmonary regurgitation in the right ventricular outflow tract. The end-diastolic velocity is elevated (arrows). D: High-velocity tricuspid regurgitation is shown and right ventricular systolic pressure is estimated to be 105 mm Hg, assuming a right atrial pressure of 15 mm Hg. See text for details.
FIGURE 7.73. A qualitative assessment of right ventricular size and function is routinely performed as part of a complete echocardiographic study. In this example, the four-chamber view shows dilation of the right atrium and right ventricle with severe right ventricular hypokinesis.

**Right Ventricular Cardiomyopathy**

ARVC is a rare but important condition in which the normal right ventricular free wall myocardium is replaced by adipose and/or collagen-containing tissue. ARVC has a wide range of clinical manifestations, but malignant ventricular arrhythmias and sudden death can occur. Because of the spectrum of severity and the challenges in establishing a firm diagnosis, rigorous diagnostic criteria have been established based on six categories of findings. Among these, functional/structural abnormalities can be assessed with either echocardiography, MRI, or right ventricular angiography. A “major criterion” for diagnosis of ARVC based on echocardiography requires the presence of regional right ventricular free wall akinesis/dyskinesis or aneurysm
formation, plus one or more of the following: (1) parasternal long-axis RVOT diameter ≥32 mm; (2) parasternal short-axis RVOT diameter ≥36 mm; or (3) a right ventricular fractional area change ≤33%. **Figure 7.75** is an example of an inferoapical aneurysm in a patient with ARVC. Also note the relative brightness of the right ventricular free wall, possibly indicating fatty tissue within the myocardium. MRI, by virtue of its three-dimensional capabilities and its ability to identify both fatty infiltration and scar, has advantages over echocardiography for assessment of the structural abnormalities that occur in patients with ARVC. An example is provided in **Figure 7.76**. An additional example of ARVC is provided in **Figure 7.77** from a patient with extensive involvement of the entire right ventricular free wall.

**FIGURE 7.74.** From a patient with longstanding secondary pulmonary hypertension, right ventricular enlargement results in systolic displacement of the septum toward the left ventricle and reversal of the normal septal curvature (A). In panels B and C, the short-axis view again illustrates the dilated right ventricle.
There is normal rounded left ventricular shape during diastole, but septal flattening in systole, due to right ventricular pressure overload.

**FIGURE 7.75.** From a patient with arrhythmogenic right ventricular cardiomyopathy, apical views demonstrate chamber enlargement with aneurysmal dilation of the right ventricular apex.
FIGURE 7.76. A cardiac MRI with images from the four-chamber (A) and short-axis (B) views shows right ventricular enlargement, hypokinesis, and aneurysm formation of right ventricular free wall.
FIGURE 7.77. Extensive right ventricular involvement in a patient with arrhythmogenic right ventricular dysplasia is shown. A: The apical four-chamber view demonstrates dilation of the right ventricle and hypokinesis of the right ventricular free wall (arrows). B: A subcostal view reveals segmental right ventricular dysfunction and aneurysmal dilation near the apex (arrows).

Suggested Readings

GENERAL CONCEPTS

ATRIAL SEPTUM AND SOURCE OF EMBOLUS
Saric M, Armour AC, Armaout MS, et al. Guidelines for the use of echocardiography in the evaluation


**HEMODYNAMICS**


**THE RIGHT VENTRICLE**


**THE LEFT ATRIUM**


Maddukuri PV, Vieira ML, DeCastro S, et al. What is the best approach for the assessment of left atrial


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Since its inception, one of the primary goals of echocardiography has been to provide hemodynamic information. This was initially accomplished using M-mode and later two-dimensional imaging, which allowed measurement of dimensions that could be translated into volumetric data. The development of Doppler echocardiography now provides a more direct and quantitative technique from which to derive hemodynamic information. Currently, Doppler imaging, combined with two-dimensional imaging, is the preferred method for the noninvasive measurement of hemodynamics and, in many situations, has supplanted cardiac catheterization for this purpose. The accuracy of the Doppler technique for measuring blood velocity has been validated in numerous ways. Through its ability to quantify blood flow, measure pressure gradients, and estimate intracardiac pressures, the utility of Doppler-derived hemodynamic data is now well established.

**USE OF M-MODE AND TWO-DIMENSIONAL ECHOCARDIOGRAPHY**

Since the early days of ultrasound, investigators have attempted to extract hemodynamic data from echocardiograms. Such approaches were indirect and qualitative, generally relying on the fact that physiologic changes in blood flow would have predictable effects on the motion of the walls and valves of the heart. One of the earliest applications arose from the recognition that right ventricular pressure and volume overload caused predictable changes in the motion of the interventricular septum. Unfortunately, little quantitative information could be derived from this observation. Thus, once Doppler techniques became available, a more direct quantitative measure of
right ventricular pressure was possible, thereby supplanting these more indirect approaches. A more relevant observation involved the early closure of the mitral valve that occurred in patients with acute, severe aortic regurgitation (Fig. 8.1). Here, the high temporal resolution of the M-mode technique provided a unique approach for timing valvular events. Premature closure of the mitral valve indicated rapidly increasing left ventricular diastolic pressure and became a reliable, if indirect, marker of hemodynamically significant aortic regurgitation before the availability of more direct noninvasive techniques.

A similar example is the B bump of mitral valve closure. This is a particular motion of the mitral valve that occurs in late diastole as the valve drifts shut with increasing left ventricular pressure (Fig. 8.2). The normal rate of mitral valve closure after atrial systole is smooth and of brief duration. In patients with elevated left ventricular diastolic pressure, the associated increase in left atrial pressure results in an abnormal pattern of mitral valve closure. The onset of mitral valve closure is premature, and mitral valve closure is interrupted because the A point occurs earlier than usual, resulting in a notch between the A point and the C point. The prolongation of the closing phase of the mitral valve has been termed the B bump and has been associated with increased left ventricular end-diastolic (and left atrial) pressure (Fig. 8.3). Efforts to quantify left ventricular diastolic pressure using this finding have been unreliable. Although the sensitivity of the finding has been debated, the presence of a B bump is consistently associated with a left ventricular diastolic pressure at the time of atrial contraction of at least 20 mm Hg. The application of Doppler techniques to the study of left atrial pressure eventually overshadowed the importance of this finding.
FIGURE 8.1. A mitral valve M-mode echocardiogram from a patient with acute aortic regurgitation. Note partial valve closure (C') in mid diastole, significantly earlier than normal. The valve does not reopen with atrial systole and then closes completely with the onset of ventricular contraction (C). Fine fluttering (FL) of the mitral valve is due to the aortic regurgitant jet.
FIGURE 8.2. A mitral valve echocardiogram demonstrating a B bump (arrows). See text for details. PW, posterior left ventricular wall.
FIGURE 8.3. A schematic demonstrates how the mitral valve echocardiogram reflects changes in left ventricular diastolic pressure. The normal relationship between mitral leaflet motion and intracardiac pressure changes is shown on the left. The genesis of the B bump reflects elevated late diastolic left atrial pressure. See text for details.

Other M-mode echocardiographic signs of altered hemodynamics also have stood the test of time. Systolic anterior motion of the mitral valve is an important finding in patients with hypertrophic cardiomyopathy and may indicate dynamic outflow tract obstruction. This is demonstrated using either M-mode or two-dimensional techniques. In these patients, partial closure of the aortic valve during mid and late systole, as seen on M-mode echocardiography, is a reliable indicator of significant outflow tract obstruction. Again, however, quantification of the gradient is not possible. One of the most useful echocardiographic indicators of hemodynamic significance is the early diastolic collapse of the right ventricular free wall that occurs when intrapericardial pressure increases in the clinical setting of tamponade (Fig. 8.4). This is discussed in detail in Chapter 9.
A partial listing of M-mode and two-dimensional echocardiographic findings indicating abnormal hemodynamics is provided in Table 8.1. Although most of these findings have been replaced by more quantitative and direct measurements using the Doppler techniques, they continue to provide useful confirmatory evidence in selected patients.

**QUANTIFYING BLOOD FLOW**

Doppler echocardiography is able to measure blood flow through its ability to quantify blood velocity. We know that the rate of flow through an orifice is equal to the product of flow velocity and cross-sectional area. Because cross-sectional area can be measured with M-mode or two-dimensional imaging and flow velocity can be determined directly with Doppler imaging, the technique provides a noninvasive measure of flow. If flow were constant (i.e., had a fixed velocity), it would be a simple matter to determine velocity at any point in time and solve the equation accordingly. In the cardiovascular system, however, flow is pulsatile and therefore individual velocities during the ejection phase must be sampled and then integrated to measure flow volume. This sum of velocities is called the time velocity integral (TVI) and is equal to the area enclosed by the Doppler velocity profile during one ejection period. This essential concept is illustrated in Figure 8.5. Integrating the area under the velocity curve is simply measuring the velocities at each point in time and summing all these velocities. It should be noted that when velocity is integrated over time, the units that result from this operation are a measure of distance (in centimeters), hence the term stroke distance, which is the linear distance that the blood travels during one flow period. When TVI and the corresponding cross-sectional area (in centimeters squared) are measured at the same point, such as through one of the four cardiac valves, their product equals stroke volume (in centimeters cubed or milliliters), which is the volume of blood ejected by the heart with each contraction (assuming no valvular regurgitation or cardiac shunt).
FIGURE 8.4. An M-mode echocardiogram from a patient with pericardial tamponade. The arrows indicate early diastolic collapse of the right ventricular free wall. The echo-free space above the right ventricular free wall represents pericardial fluid, which can also be seen posterior to the left ventricle. PE, pericardial effusion.

### Table 8.1

<table>
<thead>
<tr>
<th>Finding</th>
<th>Hemodynamic Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>M-mode</strong></td>
<td></td>
</tr>
<tr>
<td>Early closure of the mitral valve</td>
<td>Acute, severe aortic regurgitation</td>
</tr>
<tr>
<td>Increased mitral valve E point-septal separation</td>
<td>Reduced LV ejection fraction</td>
</tr>
<tr>
<td>Delayed closure of the mitral valve (B bump)</td>
<td>Elevated LV end-diastolic</td>
</tr>
<tr>
<td>Condition</td>
<td>Diagnosis</td>
</tr>
<tr>
<td>--------------------------------------------------------------------------</td>
<td>------------------------------------------</td>
</tr>
<tr>
<td>RV free-wall early diastolic collapse</td>
<td>Pericardial tamponade</td>
</tr>
<tr>
<td>Mid systolic notching of the aortic valve</td>
<td>Dynamic subaortic outflow tract obstruction</td>
</tr>
<tr>
<td>Diastolic mitral valve fluttering</td>
<td>Aortic regurgitation</td>
</tr>
<tr>
<td>Mid systolic notching of the pulmonary valve</td>
<td>Pulmonary hypertension</td>
</tr>
<tr>
<td>Rounding of the opening/closing points of a disc-type prosthetic valve</td>
<td>Mechanical restriction to disc motion</td>
</tr>
<tr>
<td>Systolic anterior motion of the mitral valve</td>
<td>Dynamic subaortic outflow tract obstruction</td>
</tr>
<tr>
<td>Early systolic downward motion (beaking) of the IVS</td>
<td>LBBB</td>
</tr>
<tr>
<td>Gradual closure of the aortic valve</td>
<td>Reduced left ventricular stroke volume</td>
</tr>
<tr>
<td>Absent pulmonary valve A wave</td>
<td>Pulmonary hypertension</td>
</tr>
<tr>
<td>Two dimensional</td>
<td></td>
</tr>
<tr>
<td>Diastolic flattening of the IVS</td>
<td>RV volume overload</td>
</tr>
<tr>
<td>Systolic flattening of the IVS</td>
<td>RV pressure overload (elevated RVSP)</td>
</tr>
<tr>
<td>Dilated IVC with abnormal respiratory variation</td>
<td>Elevated RA pressure</td>
</tr>
<tr>
<td>Exaggerated IVS bounce, with respiratory variation</td>
<td>Constriction</td>
</tr>
<tr>
<td>LBBB, left bundle branch block; RVSP, right ventricular systolic pressure.</td>
<td></td>
</tr>
</tbody>
</table>
These principles are illustrated in Figure 8.6, which demonstrates how these concepts can be applied to aortic flow to measure stroke volume. Recall from the Doppler equation the importance of the angle θ, that is, the angle between the ultrasound beam and blood flow direction. Because the cosine function varies between 0 and 1 and appears in the numerator of the Doppler equation, errors in θ will have a predictable effect on measured velocities. For example, if θ is between 0 and 20 degrees, the cosine of θ will range between 1.0 and 0.92, leading to a slight underestimation of true velocity. As θ increases to more than 20 degrees, the cosine decreases rapidly and the degree of velocity underestimation increases quickly. Hence, aligning the ultrasound beam as close as possible to the direction of flow is critical if true velocity is to be measured. Equally important, misalignment between the ultrasound beam and flow can result only in underestimation of velocity, never overestimation.

Flow rate = flow velocity x CSA
Flow velocity varies from $t_0$ to $t_z$
Sum of all velocities = TVI
TVI = $\sum V_{0\rightarrow z}$
Stroke volume = TVI x CSA

FIGURE 8.5. A schematic demonstrates the concept of flow quantification using the Doppler technique. Doppler records instantaneous velocity throughout the cardiac cycle. The area under the Doppler velocity curve represents the time velocity integral (TVI). This is the sum of all the individual instantaneous velocities throughout the ejection period. See text for details. CSA, cross-sectional area.
FIGURE 8.6. The method for quantifying stroke volume. Two measurements are required: area and time velocity integral. See text for details. $D$, diameter; SEP, systolic ejection period; TVI, time velocity integral.

Area = $\pi r^2$
Area = $\pi (D/2)^2$
Area = $D^2 \times 0.785$

TVI = Area under curve

Stroke volume = Area $\times$ TVI
Another factor that will affect the accuracy of the Doppler equation is the pattern of blood flow in which velocity is being measured. Normal flow in the heart and great vessels is laminar, meaning that the fluid is traveling at approximately the same velocity and in the same general direction. If a sample volume is placed within such a flow pattern, the Doppler will record a clean signal of uniform velocity. Flow becomes increasingly disturbed or turbulent (i.e., less laminar) as the velocity increases or the cross-sectional area changes (Fig. 8.7A). Viscosity also affects the flow profile. At the edge of the flow pattern, near the vessel wall, flow tends to be slower and more turbulent. The highest velocities and most laminar flow generally occur at the center of the profile. This spatial distribution of velocities across the three-dimensional flow is called the flow velocity profile. In a large, straight vessel, with laminar flow, it tends to be flat (Fig. 8.7B), whereas in smaller curved vessels, the profile has a parabolic shape. Velocity will be higher at the center.
and lower at the margins. Flow patterns through curved vessels, such as the aortic arch, are more complex. Here the distribution of velocities depends on the size of the vessel, the flow profile entering the curve, and the presence and location of branch vessels. If the sample volume is placed within such a flow pattern, the recorded velocity will vary, depending on the exact location.

Fortunately, flow passing through a normal heart valve or the proximal great vessels tends to be laminar with a flat profile and is therefore suitable for quantitative analysis. Because it is easier to determine the average flow velocity with a flat versus a parabolic blood profile, it is not surprising that efforts to measure blood flow attempt to use larger orifices and flow that are close to the origin of vessels. Also note that physiologic blood flow is never perfectly uniform. That is, at any point in time, a distribution of velocities occurs, resulting in a broadening of the Doppler signal. The greater the range of velocities is at any point in time, the broader is the Doppler signal. The darker line through the center of the distribution represents the modal frequency, that is, the velocity at which the largest number of blood cells are traveling (Fig. 8.8). Theoretically, this is the velocity that should be used to determine the TVI. In practice, however, it is common to trace the outer edge of the densest portion of the envelope, and studies have indicated that both techniques provide a reasonably accurate measurement of blood flow. Multiple cycles (usually three to five) should be traced and averaged to minimize error. In patients with atrial fibrillation, between 5 and 10 beats should be analyzed.
An important potential source of error in the blood flow measurement is the determination of cross-sectional area. It is essential to remember that cross-sectional area must be measured at the same point in space where the Doppler signal is sampled. For example, if blood flow is measured through the aortic valve, both the Doppler signal and the cross-sectional area must be measured at the same level. If the Doppler sample volume is placed at the level of the aortic annulus, then the cross-sectional area of the aortic annulus must be determined. The cross-sectional area can be measured in systole using either M-mode or two-dimensional imaging. In Figure 8.9, three slightly different measurements of the outflow tract diameter are obtained. In most cases, the largest dimension should be used because it most likely corresponds to the true diameter. Another approach to this problem would be to directly measure the cross-sectional area by planimetry of a short-axis image of the orifice. CT angiography, now routinely performed to determine outflow tract size prior to TAVR, has shown that many patients have
noncircular outflow tracts (Fig. 8.10). In such patients, a single linear dimension will never provide an accurate measure of cross-sectional area. Thus, when available, CT or three-dimensional echocardiography should be used for this determination. In routine practice, however, it is still reasonable to measure the diameter of the orifice, assume a circular shape, and calculate area using the formula

![Figure 8.9](image)

**FIGURE 8.9.** To measure the cross-sectional area of the left ventricular outflow tract, the diameter ($D$) must be measured carefully. The three examples demonstrate three different values for $D$ obtained from the same patient. In most cases, the correct dimension is the largest, indicating the true diameter.
“Small” Area\textsubscript{OT} → “Small” AV area

![CT scan of the left ventricular outflow tract from a patient undergoing transcatheter aortic valve replacement. As is often the case, this outflow tract is oval rather than round. If the area were determined by measuring the linear height of the outflow tract from the parasternal long-axis view (yellow arrow), and assuming a circular shape, the area would have been reported as 3.8 cm\textsuperscript{2}. Using CT, planimetry of the true shape yields an area of 4.9 cm\textsuperscript{2}. Underestimation of outflow tract area results in underestimation of aortic valve area using the continuity equation.

\begin{equation}
A = pr^2 \quad \text{[Eq. 8.1]}
\end{equation}

Because \( r = \frac{1}{2}D \), and \( D \) is what is actually measured, this can be simplified and expressed as

\begin{equation}
A = 0.785 \times D^2 \quad \text{[Eq. 8.2]}
\end{equation}

Thus, the Doppler equation for stroke volume becomes
Stroke volume = 0.785 \times D^2 \times TVI \quad \text{[Eq. 8.3]}

Considering this equation, it is obvious that any error in the measurement of the diameter of the orifice is “squared” and thus contributes greatly to errors in the final determination. For this reason, particular care must be taken to ensure accurate determination of orifice diameter. Multiple measurements should be performed. In general, the largest dimension is used because it most likely represents the true diameter and smaller measurements represent tangential cuts through the circular outflow tract. The importance of accurately measuring the outflow tract diameter is illustrated in the following example. Assume that the “true” diameter is 2 cm and the TVI is 20 cm. This would yield a stroke volume of 63 mL. Underestimation of the diameter by just 10% would have the following effect on stroke volume calculation:

\[
\text{Stroke volume} = 0.785 \times (1.8 \text{ cm})^2 \times 20 = 51 \text{ mL}
\]

Thus, a 2-mm (or 10%) underestimation in diameter would lead to a 19% underestimation (51 mL instead of 63 mL) in stroke volume.

Despite these potential sources of error, the accuracy and utility of this approach for measuring blood flow in a variety of clinical situations are well established. When performed carefully, this noninvasive technique has proven to be an accurate and reproducible way to quantify blood flow within the cardiovascular system. An example of stroke volume calculation from the aortic flow measurement is provided in Figure 8.11.

**CLINICAL APPLICATION OF BLOOD FLOW MEASUREMENT**

The Doppler approach to measuring blood flow is a general formula that can be applied anywhere that blood passes through an orifice of fixed and measurable dimensions. Thus, it is possible to measure blood flow across all four valves of the heart and in the great vessels. To do so requires pulsed Doppler sampling of flow velocity at a location where cross-sectional area also can be measured. Figure 8.12 illustrates how stroke volume can be measured through each of the four valves. In the absence of valvular regurgitation or intracardiac shunt, flow through all four valves should be
equal. The diagram demonstrates how cross-sectional area and TVI vary inversely for the different valves, but the product (cross-sectional area × TVI) is equal at each location. Of course, each site presents its unique set of challenges, and in any given patient, the measurement may or may not be feasible. Accuracy and reproducibility will improve with practice. Thus, performing flow calculation on a routine basis can be expected to increase one’s confidence in the results when clinical questions arise.

\[
SV = D^2 \times 0.785 \times TVI \\
SV = 2.4^2 \times 0.785 \times 19 \\
= 86 \text{ cc}
\]

**FIGURE 8.11.** An example of stroke volume calculation. **A:** The cross-sectional area of the outflow tract (AVd) is measured. **B:** The time velocity integral of aortic flow is determined by planimetry. The calculation for stroke volume (SV) is shown. TVI, time velocity integral.
FIGURE 8.12. This schematic demonstrates the principle of conservation of mass. In the absence of valvular regurgitation or intracardiac shunts, the stroke volume through each of the four valves should be equal. See text for details.

Although flow can theoretically be measured at any site, in practice, it is customary to measure blood flow through the aortic valve. The Doppler recording is performed using either the apical five-chamber or the apical long-axis view and the sample volume is positioned at the level of the aortic annulus, approximately 3 to 5 mm proximal to the valve (Fig. 8.11). At that location, it is usual to record the closing “click” of the aortic valve at end-systole. If the opening click is present in the Doppler recording, the sample volume should be withdrawn slightly into the outflow tract. Cross-sectional area is typically measured by recording the parasternal long-axis view and determining the diameter of the aortic annulus in systole, assuming a circular shape. Because annular size does not change much over the cardiac cycle, the precise timing of the diameter measurement is not critical. However, as previously discussed, the assumption of a circular shape is not always valid. An alternative approach employs three-dimensional imaging (either echocardiography or CT) to directly planimeter cross-sectional area.

Pulmonary valve flow can be recorded using a similar approach. The sample volume is positioned at the level of the pulmonary valve, usually from
the basal short-axis view. Alternatively, especially in children, the subcostal short-axis view can be used. The cross-sectional area is measured as the diameter of the outflow tract at the level of the annulus. An accurate measurement of this diameter is often difficult in adults because of the challenges of visualizing the lateral border of the right ventricular outflow tract. It is commonly performed in children, however, to quantify right ventricular stroke volume. This can then be compared with stroke volume in the left side of the heart to assess intracardiac shunts and valvular regurgitation. This application is covered later in this section.

Quantitating stroke volume across the mitral valve creates additional challenges. Mitral flow velocity is easily recorded from apical views and consists of two phases: an early diastolic wave (E) and a second wave associated with atrial systole (A). Several studies have demonstrated that Doppler mitral velocity can be used to quantify stroke volume provided that the cross-sectional area of the mitral valve orifice can be determined. This can be obtained using a short-axis view to planimeter its cross-sectional area. Next, an M-mode or two-dimensional echocardiographic recording of the mitral valve is used to determine the mitral orifice diameter throughout diastole. From this, the mean mitral diameter is calculated and applied to the Doppler equation. A simplified and more practical approach uses the diameter of the mitral annulus as measured from the apical views as a surrogate for cross-sectional area (Fig. 8.13). The measurement should be performed from the four-chamber view in early diastole. Then, assuming a circular shape, the area is estimated by Equation 8.1, which is $A = \pi r^2$. Alternatively, a second diameter can be measured from the apical two-chamber view and a mean value for cross-sectional area can be obtained. Mitral inflow velocity is then recorded at the level of the annulus, and the TVI is determined by planimetry. The accuracy of quantifying mitral stroke volume is debatable. Recording a clean velocity profile at the annular level (compared with the mitral leaflet tips) can be challenging. It is also more difficult to accurately measure cross-sectional area at the mitral annulus compared with the aortic annulus. For all these reasons, quantifying blood flow across the mitral and tricuspid valves is more cumbersome compared with the aortic and pulmonary valves and is performed rarely in clinical practice.
**FIGURE 8.13.** An example of calculating stroke volume (SV) through the mitral valve. **A:** The cross-sectional area of the mitral annulus is determined. **B:** Flow velocity at that level is measured using pulsed Doppler imaging. See text for details. TVI, time velocity integral.

\[
SV = 2.5^2 \times 0.785 \times 15 = 74 \text{ cc}
\]

**FIGURE 8.14.** Differences in stroke volume (SV) across the aortic and mitral valves:

\[SV_M = TVI_M \times CSA_M\]
\[SV_A = TVI_A \times CSA_A\]
\[RV_A = SV_A - SV_M\]

**FIGURE 8.14.** Differences in stroke volume (SV) across the aortic and mitral
valves may reflect regurgitation at one of these sites. In this schematic from a patient with aortic regurgitation, regurgitant volume (RVA) is simply the difference between the aortic stroke volume and the mitral stroke volume. CSA, cross-sectional area; D, diameter; TVI, time velocity integral. See text for details.

This technique for determining volumetric flow has several practical applications. The noninvasive measurement of stroke volume has obvious value, both as an absolute number and as a relative change. Stroke volume is a fundamental measure of global left ventricular systolic performance and can be readily converted to cardiac output by multiplying by heart rate. In critically ill patients, relative changes in stroke volume may indicate improvement or deterioration or may reflect a response to an intervention. In this case, it is the relative change that matters. If cross-sectional area is assumed to remain constant, changes in the TVI will reflect changes in stroke volume. This has the advantage of avoiding the potential errors that can be introduced when measuring cross-sectional area. By following changes in TVI, relatively subtle alterations in cardiac performance can be tracked.

In patients with valvular regurgitation, differences in stroke volume across different valves provide a quantitative assessment of severity. This is illustrated schematically in Figure 8.14. In the absence of regurgitation, stroke volume across all four valves should be equal. In the presence of aortic regurgitation, for example, the difference between aortic flow and mitral flow represents the aortic regurgitant volume as shown in the following formula:

\[
\text{Regurgitant volume} = \text{Aortic systolic flow} - \text{Mitral diastolic flow} \quad [\text{Eq. 8.4}]
\]

Regurgitant fraction in aortic regurgitation can also be calculated as

\[
\text{Regurgitant fraction (\%)} = \frac{\text{Regurgitant volume}}{\text{Aortic outflow volume}} \times 100\% \quad [\text{Eq. 8.5}]
\]

This type of calculation can be performed for any valve of the heart (Fig. 8.15). It assumes that the valve used as the standard for flow is not regurgitant and that a similar degree of accuracy can be achieved at each location. In addition, the calculation is complicated by the presence of valve stenosis.

A final application of this principle is the quantitation of intracardiac shunts. Determining the pulmonary-to-systemic flow ratio, or \( Q_p:Q_s \), is the
principal way to quantitate the size of the shunt (Fig. 8.16). In most cases, the shunt ratio is determined by calculating pulmonary stroke volume and comparing it with aortic stroke volume. The difference equals the net shunt volume in the absence of semilunar valve stenosis or regurgitation. This approach has been used in pediatric echocardiography with success and has been validated against invasive standards.

Aortic flow:
\[ \text{CSA}_{AV} = 3.1 \, \text{cm}^2 \]
\[ \text{TVI}_{AV} = 36 \, \text{cm} \]
\[ \text{SV}_{AV} = 112 \, \text{cc} \]

Mitral flow:
\[ \text{CSA}_{MV} = 5.3 \, \text{cm}^2 \]
\[ \text{TVI}_{MV} = 13 \, \text{cm} \]
\[ \text{SV}_{MV} = 69 \, \text{cc} \]

Regurgitant volume:
\[ 112 - 69 = 43 \, \text{cc} \]

Regurgitant fraction:
\[ \frac{43}{112} = 38\% \]

**FIGURE 8.15.** An example of how regurgitant volume (RV) and regurgitant fraction (RF) can be measured. In panels A and B, aortic stroke volume (SV) is calculated from the outflow tract area and pulsed Doppler recording. In C and D, the same calculation is performed for mitral SV. In this example, the aortic SV is 112 cc, while the mitral SV is 69 cc. The difference is the regurgitant volume (43 cc). The regurgitant fraction is 38%. CSA, cross-sectional area; TVI, time velocity integral.
In the presence of an intracardiac shunt, \( Q_p/Q_s \) provides a means to quantify the magnitude of shunting. In this example from a patient with a large secundum atrial septal defect, stroke volume (SV) through the pulmonary (left) and aortic (right) valves is measured and the \( Q_p/Q_s \) is determined. TVI, time velocity integral.

\[
\begin{align*}
Q_p \text{ SV} &= D^2 \times 0.785 \times TVI \\
&= 1.6^2 \times 0.785 \times 56 \\
&= 113 \text{ cc}
\end{align*}
\]

\[
\begin{align*}
Q_s \text{ SV} &= D^2 \times 0.785 \times TVI \\
&= 1.5^2 \times 0.785 \times 25 \\
&= 44 \text{ cc}
\end{align*}
\]

\[
Q_p/Q_s = 113/44 = 2.5
\]

**FIGURE 8.16.** In the presence of an intracardiac shunt, \( Q_p/Q_s \) provides a means to quantify the magnitude of shunting. In this example from a patient with a large secundum atrial septal defect, stroke volume (SV) through the pulmonary (left) and aortic (right) valves is measured and the \( Q_p/Q_s \) is determined. TVI, time velocity integral.

In summary, calculation of volumetric flow is possible and has been validated in a variety of clinical situations. The formulas are based on sound physiologic principles and, under optimal circumstances, provide an accurate means for quantifying flow. Measurement errors can cause significant mistakes that may or may not be apparent at the time of the calculations. As a
consequence, a small and sometimes unrecognized error in measurement can lead to an unacceptable error in the final result. For example, if aortic and mitral stroke volumes are derived to calculate regurgitant volume and if each primary calculation is off by 10%, the following scenario is possible. Assume that the correct aortic stroke volume is 90 mL and the mitral stroke volume is 60 mL, yielding a regurgitant volume of 30 mL and a regurgitant fraction of 33%. If the aortic stroke volume is high by 10% (99 mL) and the mitral stroke volume is low by the same degree (54 mL), the derived regurgitant volume is now 45 mL and the regurgitant fraction is 45%, a significant difference. To minimize the likelihood of errors, it is essential to do such calculations routinely rather than just on rare occasions. Be aware of the potential sources of error and know when image quality precludes reliable measurements.

MEASURING PRESSURE GRADIENTS

One of the most important applications of the Doppler method is to measure transvalvular pressure gradients. This approach is based on Newton’s law of conservation of energy, which states that the total amount of energy within a closed system must remain constant. Thus, as applied to blood flow measurements, the flow velocity through a valve must increase as the valve area decreases. When blood is forced through a stenotic valve, its kinetic energy (which is proportional to the square of velocity) increases, whereas its potential energy must decrease proportionately. In a pulsatile system, some energy may be lost due to inertia as the blood accelerates and decelerates. In addition, a small amount of energy may be lost in the form of heat as a result of viscous friction. These relationships were described mathematically by Bernoulli and expressed as:

\[
\Delta P = \frac{1}{2} \rho (v_2^2 - v_1^2) + \rho \int \frac{dv}{dt} \times ds + R(\mu) \quad \text{[Eq. 8.6]}
\]

where \( \Delta P \) is the pressure difference across the stenosis, \( v_1 \) and \( v_2 \) are the velocities proximal and distal to the stenosis, respectively, \( \rho \) is the mass density of blood, \( R \) is viscous resistance, and \( \mu \) is viscosity (Fig. 8.17). Essentially, the first term of the equation corresponds to kinetic energy that results from acceleration through the stenosis. The second term accounts for...
the loss of energy as the blood accelerates and then decelerates. The final term represents the losses due to viscous friction, a function of blood viscosity and velocity. Fortunately, the latter two terms are negligible (under most physiologic conditions) and the Bernoulli equation can be simplified to:

$$\Delta P = 4(v_2^2 - v_1^2)$$  \[\text{Eq. 8.7}\]

Because both velocity terms are squared, if $v_2$ is significantly greater than $v_1$, $v_1$ can be eliminated to a final simplified equation that relates the pressure decrease across a discrete stenosis to the maximal velocity distal to the valve:

$$\Delta P = 4v^2$$  \[\text{Eq. 8.8}\]

where $v$ is the maximal velocity of the stenotic jet.

The simplified Bernoulli equation has been validated in numerous clinical situations and correlates well with direct invasive measures of pressure decrease. The technique has had its greatest application in measuring the severity of valve stenosis, a topic that is also covered in several other chapters. This same approach can also be used to estimate intracardiac pressures in patients with valvular regurgitation or intracardiac shunts, such as ventricular septal defects. In essence, wherever velocity can be measured across a discrete stenosis, the Bernoulli equation allows the pressure gradient between two chambers to be determined.
The accuracy of the Bernoulli equation to predict pressure gradients across stenotic valves is well established. When using the technique clinically, several potential sources of error should be considered and, whenever possible, avoided. As will be apparent, most errors are technical in nature and result in underestimation of the true pressure gradient. The most common example occurs when the ultrasound beam cannot be properly aligned relative to the direction of blood flow. As has been discussed, when the incident angle increases beyond 20 degrees, a significant error is introduced into the Doppler equation that results in underestimation of true velocity. To avoid this problem, color Doppler imaging can be used to visualize the blood flow, thereby facilitating proper alignment. The use of multiple acoustic
windows is another way to ensure that the view providing the best alignment is recorded. Two examples of this are shown in Figures 8.18 and 8.19. In Figure 8.18, three different values for tricuspid regurgitation velocity (TRV) yield three different estimates of right ventricular systolic pressure. The correct value is the highest, in this case recorded from the apical four-chamber view, which affords the best alignment with blood flow. In Figure 8.19, two examples of aortic stenosis are shown. In both cases, the severity of aortic stenosis is underestimated from the apical window but accurately assessed from the right parasternal window. Better alignment between the ultrasound beam and the stenotic jet is the explanation for the difference.

Image quality also plays a role in the accuracy of the gradient determination. The signal-to-noise ratio will affect whether the entire Doppler envelope is recorded for analysis. If part of the envelope is “missing” because of an incomplete signal, the peak velocity will be missed and underestimation will occur. Failure to record the entire Doppler envelope will invariably lead to underestimation of velocity. Proper gain setting, optimal beam alignment, and a careful and thorough search for the best image are all necessary to accurately measure pressure gradients. The application of echo contrast agents to boost the signal of the jet is another practical way to avoid underestimation. However, when contrast is used, some noise is inevitably introduced into the signal. Some adjustment of the reject settings may be necessary, and only the densest part of the Doppler contour should be traced. An example of the use of contrast to improve the Doppler signal is provided in Figure 8.20. It should be emphasized that the maximal velocity should always be sought out and used for the calculation of gradient.

In most cases, Doppler-derived pressure gradients are compared with cardiac catheterization data. When discrepancies occur, a plausible explanation is often apparent. For example, it is important to remember that Doppler measures peak instantaneous gradient, whereas catheterization data are most often reported as peak-to-peak, which is usually less. The difference between these two values is illustrated in Figure 8.21. Another potential source of discrepancy is the nonsimultaneous nature of the studies. Valve gradients are dynamic and may vary considerably as a result of changes in volume status, heart rate, blood pressure, and contractility. If the Doppler data and the catheterization data are not recorded at the same time, differences may be expected.
The simplified Bernoulli equation ignores the proximal flow velocity ($v_1$) and estimates gradient based on the distal, or jet, velocity ($v_2$). This is an acceptable simplification if $v_2$ is significantly greater than $v_1$. However, in cases in which the proximal velocity ($v_1$) is relatively high, this simplification may be inappropriate. For example, if antegrade flow is high and/or if the gradient is low, the difference between $v_1$ and $v_2$ may be relatively small and a more appropriate version of the Bernoulli equation would be:

$$\Delta P = 4(v_2^2 - v_1^2)$$  \[Eq. 8.7\]

**FIGURE 8.18.** These three recordings of tricuspid regurgitation (TR) are taken from one patient. The different panels illustrate how various values for velocity ($V_{TR}$) yield significantly different estimates of pressure gradient (PG) and hence right ventricular systolic pressure. The correct value is usually the highest velocity value, in this case recorded from a modified apical four-chamber view. $V_{TR}$, peak tricuspid regurgitation jet velocity.
FIGURE 8.19. Two patients with aortic stenosis are included. In both cases, different values for aortic stenosis jet velocity are obtained, yielding different measures of peak gradient. In patient A, the apical view underestimates the true velocity, which is optimally recorded from the right parasternal window. In patient B, the apical window again underestimates true velocity. In this case, the peak gradient was best recorded from the suprasternal notch.
FIGURE 8.20. Administration of contrast can be used to enhance the Doppler signal and improve determination of true velocity. On the left, tricuspid regurgitation is incompletely recorded in a baseline study. After injection of agitated saline through a peripheral vein, the tricuspid regurgitation signal is enhanced and the peak velocity more accurately determined.

A potential source of error in some clinical situations involves the concept of pressure recovery. When blood accelerates through a stenotic orifice, potential energy is converted to kinetic energy and is associated with an increase in velocity and a decrease in pressure. Both the pressure difference and velocity are greatest just distal to the orifice in the vena contracta. This is the value recorded with Doppler and represents the maximal gradient across the stenosis. As the blood exits the orifice, the jet expands and decelerates. Some of the kinetic energy is converted back into potential energy, resulting in a rise in pressure downstream from the orifice. This increase in pressure is referred to as “pressure recovery” (Fig. 8.22). If a measuring catheter is positioned sufficiently far downstream that significant pressure recovery has occurred, it will measure a smaller net gradient compared with Doppler, which measures the maximal gradient at the vena contracta. In such cases, Doppler imaging will overestimate the catheterization-derived gradient, resulting in a discrepancy, although neither represents an actual error in measurement.

In most cases, pressure recovery is negligible and the gradients determined by the various methods yield similar results. In recent years, pressure recovery has been recognized as an important phenomenon and a potential explanation for discrepant results. The degree of pressure recovery is primarily determined by the anatomy and severity of the stenosis and can be quantified. Pressure recovery appears to increase with increasing jet velocity.
From a practical standpoint, one of the main factors that contributes to this phenomenon is the diameter of the ascending aorta. The smaller the aorta, the greater the likelihood of pressure recovery. If the ascending aortic diameter is less than 30 mm, one should anticipate a significant difference between Doppler and catheter-derived gradients. This may be especially relevant in congenital aortic stenosis. However, since most adults with aortic stenosis have an aortic diameter greater than 30 mm, the magnitude of pressure recovery is small in these patients. Pressure recovery has also been demonstrated in some prosthetic valves and may also occur in tapered stenoses, such as supravalvar aortic stenosis and coarctation.

FIGURE 8.21. This schematic demonstrates the relationship between aortic and left ventricular pressure in the setting of aortic stenosis. The differences between peak instantaneous, peak-to-peak, and mean gradients are demonstrated.

It is apparent that underestimation of the true gradient by the Doppler technique is more common than overestimation. A situation in which overestimation may occur is in the setting of combined aortic stenosis and mitral regurgitation. Because of the proximity of the two jets, as well as their similar timing and appearance, a misplaced Doppler beam may inadvertently record mitral regurgitation instead of aortic stenosis. Because the velocity of mitral regurgitation is invariably high, this can lead to overestimation. To avoid this problem, color flow imaging can be used to ensure spatial
orientation. By gradually moving the Doppler beam back and forth from the
left atrium to the aortic valve, both jets can be sequentially recorded. This
increases the confidence of the interpreter to distinguish one from the other.
In addition, the velocity information must “make sense.” When anatomic data
are incompatible with Doppler data, an explanation must be sought. For
example, mitral regurgitation is invariably high velocity, often 5 to 6 m/s.
The jet of aortic stenosis is typically less than that, depending, of course, on
the severity. If, by all other criteria, aortic stenosis appears mild or moderate,
but the Doppler velocity is 6 m/s, the likelihood that the jet represents mitral
regurgitation must be considered. It is also helpful to remember that the
mitral regurgitation jet will be of greater duration than the systolic ejection
period. In Figure 8.23, note the relationship between the onset of flow and the
QRS complex; mitral regurgitation begins much earlier than aortic outflow.
The onset of mitral regurgitation occurs at the time of mitral valve closure,
whereas the jet of aortic stenosis does not begin until after isovolumic
contraction. By carefully examining these time intervals within the Doppler
signals, the two jets can often be differentiated.

**FIGURE 8.22.** The concept of pressure recovery is illustrated in this schematic.
A: When the aorta is dilated, pressure recovery is unlikely. In the absence of pressure recovery, the peak gradient recorded within the vena contracta is not significantly greater than a (net) gradient recorded further downstream from the valve. In such cases, Doppler and catheter-derived gradients are similar. B: In the setting of a nondilated, or narrow, aorta, a greater degree of pressure recovery may be expected. In this case, sampling downstream will yield a lower gradient compared to that obtained within the vena contracta. In such cases, the Doppler gradient will be significantly higher than the catheter-derived gradient. See text for details.

**FIGURE 8.23.** This illustration demonstrates how the high-velocity systolic jets of aortic stenosis (A) and mitral regurgitation (B) can be differentiated. Mitral regurgitation begins earlier, during isovolumic contraction, and persists later compared with the aortic stenosis jet. See text for details. AS, aortic stenosis; MR, mitral regurgitation.

**APPLICATIONS OF THE BERNOULLI EQUATION**

A list of clinical applications of the Bernoulli equation is provided in Table 8.2. The most common use of the Bernoulli equation is to quantify the severity of valve stenosis. An example of this application is shown in Figure 8.24. By planimetry of the envelope of the stenotic jet, both maximal and mean gradients are obtained. To determine the mean gradient, the instantaneous gradients are measured at multiple points throughout the flow and their sum is divided by the duration of flow. The shape or contour of the Doppler signal also contains relevant information. Two examples of a late-peaking left ventricular outflow tract gradient are shown in Figure 8.25. This pattern is typical of dynamic obstruction as occurs with hypertrophic cardiomyopathy. In contrast, valvular stenosis is characterized by rapid acceleration of blood flow in early systole with an earlier peak velocity.
# Table 8.2 CLINICAL APPLICATIONS OF THE BERNOULLI EQUATION

<table>
<thead>
<tr>
<th>Application</th>
<th>Clinical Utility</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak velocity through a stenotic valve</td>
<td>Aortic stenosis maximal gradient</td>
</tr>
<tr>
<td>TR jet velocity</td>
<td>RV systolic pressure</td>
</tr>
<tr>
<td>LV outflow tract contour and velocity</td>
<td>HOCM gradient</td>
</tr>
<tr>
<td>Peak velocity across a VSD</td>
<td>RV systolic pressure</td>
</tr>
<tr>
<td>End-diastolic velocity of PR jet</td>
<td>Pulmonary artery diastolic pressure</td>
</tr>
<tr>
<td>Velocity through a PDA</td>
<td>Pulmonary artery systolic pressure</td>
</tr>
<tr>
<td>MR contour and velocity</td>
<td>Left ventricular dP/dt</td>
</tr>
</tbody>
</table>

HOCM, hypertrophic obstructive cardiomyopathy; MR, mitral regurgitation; PDA, patent ductus arteriosus; PR, pulmonic regurgitation; TR, tricuspid regurgitation; VSD, ventricular septal defect.

Application of the Bernoulli equation to mitral stenosis has been extensively studied. Although peak mitral valve gradient, which occurs in early diastole, can be readily determined, this is of less clinical value than mean gradient. By tracing the envelope of the mitral stenosis jet, the mean diastolic gradient across the mitral valve is obtained (Fig. 8.26). In these examples, notice how the presence of an A wave in the patient with sinus rhythm affects the mean gradient. If the jet velocities are relatively low, the simplified Bernoulli equation will tend to overestimate true gradient because the difference between $v_2$ and $v_1$ is not great. Under such circumstances, use of the modified Bernoulli equation would be more appropriate (see Eq. 8.7). This equation is cumbersome when mean (rather than peak) gradient is being measured.
FIGURE 8.24. Continuous-wave Doppler can be used to record the aortic stenosis jet. By measuring the maximal velocity of the jet, the peak pressure gradient can be estimated using the Bernoulli equation. In this example, the maximal velocity ($V_{\text{max}}$) is 3.8 m/s, and the peak and mean gradients are 58 and 34 mm Hg, respectively.

FIGURE 8.25. Two examples of late-peaking left ventricular outflow tract jets. These recordings were taken from patients with hypertrophic obstructive cardiomyopathy.
FIGURE 8.26. Three examples of mitral stenosis. A: Images from patients in atrial fibrillation; an online computer system is used to planimeter the mitral jet, thereby providing a measure of the mean pressure gradient. B: The same technique is used in a patient in sinus rhythm.
FIGURE 8.27. Two examples of ventricular septal defect. Continuous-wave Doppler imaging is used to record the maximal velocity through the defects. Using the Bernoulli equation, the left ventricular-to-right ventricular pressure gradients (PG) can be calculated. If blood pressure (BP) is known, an estimate of right ventricular systolic pressure (RVSP) can be derived as shown. A: A 5 m/s ventricular septal defect jet predicts an RVSP of 30 mm Hg. B: A much lower ventricular septal defect jet velocity (2.4 m/s) is consistent with significant pulmonary hypertension.

Because the Bernoulli equation provides information on instantaneous pressure gradient, it has several other applications. The acceleration of blood through a ventricular septal defect in systole is a reflection of the instantaneous pressure difference between the two ventricles (Fig. 8.27). By aligning the Doppler beam parallel to the ventricular septal defect jet, the peak velocity of the shunt can be determined and used to calculate the maximal pressure difference across the ventricular septum. If left ventricular systolic pressure (LVSP) is known, right ventricular systolic pressure (RVSP) can be estimated as the difference between left ventricular pressure and maximal gradient across the defect (PGjet):

\[ LV_{SP} - PG_{jet} = RV_{SP} \approx PA_{SP} \]  \hspace{1cm} [Eq. 8.9]

In the absence of aortic stenosis, cuff-measured systolic blood pressure is an acceptable surrogate for left ventricular pressure, thereby providing a noninvasive means to estimate right ventricular systolic pressure and
pulmonary artery systolic pressure ($PA_{SP}$).

Right ventricular systolic pressure can also be determined by measuring the velocity of tricuspid regurgitation jet. In this case, the tricuspid regurgitation jet is a reflection of the peak pressure difference between the right ventricle and the right atrium in systole. If that gradient can be measured using the Bernoulli equation, right ventricular systolic pressure can be estimated, provided right atrial systolic pressure is known. Most patients with elevated right heart pressure will have some degree of tricuspid regurgitation, and obtaining an accurate measure of tricuspid regurgitation jet velocity is possible from multiple views. When measuring peak TRV, care should be taken to identify the maximal modal frequency of the jet, not the ill-defined frequencies that are often recorded when the Doppler gain is set too high. In some cases, right heart contrast, using agitated saline, is necessary to clearly delineate the jet envelope. The right ventricle-to-right atrial pressure gradient may be difficult to estimate in the setting of severe tricuspid regurgitation, when there is a large color flow regurgitant jet. In this case, the peak velocity may not reflect the true pressure gradient.

This approach to determining right ventricular pressure is demonstrated in Figure 8.28. To complete the equation, right atrial pressure is estimated on the basis of jugular venous pressure or based on an assessment of inferior vena cava dimensions. By observing the degree of dilation and the respiratory variability in inferior vena cava caliber, right atrial pressure can be estimated with reasonable accuracy. An algorithm to estimate right atrial pressure noninvasively is provided in Table 8.3.
FIGURE 8.28. The Bernoulli equation is used to estimate right ventricular systolic pressure. A: A significant tricuspid regurgitation (TR) jet is demonstrated using color Doppler imaging (arrows). B: Continuous-wave Doppler imaging demonstrates a TR jet velocity of 4.9 m/s. The calculations used to estimate right ventricular systolic pressure (RVSP) are shown.

\[
\text{RVSP} = (4 \times 4.9^2) + 10 \\
= 96 + 10 \\
= 106 \text{ mmHg}
\]

Table 8.3  
ESTIMATION OF RIGHT ATRIAL PRESSURE BASED ON THE INFERIOR VENA CAVA

<table>
<thead>
<tr>
<th>IVC Diameter (cm)</th>
<th>Response to Sniff</th>
<th>RA Pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤2.1</td>
<td>&gt;50% collapse</td>
<td>3</td>
</tr>
<tr>
<td>≤2.1</td>
<td>&lt;50% collapse</td>
<td>8</td>
</tr>
<tr>
<td>&gt;2.1</td>
<td>&lt;50% collapse</td>
<td>15</td>
</tr>
</tbody>
</table>


In the setting of pulmonary regurgitation, one can measure the end-diastolic pulmonary regurgitant jet velocity. This measurement provides the pressure gradient between the pulmonary artery and the right ventricle at the end of diastole (Fig. 8.29). Combining this pressure gradient with right
ventricular diastolic pressure or right atrial pressure provides a measurement of pulmonary artery diastolic pressure. Specifically, by adding the end-diastolic pressure gradient (from the pulmonary regurgitation velocity) to the right atrial pressure, pulmonary artery diastolic pressure can be estimated. For example, if the end-diastolic pulmonic regurgitation velocity is 2 m/s, this corresponds to a gradient of 16 mm Hg and suggests that the pulmonary artery diastolic pressure is approximately 16 mm Hg higher than the mean right atrial (or right ventricular diastolic) pressure.

An estimate of pulmonary vascular resistance can be obtained by dividing the peak TRV (in meter per second) by the TVI of the right ventricular outflow tract (in centimeter). The rationale for this method is based on the recognition that pulmonary vascular resistance is directly related to the change in pressure and inversely related to pulmonary flow. The regression equation yielding the best agreement with invasively determined pulmonary vascular resistance (PVR) is:

\[
PVR = \frac{\text{TRV}}{\text{TVI}_{\text{rvot}}} \times 10 + 0.16 \quad \text{[Eq. 8.10]}
\]

This approach may have utility in distinguishing high pulmonary artery pressure due to increased pulmonary flow from pulmonary hypertension due to elevated pulmonary vascular resistance (Fig. 8.30). For example, if the pulmonary artery pressure is high, but the TRV/TVI_{rvot} is less than 0.2, this most likely indicates low pulmonary vascular resistance, with elevated pressure secondary to increased flow. In the example, the pulmonary artery systolic pressure estimated from the tricuspid regurgitation jet would be 70 mm Hg. This high pressure, along with the low pulmonary valve flow, indicated by the TVI_{ot}, is consistent with elevated pulmonary vascular resistance. While this approach to estimating pulmonary vascular resistance is conceptually attractive, the evidence base to support its routine clinical use is scant. It may be most helpful at the extremes, for example, when right ventricular stroke volume is either very high or low. In most cases, however, it should not be used as a substitute for invasive measurements.
FIGURE 8.29. Three examples of Doppler recording of pulmonary regurgitation. By measuring the jet velocity at end-diastole, the pressure gradient between the pulmonary artery and the right ventricle in late diastole can be determined. In these three examples, the end-diastolic gradient ranges from 4 to 34 mm Hg. PG, pressure gradient.
PVR = TRV/TVI_{OT} \times 10 + 0.16
= (4.17/9) \times 10 + 0.16
= 0.46 \times 10 + 0.16
= 4.8 \text{ Wood units}

FIGURE 8.30. Pulmonary vascular resistance (PVR) can be estimated by measuring the peak velocity of the tricuspid regurgitation jet (TRV) and the time velocity integral (TVI) of the right ventricular outflow tract. See text for details.

The Bernoulli equation can also be used on the left side of the heart to estimate left ventricular end-diastolic pressure in patients with aortic regurgitation. By measuring the end-diastolic velocity of the aortic regurgitation jet, left ventricular end-diastolic pressure can be determined by subtracting the gradient from the aortic diastolic pressure (Fig. 8.31). The problem with this calculation is that end-diastolic aortic pressure is difficult to estimate noninvasively. It is generally not acceptable to substitute diastolic blood pressure (derived from a cuff measurement) for this value. Also, because left ventricular end-diastolic pressure varies over a relatively narrow range, small errors in the calculation can lead to significant clinical errors in the final estimate.

A final application of the Bernoulli equation involves the use of mitral regurgitation to estimate the rate of left ventricular pressure increase during early systole, also known as dP/dt. Because there is little change in left atrial pressure during the period of isovolumic contraction, the early mitral regurgitation jet velocity reflects dP/dt. By measuring the slope of the mitral regurgitation acceleration velocity, dP/dt can be determined. This is done by measuring the time interval between 1 m/s and 3 m/s on the mitral regurgitation jet, as shown in Figure 8.32. By the Bernoulli equation, this
interval corresponds to an increase in pressure difference from 4 to 36 mm Hg, a net change of 32 mm Hg. Thus, \( \frac{dP}{dt} \) is calculated as 32 divided by the time interval, expressed in mm Hg/s. Several studies have demonstrated a good correlation between this Doppler approach and catheter-derived values for \( \frac{dP}{dt} \). Some examples of calculating \( \frac{dP}{dt} \) are provided in Figure 8.33.

\[ \text{FIGURE 8.31.} \text{ The Bernoulli equation can be used to estimate left ventricular end-diastolic pressure (LVEDP), as shown in this schematic. By measuring the velocity of the aortic regurgitation (AR) jet at end-diastole, the aortic-to-left ventricular pressure gradient is estimated. By subtracting this value from the aortic diastolic pressure, LVEDP is determined. See text for details.} \]

\[ \text{DETERMINING PRESSURE HALF-TIME} \]

Pressure half-time was originally developed and used in the cardiac catheterization laboratory for evaluating patients with mitral stenosis. By simultaneously plotting left atrial and left ventricular pressure curves, the contour of the diastolic pressure gradient across the mitral valve could be evaluated. Pressure half-time is the time required for the peak pressure gradient to be reduced by one-half (Fig. 8.34). Thus, if the maximal pressure
gradient is 14 mm Hg, then the pressure half-time is the time required for the instantaneous gradient to decrease from 14 to 7 mm Hg. With Doppler imaging, we are actually measuring velocity rather than pressure. Because of the quadratic relationship between the two parameters, the Doppler pressure half-time is the time required for the peak velocity to decrease to a value equal to peak velocity divided by √2. Because √2 equals approximately 1.4, pressure half-time becomes the time required for the initial velocity to decrease to a value of peak velocity divided by 1.4, roughly the same as peak velocity multiplied by 0.7. Thus, the arithmetic involved in deriving pressure half-time from velocity data is summarized as follows:

\[
\frac{dP}{dt} = \frac{(36 - 4)}{t} = 32 \div t \text{ (sec)}
\]

**FIGURE 8.32.** From a continuous-wave recording of the mitral regurgitation jet, \( \frac{dP}{dt} \) can be calculated. The schematic demonstrates this approach. See text for details.
In the setting of mitral stenosis, pressure half-time is a useful measure of severity (Fig. 8.35). As stenosis worsens, pressure half-time increases, that is, the decrease in velocity during diastole occurs more slowly. It has been empirically shown that mitral valve area is approximately equal to 220 divided by pressure half-time. The advantage of pressure half-time is that it is
less dependent on heart rate and flow than are other measures of severity, such as gradient. Thus, it is especially useful in patients with atrial fibrillation, in whom variations in the R-R interval alter the diastolic gradient more so than the pressure half-time.

There are several limitations to the pressure half-time approach to mitral stenosis. For example, conditions that alter the diastolic compliance of the left atrium or ventricle (such as left ventricular hypertrophy) will also affect flow velocity and, hence, the pressure half-time. Aortic regurgitation causes the left ventricular pressure to increase more quickly in diastole than would otherwise occur. This can lead to a shortening of the pressure half-time and an underestimation of mitral stenosis severity. Of greater clinical relevance, the temporal changes in atrial and ventricular compliance that accompany balloon mitral valvuloplasty create an unsteady state during which pressure half-time may be inaccurate. This is a temporary problem, lasting between 48 and 72 hours after the procedure. After that, compliance stabilizes and the half-time method can be used to assess the success of the procedure.

FIGURE 8.34. The determination of pressure half-time of the mitral stenosis jet.
The pressure half-time formula has also been applied to aortic regurgitation jets. In this case, the rate of decrease of the jet velocity during diastole is a reflection of the rate of increase of left ventricular diastolic pressure and the rate of decrease of aortic diastolic pressure. The more quickly the left ventricular and aortic pressure curves approach each other during diastole, the steeper the slope of the aortic regurgitation flow profile is and the shorter the pressure half-time (Fig. 8.36). As aortic regurgitation worsens, left ventricular pressure increases more quickly, aortic pressure decreases more quickly, and pressure half-time shortens.

Although there is a general relationship between aortic regurgitation severity and pressure half-time, it must be emphasized that several factors can also affect this value. For example, in the setting of acute aortic regurgitation, left ventricular pressure increases rapidly during diastole as blood fills a normal-size left ventricle from both the aortic root and the mitral valve. This rapid increase in left ventricular pressure will tend to shorten pressure half-time. In contrast, in the presence of long-standing aortic regurgitation in which the left ventricle is markedly dilated and compliant, a significant amount of aortic regurgitation can occur with a relatively flat left ventricular diastolic pressure curve and a long pressure half-time. These differences are illustrated in Figure 8.36. Thus, pressure half-time is affected by both severity and acuity, and differentiating these factors in an individual patient can be difficult.
FIGURE 8.35. From a patient with rheumatic mitral stenosis, Doppler is used to calculate the mean gradient (MnPG) and the pressure half-time ($P_{1/2}$) of the mitral valve flow. A: From the parasternal long-axis view, the mitral valve is thickened and domes in diastole. The left atrium is dilated. Using the Doppler tracing from the apical four-chamber view, mean gradient can be determined by planimetry of the diastolic inflow tracing (B) and pressure half-time can be derived from the slope of the deceleration curve (C). See text for details.
FIGURE 8.36. The contour of the aortic regurgitation (AR) jet reflects the instantaneous pressure difference between the aorta and the left ventricle during diastole. **A:** Mild AR is demonstrated. The relationship between the pressure tracings and the Doppler contour is illustrated. **B:** More severe AR results in a steeper slope of the AR jet. See text for details.
The continuity equation is based on Newton’s second law of thermodynamics, involving the conservation of mass. As it applies to Doppler imaging, this principle states that the volumetric flow rate through the cardiovascular system is constant, assuming that the blood is noncompressible and the conduit is inelastic. Stated differently, the flow rate (or volume of blood passing through any given point over time) is the same at all points along the circuit. Because flow rate is the product of the TVI and the cross-sectional area, this relationship can be used to solve for the cross-sectional area as follows:

\[
\text{Flow proximal} = \text{flow distal} \\
A_1 \times \text{TVI}_1 = A_2 \times \text{TVI}_2 \\
A_2 = A_1 \times \frac{\text{TVI}_1}{\text{TVI}_2} \quad \text{[Eq. 8.12]}
\]

By sampling the TVI at two points and measuring the cross-sectional area at one point, the other cross-sectional area can be determined using this equation (Fig. 8.37). For example, to calculate the cross-sectional area of a stenotic aortic valve, the following three measurements must be made: (1) the TVI of the left ventricular outflow tract, using pulsed Doppler recording just proximal to the stenotic valve; (2) the TVI through the valve, using continuous-wave Doppler imaging; and (3) the cross-sectional area of the outflow tract, at the same point where flow was measured.
FIGURE 8.37. This illustration demonstrates how aortic valve area is calculated. In A, pulsed Doppler of aortic outflow tract area indicates a time velocity integral (TVI) of 26 cm. In B, the outflow tract (OT) diameter is used to calculate the area, in this case, 3.46 cm². In C, continuous wave Doppler records the aortic stenosis jet with peak velocity of 4.7 m/s and a TVI of 127 cm. The valve area is therefore 0.7 cm².
FIGURE 8.38. The relationship among aortic stenosis jet velocity, valve area, and stroke volume. See text for details. AV, aortic valve; TVIOT, time velocity integral of the left ventricular outflow tract.

The advantages of the continuity equation are that it is unaffected by valvular regurgitation and provides quantitative assessment of severity even in the presence of left ventricular dysfunction (when gradient alone may lead to underestimation of severity). Figure 8.38 is a schematic that demonstrates the critical dependence of the Bernoulli equation on stroke volume. The two curves depict the relationship between jet velocity and aortic valve area at different levels of left ventricular function, indicated by different flow rates (the TVIot values). Beginning at point A, with a peak gradient of 32 mm Hg and a valve area of 1.3 cm², a worsening of stenosis (at the same flow rate) corresponds to a move to point B, which is a gradient of 74 mm Hg and a valve area of 0.8 cm². This would be typical progression of stenosis with preserved ventricular function. Alternatively, a decrease in flow rate or stroke volume without a change in valve area would imply shifting to the higher curve. On this curve, if the valve area is still 1.3 cm², the corresponding gradient will decrease to 15 mm Hg (point C). At this new stroke volume, a
progression of aortic stenosis to a new valve area of 0.8 cm\(^2\) would return the gradient to the original value of 32 mm Hg (point D). It is apparent that the same gradient can reflect widely different valve areas, depending on the flow rate through the valve. Clearly, in the setting of changing flow states, gradient alone cannot convey adequate diagnostic information about stenosis severity. It is in these situations that the continuity equation can be most helpful.

The continuity equation can be applied to any of the four valves within the heart, although usually it is the aortic valve that is evaluated with this technique. In the setting of left ventricular dysfunction, it can be performed both at baseline and during dobutamine stress to differentiate between severe valvular stenosis and less severe stenosis in the setting of low flow rates. The clinical application of the continuity equation as it applies to the aortic valve is covered in Chapter 10.

**PROXIMAL ISOVELOCITY SURFACE AREA**

A novel application of the continuity principle involves the proximal isovelocity surface area method. As blood converges toward an orifice, Doppler flow imaging reveals concentric shells or hemispheres, which represent isovelocity surfaces (Fig. 8.39). As the blood accelerates toward the orifice, velocity aliasing occurs and a distinct red–blue interface occurs at the boundary of the shells. At this interface, the velocity is equivalent to the Nyquist limit, which can be read off the velocity color scale. By adjusting the Nyquist limit, the size of the shell can be maximized to allow its surface area to be measured according to the formula:

\[
\text{Surface area} = 2\pi r^2 \quad \text{[Eq. 8.13]}
\]

By the continuity equation, we know that flow rate is held constant as blood converges toward the orifice. Thus, flow rate through any given shell will equal the flow rate through the orifice. The rate of flow through any hemispheric shell is the product of the hemisphere area and the flow velocity (i.e., the aliasing velocity). Thus, the following equation can be derived:

\[
\text{Flow rate} = 6.28 \times r^2 \times \text{Aliasing velocity} \quad \text{[Eq. 8.14]}
\]

Similarly, the flow rate through a regurgitant orifice is given by the
equation:
\[ \text{Flow rate} = \text{ERO} \times \text{Velocity}_{\text{jet}} \quad [\text{Eq. 8.15}] \]

We can then calculate the effective regurgitant orifice (ERO) according to the formula:
\[ \text{Surface area} = 2\pi r^2 \quad [\text{Eq. 8.13}] \]
Flow = $2\pi \times r^2 \times V_a$

ERO = Flow + $V_{MR}$

RV = ERO × TVI_{MR}
FIGURE 8.39. Determination of mitral regurgitation (MR) severity by the proximal isovelocity surface area method. **A:** The schematic demonstrates how regurgitant flow converges and accelerates in a series of isovelocity shells, indicated by the red and blue patterns. **B:** The radius of the shell is measured, after the baseline has been shifted to maximize its size. From this, the surface area of the shell is determined. **C:** Using the continuity equation, the calculations required to measure flow, effective regurgitant orifice (ERO) area, and regurgitant volume (RV) are demonstrated. See text for details. $r$, radius; TVI, time velocity integral; $V_a$, aliasing velocity; $V_{MR}$, maximal MR jet velocity.

![Figure 8.39](image1.png)

FIGURE 8.40. An example of how mitral regurgitation severity is determined using the proximal isovelocity surface area method. The calculations are as described in the text. ERO, effective regurgitant orifice; TVI, time velocity integral.

![Figure 8.40](image2.png)

Regurgitant volume (RV, in milliliters) then becomes

$$RV = ERO \times TVI_{MR} \quad [\text{Eq. 8.17}]$$

Thus,
This is illustrated in Figure 8.40.

Although attractive in concept, the routine clinical use of proximal isovelocity surface area has its limitations. Assumptions about the hemispheric shape of the isovelocity shells may be oversimplified. Three-dimensional echocardiography has demonstrated that some isovelocity shells may, in fact, be nonhemispheric. Although the surface area of a noncircular shell may still be calculated, this adds an additional complexity to the equations and introduces another potential source of error. Another assumption states that the shells are converging toward an orifice that lies within a flat plane. In the case of mitral regurgitant flow, this is clearly not the case and some correction is often required. Furthermore, the calculations are cumbersome and the potential for measurement error must always be considered. This is particularly true with regard to the radius of the isovelocity shells, where precise identification of the center of the regurgitant orifice can be especially challenging.

\[
RV = ERO \times TVI_{MR} \tag{Eq. 8.17}
\]

Thus,

\[
RV = \frac{2\pi r^2 \times \text{Al. vel.}}{\text{Vel}_{MR} \times TVI_{MR}} \tag{Eq. 8.18}
\]

FIGURE 8.41. Another example of the proximal isovelocity surface area method for estimating the severity of mitral regurgitation is provided. In A, an isovelocity shell is recorded as flow accelerates toward the mitral regurgitant orifice. The radius is indicated by the white arrow. In this example, most of the mitral regurgitation flow occurs in late systole (B). Without correcting for this, regurgitant volume would be overestimated. See text for details.
The PISA calculation also assumes that mitral regurgitation occurs throughout systole at a constant flow rate. In Figure 8.41, from a patient with mitral valve prolapse, a well demarcated isovelocity shell is recorded (Fig. 8.41A). However, the continuous-wave Doppler shows that most of the regurgitation occurs in the latter half of systole (Fig. 8.41B). Without correcting for this, PISA would overestimate regurgitant volume. For all these reasons, proximal isovelocity surface area has not yet become a routinely performed measurement. Its application to quantifying mitral regurgitation is covered more in Chapter 11.

**MYOCARDIAL PERFORMANCE INDEX**

The myocardial performance index (MPI) was developed in the mid-1990s as an expression of global ventricular performance. It is a simple index that includes both systolic and diastolic parameters and can be applied to either the left or the right ventricle. The MPI incorporates three basic time intervals that are readily derived from Doppler recordings: ejection time (ET), isovolumic contraction time (IVCT), and isovolumic relaxation time (IVRT). From these values, the following calculation is performed (Fig. 8.42):

\[
MPI = \frac{IVCT + IVRT}{ET} \quad [Eq. 8.19]
\]

Systolic dysfunction is associated with a prolongation of IVCT and a
shortening of the ET. Diastolic dysfunction often leads to lengthening of the IVRT. Thus, both systolic and diastolic dysfunction will result in an increase in the MPI. The reported normal range for the MPI is 0.39 ± 0.05. Values greater than 0.50 are considered abnormal. Not surprisingly, this measurement has been shown to be a useful tool to risk stratify patients with a broad range of diseases. The MPI can also be used to assess right ventricular function. For the right side of the heart, the normal MPI is 0.28 ± 0.04. An increased right ventricular MPI is a sensitive and specific marker of pulmonary hypertension. Thus, the MPI may be of value in patients in whom tricuspid regurgitation is either not present or cannot be quantified to assess for pulmonary hypertension. The MPI also appears to provide powerful prognostic information. Further studies are needed, however, to determine its place among the other Doppler prognostic variables.

**FIGURE 8.42.** This schematic demonstrates how the myocardial performance index (MPI) is derived. See text for details. ET, ejection time; IVCT, isovolumic contraction time; IVRT, isovolumic relaxation time.

Suggested Readings

**GENERAL CONCEPTS**


**Gradients & Stenosis**


**Quantitative Flow**


**Pressure Recovery**


**Regurgitation**


Samstad SO, Hegrenaes L, Skjaerpe T, Hatle L. Half time of the diastolic aortoventricular pressure


**Right Heart**


**Miscellaneous**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Chapter 9
Pericardial Diseases

CLINICAL OVERVIEW

Anatomically, the pericardium consists of two layers. The visceral pericardium is contiguous with the epicardium and the parietal pericardium is the thicker fibrous sac surrounding the heart. Although it is often the parietal pericardium that is typically referred to as the pericardium, it should be emphasized that most disease states simultaneously involve both the parietal and the visceral pericardia. Normally, there is 5 to 10 mL of normal buffering fluid within the pericardial space. The pericardium encases all four chambers of the heart and extends 1 to 2 cm up the great vessels. The pericardium similarly reflects around the proximal pulmonary veins. The pericardial reflection around the great vessels limits the size of the pericardial space at these junctures. The reflection of the parietal pericardium around the great vessels at the base of the heart creates the transverse and oblique pericardial sinuses which may result in fluid collections in the area of the left atrial appendage.

The pericardium restrains the four cardiac chambers within a relatively confined volume and space within the thorax. Because of pericardial constraint, the total volume of the four cardiac chambers is limited, and alterations in the volume of one chamber must, by necessity, be reflected in an opposite change in volume of another chamber. An exaggerated linking of intracardiac volumes is the underlying pathophysiology for development of pulsus paradoxus and other findings seen in cardiac tamponade and pericardial constriction.

Pericardial disease can present as several different clinical scenarios, and for each of these, echocardiography can play a significant role. Pericardial
effusions can accumulate in any infectious or inflammatory process involving the pericardium. Most infectious and inflammatory processes involve both layers of the pericardium. Table 9.1 outlines diseases that can affect the pericardium. Acute pericarditis of any etiology may result in accumulation of variable amounts of pericardial fluid. In its early phases, inflammation may be present in the absence of any significant accumulation of pericardial fluid. It is important to evaluate left ventricular function in patients presenting with suspected acute pericarditis to exclude a component of myocarditis.

Because the pericardial space is limited in size, accumulation of significant pericardial fluid reduces the total volume that the four cardiac chambers can contain and may result in hemodynamic deterioration related to functional underfilling of the ventricles. Hemodynamic compromise is related to elevated intrapericardial pressure, which in turn is related to the volume of pericardial fluid, the rate at which it accumulates, and the compliance or distensibility of the pericardium. As such, a slowly developing large effusion may be associated with less hemodynamic compromise than a smaller but more rapidly developing effusion. Inflammatory processes of the pericardium typically result in pain and fluid accumulation and more chronically can result in fibrous stranding and stiffening of the pericardium which can lead to pericardial constriction. Other types of pericardial pathology, such as pericardial cysts and congenital absence of the pericardium, are often noted as incidental findings in asymptomatic individuals or may be associated with atypical and highly variable symptomatology.

### Table 9.1 ETIOLOGY OF PERICARDIAL DISEASE

<table>
<thead>
<tr>
<th>Category</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic</td>
<td>Acute idiopathic pericarditis(^a)</td>
</tr>
<tr>
<td></td>
<td>Chronic idiopathic effusion</td>
</tr>
<tr>
<td>Infectious</td>
<td>Viral</td>
</tr>
<tr>
<td></td>
<td>Bacterial direct infection (postprocedure)</td>
</tr>
<tr>
<td></td>
<td>Tuberculosis</td>
</tr>
<tr>
<td></td>
<td>Spread from contiguous infection (e.g., pneumonia)</td>
</tr>
<tr>
<td></td>
<td>Fungal</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>Associated with connective tissue disease</td>
</tr>
<tr>
<td>Indication</td>
<td>Appropriateness</td>
</tr>
<tr>
<td>------------</td>
<td>----------------</td>
</tr>
<tr>
<td>19.</td>
<td>Hypotension or hemodynamic instability of uncertain or suspected cardiac etiology</td>
</tr>
<tr>
<td>23.</td>
<td>Suspected complication of myocardial ischemia/infarction, including but not limited to acute mitral regurgitation, ventricular septal defect, free-wall rupture/tamponade, shock, right ventricular involvement, HF, or thrombus</td>
</tr>
<tr>
<td>32.</td>
<td>Severe deceleration injury or chest trauma when valve injury, pericardial effusion, or cardiac injury are possible or suspected</td>
</tr>
<tr>
<td>59.</td>
<td>Suspected pericardial conditions</td>
</tr>
<tr>
<td>60.</td>
<td>Routine surveillance of known small pericardial effusion with no change in clinic status</td>
</tr>
</tbody>
</table>
ECHOCARDIOGRAPHIC AND MULTIMODALITY EVALUATION OF THE PERICARDIUM

Detection of pericardial disease was one of the first clinical uses of echocardiography. It remains the primary imaging technique for diagnosis and management of virtually all forms of pericardial disease (Table 9.2). Anatomically, the pericardium can be evaluated with M-mode, two-dimensional, and three-dimensional echocardiography as well as intracardiac ultrasound. Normally, there may be a small amount of fluid in the pericardial space that typically collects in the dependent areas. It is most often appreciated as a very small echo-free space in the posterior atrioventricular groove. This space may increase in size during systole (Figs. 9.1 and 9.2). In the absence of pericardial effusion, dramatic thickening, or calcification, it is unusual to directly visualize the pericardium with either M-mode or two-dimensional echocardiography. Intracardiac ultrasound has been used to directly visualize the pericardium but is infrequently used for this purpose.

More recently cardiac CT and MRI have shown value in assessing pericardial disease including detection, localization, and quantization of pericardial fluid. CT and MRI have substantial value in assessing pericardial thickness and can detect the exaggerated respiratory ventricular interdependence that is the hallmark of pericardial constriction. Cardiac CT is the most sensitive technique for detection of pericardial calcification.

Detection and Quantitation of Pericardial Fluid

Pericardial effusion can be detected with all the traditionally used echocardiographic techniques. On M-mode echocardiography, pericardial...
effusion appears as an echo-free space both anterior and posterior to the heart. The size of the echo-free space is directly proportional to the amount of fluid. There are no accurate M-mode techniques for quantifying the absolute volume of pericardial fluid. It should be emphasized that an isolated anterior free space is not specific for pericardial fluid and may be due to mediastinal fat, fibrosis, thymus, or other tissues.

FIGURE 9.1. M-mode echocardiograms recorded in patients with pericardial effusions. A: Note the echo-free space (arrow) immediately behind the posterior
Two-dimensional echocardiography is the most commonly used method to screen for and quantify pericardial effusion. Most echocardiography laboratories visually quantify pericardial effusion on the basis of the magnitude of separation of the visceral and parietal pericardia in diastole. Typically, a minimal pericardial effusion represents the normal amount of pericardial fluid in a disease-free state (Fig. 9.2). It is visualized as a small echo-free space in the posterior atrioventricular groove visible only in systole when the heart has pulled away from the pericardium. A small effusion is defined as one resulting in as much as 1 cm of posterior echo-free space, with or without fluid accumulation elsewhere (Fig. 9.3). Smaller effusions tend to collect in the dependent aspect of the pericardial space and, as such, their exact position may vary with patient position. Moderate effusions have been defined as 1 to 2 cm of echo-free space and large effusions as more than 2 cm of maximal separation (Figs. 9.4 to 9.8). In very large effusions, the heart may swing within the pericardial space (Figs. 9.7 and 9.9). It should be emphasized that these definitions may vary from laboratory to laboratory and in clinical practice semiquantitative description of pericardial fluid size is standard practice. The effusion should also be characterized as to the presence or absence of hemodynamic compromise.

On two-dimensional echocardiography, pericardial effusion tends to be most prominent in the more dependent (i.e., posterior in a patient in a supine position) area and frequently appears maximal in the posterior atrioventricular groove (Figs. 9.2 to 9.4). Using additional views including the parasternal short-axis, apical, and subcostal views, the circumferential extent of an effusion can be reliably determined. In Figure 9.7 the circumferential extent of the effusion is confirmed in the short-axis view. As the heart is not constrained by an inflammatory component, the heart moves freely within the pericardial space buffered by the large pericardial effusion. This variable cardiac position from beat to beat, referred to as a “swinging heart,” is the etiology of electrical alternans seen on the electrocardiogram (Fig. 9.9).
Parasternal long-axis echocardiogram recorded in a patient with a
minimal pericardial effusion. This amount of pericardial fluid represents the normal fluid seen in disease-free individuals. **A:** Recorded at end diastole. **B:** Recorded at end systole. Note that at end diastole, there is no separation between the epicardium and the pericardium. At end systole, the epicardium has lifted off the pericardium revealing a very small pericardial effusion, maximal in the posterior interventricular groove (arrow).

**Video 9-2**

Effusions may be localized or loculated rather than circumferential. This is not uncommon after cardiac surgery or cardiac trauma in which an inflammatory component of the pericardial effusion may result in unequal distribution of fluid in the pericardial space. **Figure 9.10** was recorded in an individual with a laterally localized pericardial effusion.

The pericardium reflects around the pulmonary veins which limits the size of a pericardial effusion behind the left atrium. Previous guidelines had suggested that a fluid collection behind the left atrium was more likely to be plural than pericardial. There are numerous exceptions to this rule, and larger pericardial effusions often collect behind the left atrium as well (Fig. 9.11). Additionally, pericardial fluid may collect in the transverse sinus, which is a potential space bordered by the left atrium and great vessels (Fig. 9.12). In this instance, pericardial fluid may surround the left atrial appendage, left atrium, and pulmonary artery and may be confused for an abscess cavity or part of complex atrial appendage anatomy. Use of contrast for blood pool opacification often resolves any confusion regarding the latter (Fig. 9.12B).
FIGURE 9.3. Parasternal long-axis (A) and short-axis (B) echocardiogram
recorded in a patient with a small pericardial effusion. Note the echo-free space, maximal in the posterior interventricular groove (arrow). In the real-time image, this pericardial effusion can be seen to be present both in diastole and in systole.

Video 9-3a

coming soon

Video 9-3b

Three-dimensional echocardiography provides a unique imaging perspective on the size and distribution of pericardial effusion but has not been shown to be of incremental clinical benefit (Figs. 9.13 and 9.14). Theoretically, three-dimensional echocardiography provides an accurate technique for determining pericardial fluid volume and distribution. Using this technique, the three-dimensional volume of the entire pericardial space can be calculated. The overall total volume of the entire heart (all four
chambers) is then likewise calculated, and the pericardial fluid volume is calculated as the difference between these two volumes. Three-dimensional echocardiography is limited for this purpose because registration of a three-dimensional data set of a significant size to encompass the entire pericardial volume in larger effusions is often not possible. Although probably accurate for determining the volume of pericardial fluid, this technique has had little clinical acceptance because of the lack of a clinical need for determining precise pericardial volume as opposed to its hemodynamic effect.

FIGURE 9.4. Parasternal long-axis view recorded in a patient presenting with a large pericardial effusion. Note the large effusion located predominantly in the posterior aspect (double-headed arrow).
FIGURE 9.5. Apical four-chamber view recorded in the same patient presented in Figure 9.4. Note the large posterolaterally located effusion (double-headed arrow), as well as the effusion in the area of the right atrium and the invagination of the right atrial wall (arrow) indicative of hemodynamic compromise.
Video 9-5
FIGURE 9.6. Parasternal long- and short-axis views of the heart in a patient with a circumferential moderate pericardial effusion (arrows). Note the effusion posterior to the left ventricle and anterior to the right ventricle and a mobility of the heart within the pericardial space in the real-time image.
FIGURE 9.7. Parasternal short-axis view recorded in a patient with a massive pericardial effusion. In the real-time image note the free “swinging” motion of the heart within the pericardial space. Marked left ventricular hypertrophy secondary to hypertensive heart disease is also present.

Video 9-7

Direct Visualization of the Pericardium

In disease-free states, the normal pericardium is rarely visualized with any of
the traditional echocardiographic modalities. Intracardiac ultrasound can potentially visualize the actual thickness of the pericardium but is obviously an invasive technique. In the absence of a pleural effusion, which creates a fluid layer on either side of the pericardium, the exterior portion of the parietal pericardium abuts normal intrathoracic structures, and, therefore, its thickness and character cannot reliably be separated from the surrounding tissues. When both pericardial and pleural effusions are present, the thickness of the pericardium in that area can be ascertained from the transthoracic approach (Fig. 9.15). In instances of marked fibrosis and calcification, it may be possible to infer substantial pericardial thickening, but actual measurement of pericardial thickness is problematic. In the presence of calcific pericarditis, there may be marked shadowing seen posterior to the pericardium (Fig. 9.16). It should be emphasized that the normal pericardium is a highly reflective structure and that a bright pericardial echo alone should not be used to establish the diagnosis of constrictive pericarditis or of a thickened pericardium.

**FIGURE 9.8.** Subcostal echocardiogram reveals a moderate to large pericardial effusion. Note the effusion surrounding the entire heart, with its greatest dimension lateral to the left ventricular free wall. Fluid is clearly seen surrounding
the right atrium and between the pericardium and the right ventricle.

Video 9-8
FIGURE 9.9. Apical four-chamber view recorded from a patient with a large pericardial effusion and a swinging heart. The two panels were recorded at the
same time point from different cardiac cycles. Note the marked change in position of the heart within the pericardial space, which can be appreciated as a swinging heart in the real-time image. The arrows denote the position of the cardiac apex at the same point in the cardiac cycle for two different cycles. This variable position within the thorax is the cause of electrocardiographic electrical alternans.

**Video 9-9**

Masses and stranding, which can occur on either the visceral or parietal pericardium, can be visualized with two-dimensional echocardiography. Detection of stranding implies an inflammatory or possibly hemorrhagic or malignant etiology of the pericardial effusion (Figs. 9.17 and 9.18). It is often seen in uremic or infectious pericarditis. Masses within the pericardium can be the result of metastatic disease (Figs. 9.19 and 9.20) but are often seen in pericardial effusions due to an inflammatory process as well.

Historically, M-mode echocardiography was used for assessment of pericardial disease. Typically, the heart lifts off the parietal pericardium in systole. By increasing the damping of the M-mode beam to a point at which the myocardium is no longer visualized, the M-mode echocardiogram will visualize only the relatively denser pericardial echoes. Persistence of a bright pericardial signal with progressive damping was one of the M-mode signs of pericardial thickening and/or constriction (Fig. 9.21).
FIGURE 9.10. Apical four-chamber (A) and parasternal short-axis (B) views recorded in a patient with a moderate, localized, predominantly lateral PEF. This echocardiogram was recorded approximately 2 weeks after cardiac surgery.

Computed tomography and cardiac magnetic resonance imaging play a
valuable role in pericardial disease. They can detect pericardial fluid and, depending on fluid density, suggest a hemorrhagic etiology on the basis of analyzing the Hounsfield unit of the fluid. Their primary advantage over echocardiography is for direct visualization of pericardial thickness (Figs. 9.22 and 9.23). Cardiac MR can provide evidence of pericardial thickening and delayed gadolinium enhancement provides evidence of active pericardial inflammation. Dynamic imaging with either cardiac MR or CT can also demonstrate evidence of ventricular interdependence in pericardial constriction.

FIGURE 9.11. Parasternal long-axis view recorded in an otherwise healthy female presenting with hypotension and shock. In this parasternal long-axis view, note the large pericardial effusion. Note the presence of a smaller amount of pericardial effusion behind the left atrium (arrow). On the accompanying M-mode, note the variable location of the cardiac chambers consistent with a swinging heart and the diastolic RV free wall collapse (arrow), suggesting hemodynamic compromise in this patient with clinical cardiac tamponade.
FIGURE 9.12. Transesophageal echocardiogram recorded in a patient being evaluated for left atrial appendage thrombus. In **A**, note the left atrial appendage, as well as echo-free spaces superior and medial to the body of the left atrial appendage (*small arrows*). In **B**, intravenous contrast has been given for blood pool opacification, and one can clearly delineate the true boundary of the left atrial appendage versus the nonenhanced echo-free spaces which represent fluid in the transverse and oblique sinuses.
FIGURE 9.13. Transthoracic real-time three-dimensional imaging in a patient with a moderate pericardial effusion in parasternal long- and short-axis views. Note the
circumferential effusion surrounding the left and right ventricles (*arrows*) and the excellent visualization of the extent of free fluid surrounding the heart.

Video 9-13a

Video 9-13b
FIGURE 9.14. Real-time three-dimensional echocardiogram recorded in a patient with a large pericardial effusion (at the time of pericardial synthesis, 1,000 cc of bloody fluid was drained). Note the distribution of the pericardial fluid medially, laterally, and anteriorly (long arrows). Note the nodular thickening over the right ventricular free wall as evidence of marked inflammatory response. Video 9-14
FIGURE 9.15. Parasternal long-axis echocardiogram recorded in a patient with a small pericardial effusion and a larger pleural effusion (PL). The presence of concurrent pericardial and pleural fluid allows identification of the parietal pericardium. In this instance, the pericardial thickness can be seen to be approximately 2 mm. Note the position of the two fluid collections with respect to the descending thoracic Ao.
FIGURE 9.16. Parasternal long-axis echocardiogram recorded in a patient with a partially calcified posterior pericardium (*black arrows*). The posterior pericardium has pathologic echo intensity and appears thickened, although because of reverberation, the actual thickness cannot be reliably determined. The markedly echogenic pericardium has resulted in reverberation artifact, creating a double image of the left ventricular cavity behind the pericardial space, best appreciated in the real-time image.
FIGURE 9.17. Subcostal view recorded in a patient with a moderate pericardial effusion related to uremic pericarditis. Note the multiple fibrous strands (arrows) in the pericardial space, many of which appear to bridge the parietal and visceral pericardia.
**FIGURE 9.18.** Apical four-chamber view recorded in a patient with an inflammatory pericardial effusion. Note the stranding within the pericardial space between the visceral and parietal pericardium (*arrows*).
FIGURE 9.19. Parasternal long-axis view recorded in a patient with a metastatic malignancy and pericardial effusion. Note the nodular echodensities over the right ventricular outflow tract (arrows). In the M-mode echocardiogram (inset), note the mild intermittent diastolic collapse of the right ventricular free wall (arrows) suggestive of hemodynamic compromise.
FIGURE 9.20. Parasternal short-axis view recorded in a patient with metastatic malignancy and large pericardial effusion. Note the diffuse thickening and nodular masses on the epicardial surface of the right ventricle (downward-pointing arrows) and the two distinct masses within the pericardium, arising from the parietal
pericardium (horizontal arrows).

Video 9-20

**FIGURE 9.21.** M-mode echocardiogram recorded in a patient with constrictive pericarditis and thickened posterior pericardial echoes. To the right of this frame, in the area marked by the black bracket, damping has been increased to suppress the fainter myocardial echoes. Note that the bright pericardial echo has not been suppressed. Also note the flat motion of the posterior wall after the initial rapid posterior motion (arrow) of the endocardium. PW, posterior wall.
Differentiation of Pericardial From Pleural Effusion

A left pleural effusion results in an echo-free space posterior to the heart in a patient in a supine or left lateral position and may be confused for pericardial fluid. There are several echocardiographic clues that help distinguish pericardial from pleural fluid. As noted previously, the pericardial reflections surround the pulmonary veins and tend to limit the potential space behind the left atrium. Because of this, fluid appearing exclusively behind the left atrium is more likely to represent pleural than pericardial effusion. One of the more...
reliable distinguishing features between a pericardial and a pleural effusion is the location of the fluid-filled space with respect to the descending thoracic aorta (Fig. 9.15). The pericardial reflection is typically anterior to the descending thoracic aorta, and therefore, fluid appearing posterior to the descending thoracic aorta is more likely to be pleural, whereas fluid appearing anterior to the aorta is more likely to be pericardial. These observations apply to differentiating pericardial from pleural fluid in the parasternal views. In the apical four-chamber view, separation of a localized lateral pericardial effusion from a pleural effusion can often be problematic. When both pericardial fluid and pleural fluid are present, one can frequently identify the parietal pericardium, which serves as an excellent anatomic landmark to define the extent of each of the two fluid collections.
Accumulation of increasing amounts of pericardial fluid results in predictable hemodynamic alterations. Normal intrapericardial pressure ranges between –5 and +5 cm of water and fluctuates with respiration. Because of the constraining effect of the pericardium on the combined volume of the four cardiac chambers, respiratory variation in intrapericardial pressure results in linked variation in filling of the right and left ventricles. With inspiration, intrathoracic and intrapericardial pressures decrease. The result of this is to augment flow into the right heart and reduce flow out of the pulmonary veins. This results in augmented right ventricular filling and stroke volume. Because the total intrapericardial space is limited, there is a compensatory decrease in left ventricular diastolic volume and stroke volume in early inspiration. In expiration, intrathoracic pressure and intrapericardial pressure increases, resulting in a mild decrease in right ventricular diastolic filling and a subsequent increase in left ventricular filling. This respiratory variation of left and right ventricular filling (respiratory ventricular interdependence) is sufficient to create changes in stroke volume and systemic blood pressure with the respiratory cycle (Fig. 9.24). Typically, the normal respiratory variation in stroke volume results in no more than a 10 mm Hg inspiratory decrease in arterial systolic pressure. Respiratory variation in systolic blood pressure >10 mm Hg (pulsus paradoxus) in the presence of significant pericardial effusion is considered evidence of hemodynamic compromise and defines clinical cardiac tamponade. In addition to pericardial disease, any process that results in increased pressure variation with the respiratory cycle, such as decompensated obstructive lung disease, may result in greater variation in global cardiac filling, stroke volume, and arterial pulse pressure and thus create respiratory-dependent decreases in blood pressure independent of pericardial disease.
FIGURE 9.24. Schematic depiction of the generation of pulsus paradoxus in hemodynamically significant pericardial effusion. Both normal physiology and tamponade physiology are depicted in both inspiration and expiration. In the normal situation, the relative size and geometry of the right and left ventricles are preserved in both inspiration and expiration, and there is little variation of either ventricular outflow or inflow, as depicted by the schematics within the chambers. Exaggerated ventricular interaction in a hemodynamically significant pericardial effusion is shown on the right. Note the relatively greater right ventricular size during inspiration with both augmented inflow and outflow and the concurrent decrease in left ventricular size, outflow tract flow velocity profile, and mitral valve inflow. During expiration (at lower right), left ventricular filling is again augmented as is left ventricular outflow at the expense of reduced right ventricular volume and decreased right ventricular Doppler flow velocities.

The overall effect of an increasing volume of pericardial fluid is to limit the total blood volume allowable within the four cardiac chambers and to, therefore, exaggerate the respiration-dependent ventricular interaction. When intrapericardial pressure approaches normal filling pressures of the heart, it becomes the determining factor for the passive intracardiac pressures including right and left atrial pressures, right ventricular diastolic pressure, pulmonary artery diastolic pressure, left ventricular diastolic pressure, and pulmonary capillary wedge pressure. With elevation of intrapericardial pressure above normal filling pressure, the diastolic pressure in all four cardiac chambers becomes equalized and is determined by intrapericardial pressure. This is the physiologic basis of cardiac tamponade. Because the left
ventricle has a stiffer wall and its diastolic filling is determined by a variety of factors including active relaxation, left ventricular filling is impacted less than the right ventricular filling.

As a result of increased intrapericardial pressure and the limitation on overall cardiac volume, the interaction between the right and left ventricles becomes exaggerated. Figure 9.24 schematizes the interaction between the right and left ventricles in a hemodynamically significant pericardial effusion and outlines the mechanism for pulsus paradoxus. In a pericardial effusion with pathologic elevation of intrapericardial pressure, inspiration results in a disproportionately greater filling of the right ventricle and subsequently greater compromise of left ventricular filling. During expiration, the process is reversed and right ventricular filling is impeded to a substantially greater degree. This results in exaggeration in the respiratory-dependent phasic changes in right and left ventricular stroke volume and subsequently in an exaggerated (>10 mm Hg) decrease in systolic arterial blood pressure with inspiration. This is the mechanism of a pathologic pulsus paradoxus as is seen in cardiac tamponade.

**Echocardiographic Findings in Cardiac Tamponade**

There are multiple echocardiographic features described in patients with hemodynamic compromise and frank cardiac tamponade (Table 9.3). It should be emphasized that cardiac tamponade is a clinical diagnosis. Echocardiographic findings may suggest a hemodynamic abnormality that may be the substrate for tamponade, but echocardiographic abnormalities alone do not establish the diagnosis of clinical cardiac tamponade. One of the earliest proposed signs of cardiac tamponade was a swinging heart, detected on either M-mode or two-dimensional echocardiography (Fig. 9.9). Detection of a swinging heart is simply a marker of a large pericardial effusion in which the heart is free to float within the pericardial space in a phasic manner. A large pericardial effusion is more likely to be associated with elevated intrapericardial pressure, and hence the relationship between a swinging heart and hemodynamic compromise is indirect, rather than being direct evidence of elevated intrapericardial pressure. Because cardiac position varies from beat to beat within the pericardium, its position in relation to an electrocardiographic lead also varies. This is the mechanism of electrical
alternans seen in large pericardial effusions.

More specific signs of hemodynamic compromise have included evidence of elevation in intrapericardial pressure. Diastolic right ventricular outflow tract collapse and exaggerated right atrial collapse during atrial systole (ventricular diastole) are well validated as evidence that intrapericardial pressure exceeds cardiac filling pressures. The earliest description of diastolic right ventricular collapse was obtained using M-mode echocardiography where characteristic posterior motion of the anterior right ventricular wall was noted in diastole (Fig. 9.25). This observation was subsequently confirmed using two-dimensional echocardiography. In patients with elevated intrapericardial pressure, intracavitary cardiac pressure transiently falls below intrapericardial pressure in early diastole, and hydrodynamic compression of these more distensible structures occurs. Anatomically and experimentally, the right ventricular outflow tract is the more compressible area of the right ventricle. In early diastole, immediately after closing of the pulmonary valve, at the time of opening of the tricuspid valve, the right ventricular outflow tract will paradoxically collapse inward (Figs. 9.26 and 9.27). This is evidence that intrapericardial pressure exceeds right ventricular diastolic pressure at this point in the cardiac cycle, and hence the underlying substrate for tamponade is likely to be present. Collapse of the right ventricle is often best appreciated in the parasternal long- and short-axis views but occasionally can be appreciated in the apical four-chamber view. When collapse extends from the more compressible outflow tract to the body of the right ventricle, this is evidence that intrapericardial pressure is elevated more substantially.

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<th>Table 9.3: ECHO DOPPLER FINDINGS IN PERICARDIAL DISEASE</th>
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Doppler
- Exaggerated respiratory variation in inflow velocity
- Phasic variation in right ventricular outflow tract/left ventricular outflow tract flow velocity/TVI
- Exaggerated respiratory variation in inferior vena cava flow

**Constrictive pericarditis**
- Anatomic features
  - Thickened pericardium
  - Dilated inferior vena cava
  - Exaggerated ventricular septal shift with inspiration
  - Septal “shudder” or “bounce”
- M-mode
  - “Flattened” posterior wall motion in diastole (M-mode)
  - Appearance of pericardium with damping (M-mode)
- Doppler
  - Exaggerated E/A of mitral inflow E/A ratio
  - Exaggerated respiratory variation in E velocity
  - Reduced lateral annular e’
  - “Annulus reversus” (medial e’ > lateral e’)
  - Expiratory hepatic vein flow reversal

**FIGURE 9.25.** M-mode echocardiogram recorded in a patient with a large pericardial effusion and evidence of hemodynamic compromise. In the upper panel, note the orientation of the M-mode through the right ventricular outflow tract and left ventricle. On the M-mode echocardiogram, note the motion of the
right ventricular free wall. End systole is noted by the *large downward-pointing arrow*, after which there is further collapse of the right ventricular-free wall (*small arrow*) in early diastole. The timing of this event can also be confirmed by noting that it occurs concurrently with an open mitral valve (*upward-pointing arrow*). This is evidence of elevated intrapericardial pressure and hemodynamic compromise and suggests the possibility of clinical cardiac tamponade.

**FIGURE 9.26.** Parasternal long-axis view recorded in the same patient is presented in Figure 9.25. This frame was recorded in early diastole. Note the distinct downward motion of the right ventricular free wall (*larger arrow*) in early diastole. Note the closed aortic valve and the open mitral valve (*smaller arrow*). This is evidence of elevated intrapericardial pressure and suggests the possibility of cardiac tamponade.
As a corollary of this, exaggerated right atrial collapse is seen, which is an indication of impeded right atrial filling (Figs. 9.28 and 9.29). This occurs with timing opposite that of right ventricular collapse. It is identifiable on two-dimensional echocardiography, typically from the subcostal or apical four-chamber view. Because the right atrium normally contracts in volume with atrial systole, the degree of right atrial collapse must be quantified with respect to either the magnitude of collapse or the duration for which it remains collapsed. In the presence of marked elevation of intrapericardial pressure, the right atrial wall remains collapsed throughout atrial diastole, and buckles inward, reversing the normal wall curvature. In situations in which a localized effusion is resulting in hemodynamic compromise, one may occasionally encounter isolated compression (usually diastolic) of the left atrium or left ventricle. In cases of advanced intrapericardial pressure elevation, the right-sided chambers may be compressed and underfilled throughout the cardiac and respiratory cycles. This is most commonly seen in acutely developing effusions.

**Doppler Findings in Tamponade**

Doppler interrogation can document exaggerated phasic variation in mitral and tricuspid inflow and aortic and pulmonary outflow. With inspiration tricuspid inflow is augmented and decreases with expiration. Reciprocal changes are seen in mitral inflow. Under normal circumstances, peak velocity of mitral inflow varies by 15% or less with respiration and tricuspid inflow.
by 25% or less. Variation in peak velocity and time velocity integral of aortic
and pulmonary flow profiles normally is less than 10%. In the presence of
hemodynamically significant pericardial effusion, exaggerated ventricular
interdependence develops, respiratory variation in filling is exaggerated
above these thresholds, and, as a consequence, respiratory variation in
outflow tract velocities and time velocity integral is likewise exaggerated
(Figs. 9.30 and 9.31). These Doppler findings are the corollary of a pulsus
paradoxus.
FIGURE 9.27. Parasternal short-axis view recorded at the base of the heart in a patient with a hemodynamically significant pericardial effusion and right ventricular outflow tract collapse. **A:** Recorded at end systole, revealing normal right ventricular outflow tract geometry. **B:** Recorded in early diastole. Note the closed pulmonary valve (*horizontal arrow*). There is definite collapse inward at the right ventricular outflow tract free wall (*vertical arrow*), suggesting that pericardial pressure exceeds right ventricular diastolic pressure at that point in the cardiac cycle. 🎥
FIGURE 9.28. Apical four-chamber view recorded in a patient with a large pericardial effusion and evidence of hemodynamic compromise. Note the dramatic invagination of the right atrial free wall (arrow) in this patient with overt cardiac tamponade. [Video 9-28]
FIGURE 9.29. Subcostal echocardiogram recorded in a patient with a large pericardial effusion and hemodynamic compromise. In this frame, recorded at the end of atrial systole, note the persistent inward collapse of the right atrial free wall, the duration and magnitude of which is better appreciated in the real-time image.  

Video 9-29
Pulsed Doppler imaging of vena caval and hepatic vein flows can also reflect the elevated intrapericardial pressure and altered filling patterns. Normally, vena caval flow occurs in both systole and diastole and is nearly continuous. In the presence of elevated intrapericardial pressure, flow during diastole is truncated and the majority of flow into the heart occurs during ventricular systole. The hepatic vein flow pattern may also reflect the exaggerated respiratory phase dependency of right ventricular filling (Fig. 9.32).
FIGURE 9.30. Doppler recordings of transtricuspid (A) and transmitral (B) inflow velocities recorded in a patient with a hemodynamically significant pericardial effusion and clinical evidence of cardiac tamponade. The inspiratory (I) and expiratory (E) phases are as noted on the respirometer. For the tricuspid valve, note the augmented inflow during inspiration with diminished inflow during expiration. Note the opposite effect seen on the transmitral inflow velocity.
FIGURE 9.31. Doppler flow profile of the pulmonary outflow tract and left ventricular outflow tract recorded in a patient with clinical cardiac tamponade. There is augmented pulmonary flow with inspiration and a reciprocal decrease in left ventricular outflow at the same point in the respiratory cycle. This reciprocal
and phasic variation with respiration is physiologic evidence of exaggerated interventricular interdependence and the underlying phenomenon causing pulsus paradoxus. E, expiration; I, inspiration.

There is a well-defined hierarchy with which these findings occur in hemodynamically significant pericardial effusions. Typically, the earliest feature to be noted is exaggerated respiratory variation of tricuspid inflow. Subsequent to this, pathologic exaggeration in mitral inflow patterns can be noted. Abnormal right atrial collapse typically occurs at lower levels of intrapericardial pressure elevation than does right ventricular outflow tract collapse. Right ventricular free wall collapse is seen only later in the development of elevated intrapericardial pressures. With milder elevations in intrapericardial pressure, right ventricular diastolic collapse may be seen in expiration but not in inspiration when right ventricular filling is augmented. Intermittent collapse is often best documented with M-mode echocardiography (Fig. 9.33). When intrapericardial pressure is elevated and consistently exceeds intravascular pressures, all these findings will be present simultaneously.

There are several instances in which these changes may not be seen. In general, any underlying disease which interferes with normal phasic ventricular interdependence may reduce the magnitude of both clinical findings and echocardiographic abnormalities in cardiac tamponade. Significant left ventricular hypertrophy, resulting in relatively fixed rates of left ventricular filling, may reduce the degree of ventricular interdependence with respiration. Respiratory variation of ventricular outflow and stroke volume, and consequently pulsus paradoxus, may be reduced. In such instances, it is not uncommon to visualize varying degrees of global cardiac underfilling with relatively small ventricular and atrial chambers but without a dramatic respiratory variation in filling (Fig. 9.34). A common reason for echocardiographic findings to be absent is right ventricular hypertrophy, usually due to pulmonary hypertension. In this case, the thick, noncompliant right ventricular wall is not compressed by the relatively modest elevation in pericardial pressure seen in early diastole with active left ventricular relaxation, and both clinical and echocardiographic signs of compromise may be minimal or masked (Fig. 9.35). Often, relative hypotension is present but without pulsus paradoxus. Thickening of the ventricular wall due to
malignancy, an overlying inflammatory response, or an overlying thrombus in hemorrhagic pericarditis may have the same effect. Similarly, because the magnitude of ventricular interaction is directly related to ventricular volume, these signs may be absent in low-pressure tamponade, as may be seen in hypovolemic patients.

**FIGURE 9.32.** Pulsed Doppler imaging of the hepatic vein recorded in a patient with a hemodynamically significant pericardial effusion. Note the loss of forward flow in the hepatic veins during the expiratory (E) phase of the respiratory cycle. Flow out of the hepatic veins is confined exclusively to the early inspiratory (I) phase.

**PERICARDIAL CONSTRICTION**

Pericardial constriction is a relatively uncommon entity in contemporary practice and as a consequence often goes unrecognized. The clinical signs and symptoms of pericardial constriction are often vague and may have been present for several years before the diagnosis is finally established. Historically, the classic form of pericardial constriction was calcific constriction secondary to tuberculous pericarditis, which is rarely
encountered in contemporary practice (except in regions and populations where TB remains prevalent). Many of the classic physical findings and observations in pericardial constriction were derived from patients with this classic type of calcific constriction. It should be emphasized that other forms of constriction may not share all the classic hemodynamic, physical examination, and echocardiographic findings seen in classic calcific constriction. More commonly in today’s practice, constrictive pericarditis is the result of infectious or inflammatory processes such as connective tissue disease or radiation therapy or develops several years after cardiac surgery or trauma. Transient constrictive pathophysiology can follow virtually any form of pericardial inflammation, and transient constrictive physiology can occasionally occur in the course of otherwise self-limited pericarditis, connective tissue disease, or other inflammatory processes, or after cardiac surgery.

FIGURE 9.33. M-mode echocardiogram recorded through the right ventricular outflow tract in a patient with pericardial effusion and evidence of early hemodynamic compromise. In this instance, right ventricular outflow tract collapse is seen only intermittently (arrows) and occurs during expiration, whereas right ventricular filling is less impeded during inspiration.
FIGURE 9.34. Subcostal view recorded in a patient with marked left ventricular hypertrophy and large pericardial effusion. There is persistent underfilling of the right ventricle, which is compressed throughout the respiratory cycle. At the time of this echocardiogram, the patient was normotensive (130/80 mm Hg) and became overtly hypertensive following pericardiocentesis. Note the absence of respiratory variation in the mitral inflow pattern, which correlated with absence of pulsus paradoxus.
FIGURE 9.35. Transthoracic apical four-chamber view recorded in a patient with large pericardial effusion and hypotension on the background of severe pulmonary hypertension and right ventricular hypertrophy, which decreases the tendency to right ventricular collapse. Note the absence of any right-sided compromise.
Anatomically, constriction occurs when there is stiffening of the pericardium. It is typically the parietal pericardium that becomes the constricting force, although variable degrees of visceral pericardial involvement occur and commonly the two pericardial layers become fused. This is often seen in association with demonstrable pericardial thickening, again predominantly involving the parietal pericardium but also with inflammation and stiffening of the visceral pericardium. In classic calcific constrictive pericarditis, the pericardium forms a rigid shell in which the cardiac chambers are encased and therefore are relatively isolated from changes in intrathoracic pressure. This results in a dissociation of intrathoracic and intracardiac pressures and intrathoracic pressure changes are not transmitted to each chamber equally. Typically with the inspiratory decrease in intrathoracic pressure right heart filling is augmented. Because the pulmonary veins are outside the boundary of pericardial constraint, they are subject to the lower pressure which reduces left heart filling. This results in a similar pattern of exaggerated ventricular interdependence as seen with tamponade (Fig. 9.24) and is visually observed as an inspiratory shift of the septum to the left. The process is reversed with expiration. It should be emphasized that the constrictive process may not be uniform and the classic pathophysiology may not always be present, or be present to variable degrees such that not all clinical or echocardiographic findings are uniformly present.

**Echocardiographic Diagnosis**

The diagnosis of constrictive pericarditis requires a combination of clinical and echocardiographic findings. There are no absolutely sensitive and specific echocardiographic or Doppler indicators of constriction; instead, multiple clinical, anatomic, and physiologic observations must be combined to establish the diagnosis. Although pericardial constriction is most often associated with thickening of the pericardium, actual detection of a thickened pericardium is often difficult with echocardiography. If an effusion is also present, and especially if pericardial fluid and pleural fluid are both present, the thickness of the pericardium may be directly visualized by transthoracic or transesophageal echocardiography (Fig. 9.15). When directly visualized,
The normal pericardium is no more than 1 to 2 mm in thickness. Additional indicators of a thickened pericardium include its persistence during gradual damping of an M-mode beam through the posterior left ventricular wall (Fig. 9.21). If calcific pericardial disease is present, ultrasound shadowing may occur and give hints as to the underlying pathology (Figs. 9.16 and 9.36). In many instances, the pericardial space between the visceral and the parietal pericardium may appear filled with a vague echo-dense substance representing a combination of actual pericardial thickening and organized inflammatory pericardial fluid (Figs. 9.37 and 9.38).

While it is often difficult to directly visualize pericardial thickening with echocardiographic techniques, both cardiac computed tomography and cardiac magnetic resonance imaging can provide a high resolution, accurate assessment of direct pericardial anatomy, including quantification of actual pericardial thickness (Figs. 9.22 and 9.23). Computed tomography and standard chest radiography (Fig. 9.39) can be used to confirm the presence of calcification in the pericardium. Chest computed tomography is the most sensitive and comprehensive technique for detection of pericardial calcification (Figs. 9.40 and 9.41). Magnetic resonance imaging is not a reliable technique for detection of calcification and if calcific disease of the pericardium is suspected, cardiac/chest CT will be indicated.

It should be emphasized that not all cases of pericardial constriction will be associated with calcification or even anatomical thickening of the pericardium. Such patients represent a well-defined subset of constrictive pericarditis in which a normal thickness pericardium has become pathologically stiffened and noncompliant, leading to constrictive physiology, highlighting the importance of hemodynamic assessment along with the anatomical assessment. If the two-dimensional imaging findings and Doppler evaluation are consistent with constrictive physiology, confirmation of hemodynamics by catheterization is indicated.
FIGURE 9.36. Transesophageal echocardiogram performed at 0 and 133 degrees in a patient with calcific constrictive pericarditis. In both images, far-field gains have been increased to maximum in spite of which no echorefective targets are appreciable. Note the bright, reflective rim around the pericardium (small arrows) and in the 133-degree view, the echo-dense areas in the right ventricular outflow tract (arrow) resulting in even more marked shadowing. [Video 9-36a] [Video 9-36b]
FIGURE 9.37. Parasternal long-axis view recorded in a patient with acute pericarditis and subsequent consolidation of the pericardial fluid. The central illustration is a parasternal long-axis view revealing consolidated fluid within the pericardial space as noted by the two inward-pointing arrows. Also note the consolidated material adjacent to the left atrium (longer single arrow). In the real-time image note the “shudder” of the ventricular septum suggesting development of constrictive physiology. The side panel at the upper left was recorded 2 weeks earlier at which time the pericardial space was filled predominantly with free pericardial fluid (arrows) with some stranding also noted.
FIGURE 9.38. Apical four-chamber view recorded in a patient with an inflammatory, organized pericardial effusion. The downward-pointing arrow denotes the outer margin of the parietal pericardium. The upward- and leftward-pointing arrows denote a thick rind of organized material within the pericardial space.
Respiratory-dependent interaction of right and left ventricular filling manifests as exaggerated septal position shifts with respiration and is a hallmark of constriction. This can be noted both on M-mode and two-dimensional echocardiography. Use of a respirometer may help identify respiratory-dependent motion. These interventricular septal motion abnormalities are a reflection of respiratory-dependent variations in right and left ventricular volume throughout the cardiac and respiratory cycles. As the total intracardiac volume is limited by the constrictive pericardium, any inspiratory increase in right-sided filling must be accompanied by a reciprocal decrease in left-sided filling. This results in an exaggerated respiratory variation in septal position as noted in Figures 9.42 to 9.45. In the absence of pathologically increased respiratory effort, exaggerated ventricular interdependence may be the most specific echocardiographic indicator of constriction. Exaggerated ventricular interdependence can be documented from many standard transthoracic imaging windows using long capture of multiple cardiac cycles to include both inspiratory and expiratory phases. If clear evidence of exaggerated ventricular interdependence is noted by echocardiography or cardiac magnetic resonance imaging and is consistent with the clinical presentation, the diagnosis can usually be established.
FIGURE 9.39. A chest x-ray performed in a patient with calcific pericarditis. Note the calcified rim of pericardium along the inferoapical boundary of the heart (arrows).
FIGURE 9.40. Chest CT recorded in the same patient depicted in Figure 9.39 showing the high-resolution detection of calcium in the pericardium by cardiac CT (arrows). Note the substantially more obvious calcium on the CT than on the standard chest x-ray.
FIGURE 9.41. Volume-rendered computed tomography of the heart performed in a patient with a history of an acute inflammatory pericarditis occurring 10 years previously. Note the irregularly shaped calcific deposits on the surface of the pericardium (arrows). This view is rotated to visualize the posterior aspect of the heart.

FIGURE 9.42. M-mode echocardiogram recorded in a patient with constrictive pericarditis showing marked phasic respiratory-dependent downward motion of
the ventricular septum (arrow). Note that with inspiration (I), there is expansion of the right ventricular cavity with abrupt posterior motion of the ventricular septum consistent with exaggerated interdependence.

FIGURE 9.43. Apical four-chamber view recorded in a patient with constrictive pericarditis and abnormal motion of the ventricular septum. In this static image, note the normal chamber sizes and configuration of the left ventricle. This diastolic frame was recorded in early inspiration and there is leftward bowing of the septum (arrow). In the real-time image, note the abnormal “shuddering” motion of the ventricular septum in diastole which is a hallmark of pericardial constriction.
FIGURE 9.44. Subcostal short-axis view recorded in a patient with constrictive pericarditis showing marked respiratory-dependent phasic variation in right ventricular size and septal position. The central image was recorded during inspiration and reveals a dilated right ventricle and displacement of the septum toward the left ventricle (arrows). The image in the inset was recorded at end diastole during expiration and reveals normal left ventricular geometry and a smaller right ventricle. This phasic respiratory variation is better appreciated in the real-time image.
FIGURE 9.45. M-mode echocardiogram recorded in a patient with constrictive pericarditis. Note the flat position of the posterior wall during diastole after initial rapid filling. Also note the abnormal motion of the ventricular septum (double arrows). PW, posterior wall.
M-mode abnormalities that have been noted in patients with constrictive pericarditis include relatively abrupt relaxation of the posterior wall with subsequent flattening of endocardial motion throughout the remainder of diastole (Fig. 9.21) and abnormal septal motion (Fig. 9.42). Several different septal motion abnormalities have been noted, many of which mimic conduction disturbances and mild right ventricular volume or pressure overload patterns. Typically, early diastolic notching may be seen, followed by paradoxical and then normal motion of the ventricular septum. Septal motion reflects the competitive filling of the two ventricles. With constriction, the ventricles fill in an alternative fashion and thus produce a “shuddering” pattern of diastolic septal motion when viewed with real-time two-dimensional imaging (Fig. 9.43).

A final indirect sign of constriction is dilation and lack of respiratory variation of the inferior vena cava diameter (Fig. 9.46). This finding will also be seen in any disease state resulting in marked elevation of right atrial pressures.
Doppler Echocardiographic Findings in Constriction

Doppler echocardiography provides direct evidence of the abnormal intracardiac flow relationships in pericardial constriction and is instrumental in establishing the diagnosis. The classic Doppler findings of pericardial constriction are an exaggerated E/A ratio of mitral valve inflow with a short deceleration time and exaggerated respiratory variation in E-wave velocity (Figs. 9.47 and 9.48). Although an elevated E/A ratio with a short deceleration time can be seen in any disease state with restrictive or constrictive physiology, exaggerated respiratory variation is a relatively reliable sign of pericardial constriction. In modern practice and in patients with concurrent primary disease of the ventricle or valves one may see less typical patterns in which there is a normal or reversed E/A ratio with exaggerated respiratory variation or in which only the tricuspid valve inflow reveals classic changes. Exaggerated respiratory variation in constriction will be seen during normal, quiet, nonlabored respiration, whereas an exaggerated respiratory variation in E-wave velocity could be seen in instances of primary respiratory distress as well. Typically, variation of 25% or more in the mitral E-wave velocity between inspiration and expiration has been considered abnormal. The changes noted in atrioventricular valve inflow are maximal with the first several heartbeats after onset of inspiration and occur on a reciprocal basis when the mitral and tricuspid flow patterns are compared. Evaluation of mitral inflow patterns may also reveal exaggerated respiratory variation in the isovolumic relaxation time of the left ventricle. Doppler interrogation of the hepatic veins often reveals an increase in diastolic flow reversal with expiration (Figs. 9.49 and 9.50). M-mode color Doppler of mitral inflow reveals an abnormally steep velocity of propagation of mitral inflow (Fig. 9.51).
FIGURE 9.47. Pulsed Doppler recording of mitral (A) and tricuspid (B) inflow in a patient with constrictive pericarditis. Note the exaggerated respiratory variation of the E-wave velocity and the reciprocal relationship between mitral and tricuspid inflow E-wave velocity, dependent on the phase of the respiratory cycle. Note the augmented velocity during inspiration (I) and reduced velocity during expiration (E) for the tricuspid valve (B) compared with the reverse pattern of mitral inflow (A).
FIGURE 9.48. Pulsed Doppler recording of the mitral (upper panel) and tricuspid (lower panel) valve inflows in a patient with documented calcific constriction. Notice the relatively mild degree of variation in mitral inflow from expiration (E) to inspiration (I) but the dramatic respiratory variation in tricuspid inflow in this patient.
FIGURE 9.49. Pulsed Doppler recording of mitral inflow and hepatic vein flow recorded in a patient with constrictive pericarditis. A: Note the marked respiratory variation in mitral E-wave velocity similar to that depicted in Figure 9.48. B: Note the marked early expiratory (E) reversal of flow in the hepatic vein. This results in marked respiratory variation in forward flow in the hepatic vein. I, inspiration.

**Annular Velocities**
Doppler tissue imaging to determine annular velocities has provided both valuable insight and new diagnostic criteria for constrictive pericarditis. In a normal heart without a constrictive pericardium, the lateral mitral annular...
velocity (e') exceeds that of the medial annulus. The underlying pathophysiology of this is that during systole and diastole there is greater longitudinal motion of the lateral wall and annulus toward the apex than of the medial annulus. With constrictive pericarditis, the thickened constrictive pericardium limits the motion of the lateral annulus but has no effect on the medial annular velocity. There is therefore both preservation of medial velocities and a compensatory augmentation of apex to base shortening of the septum relative to the lateral wall. In a normal situation, the lateral to medial annular e' ratio exceeds 1.2. Reversal of this ratio has been termed “annulus reversus” and has been documented as a reliable sign of constrictive pericarditis with overall accuracy equivalent to or exceeding that of numerous traditional echocardiographic and Doppler findings. Limited studies have also suggested that the lateral medial annular ratio may revert toward normal following successful surgical pericardiectomy (Fig. 9.52).
FIGURE 9.50. Pulsed Doppler recording of mitral inflow (A), tricuspid inflow (B), and hepatic vein flow (C) from a patient with constrictive pericarditis. In this example from a patient with concurrent diastolic dysfunction, note the reduced E/A ratio of mitral inflow with little respiratory variation. **Middle:** There is definite exaggerated respiratory variation of the tricuspid flow. **Bottom:** Note the respiratory dependency of forward flow in the hepatic vein, with flow confined to inspiration (INS) and the expiratory reversal (ER) of flow. EXP, expiration.

![FIGURE 9.50. Pulsed Doppler recording of mitral inflow (A), tricuspid inflow (B), and hepatic vein flow (C) from a patient with constrictive pericarditis.](image)

FIGURE 9.51. Color Doppler M-mode recording in a patient with constrictive pericarditis. Note the very steep velocity of propagation ($V_p$), averaging more than 200 cm/sec in this example. Mitral inflow $V_p$ with this technique may assist in distinguishing constrictive from restrictive physiology.

![FIGURE 9.51. Color Doppler M-mode recording in a patient with constrictive pericarditis.](image)
FIGURE 9.52. Mitral valve inflow (lower panel) and lateral and medial Doppler annular velocities (upper and middle panels) recorded in a patient with constrictive pericarditis. In the mitral inflow, note the E/A ratio of 2.1 and short deceleration time of 114 milliseconds. Note the lateral annular velocity of 15.8 cm/sec and the medial velocity of 21.4 cm/sec indicative of “annulus reversus,” a sign of pericardial constriction.

Many of the anatomical and Doppler-based criteria for the diagnosis of
constrictive pericarditis are dependent on respiratory variation and septal position and Doppler or tissue Doppler velocities. In the presence of atrial fibrillation with a markedly irregular rhythm, beat to beat variability in flow parameters is to be expected and as such assessing the independent impact of respiratory variation may become problematic. While the diagnostic accuracy of E-wave with respiration is diminished (unless there is minimal heart rate variability) in the presence of atrial fibrillation, other observations such as inspiratory septal shift, septal shudder, analysis of hepatic vein flow reversal and annular velocities appear to retain their diagnostic accuracy in the presence of atrial fibrillation.

**Effusive Constrictive Pericarditis**

Effusive constrictive pericarditis represents a combination of constrictive and tamponade physiology. Common causes of effusive constrictive pericarditis are malignancy and radiation therapy; however, it may be seen in any inflammatory pericardial disease. Patients with effusive constrictive pericarditis present with pericardial effusion, often with evidence of marked inflammation (Figs. 9.53 and 9.54). Although hemodynamic embarrassment and tamponade may be present, the thickening of the visceral pericardium may prevent right ventricular or right atrial free wall collapse. This may result in a decreased accuracy of individual echocardiographic and Doppler flow patterns for the diagnosis of hemodynamic compromise. From a clinical standpoint, the diagnosis is often established in a patient with hemodynamic compromise and moderate pericardial effusion in whom jugular vein distention and abnormal hemodynamics persist after pericardiocentesis. After pericardiocentesis, the effusive component resolves and hemodynamics and echocardiographic findings become more consistent with constriction (Figs. 9.53 to 9.55). Cardiac MR or CT can reliably identify the thickened pericardium and MR with gadolinium can document pericardial inflammation (Figs. 9.56 and 9.57).
FIGURE 9.53. Parasternal long-axis views recorded in the same patient depicted in Figures 9.4 and 9.5. These images were recorded after removal of 650 cc of pericardial fluid. Note the small residual pericardial effusion (arrows) which has organized components. In the real-time images, note the “shudder” of the ventricular septum and exaggerated respiratory septal position consistent with constrictive physiology and the diagnosis of effusive constrictive pericarditis.

Video 9-53

Constrictive Pericarditis Versus Restrictive Cardiomyopathy
Both constrictive pericarditis and restrictive cardiomyopathy often present as chronic indolent diseases with evidence of volume overload. When classic anatomic abnormalities such as cardiac amyloid or other infiltrative cardiomyopathy are noted, the distinction is not difficult. More commonly, the differential diagnosis is between an idiopathic restrictive cardiomyopathy and occult constrictive pericarditis. In these cases, it is important to rely on multiple parameters from a comprehensive Doppler and echocardiographic examination to establish the diagnosis. Multimodality imaging with cardiac CT and MRI play an increasingly valuable role in separating these entities. Differentiating features include marked biatrial enlargement in a restrictive cardiomyopathy but relatively normal chamber sizes in constriction. In both instances, an elevated E/A ratio may be noted with a shortened deceleration time. Respiratory variation of the E-wave velocity is increased in constriction, whereas it is normal in restrictive cardiomyopathy. Left ventricular isovolumic relaxation time also shows greater respiratory variation in constriction when compared with restrictive cardiomyopathy. Hepatic vein and superior vena caval blood flow patterns also have distinguishing features. Typically, in patients with constrictive pericarditis, systolic antegrade flow is enhanced with inspiration, whereas in restriction, there is less respiratory variation and diastolic flow typically exceeds systolic flow.
FIGURE 9.54. Parasternal short-axis view recorded in the same patient presented in Figure 9.53. Note the residual small pericardial effusion which has consolidated components suggestive of an inflammatory response. In the real-time image, notice the “shuddering” motion of the ventricular septum most prominent during inspiration and the marked downward septal shift with inspiration, all consistent with constrictive physiology. [Video 9-54]

Video 9-54
FIGURE 9.55. M-mode echocardiogram recorded in the same patient and timing presented in Figure 9.54. On the M-mode, note the abnormal septal motion with an early systolic downward beak (small arrow), and increase in right ventricular size with inspiration (large arrow) consistent with constrictive physiology.
FIGURE 9.56. Cardiac MRI from the patient presenting in Figures 9.53 to 9.55. Note the marked thickening of the parietal pericardium (double headed arrows) and pleural effusions (PL).
FIGURE 9.57. Delayed gadolinium enhancement MRI in the same patient presented in Figure 9.56. Note the marked late delayed gadolinium enhancement throughout the entire pericardium (arrows) indicative of an inflammatory response in the pericardium.

More recently, Doppler tissue imaging of the mitral annulus has been used to differentiate constrictive pericarditis from restrictive cardiomyopathy. In constriction, there is more rapid annular early relaxation (e') compared with restriction where diastolic velocities are diminished to below normal (Fig. 9.58).

A final method for differentiating constrictive from restrictive processes is the velocity of propagation ($V_p$) of mitral inflow determined from mitral color Doppler M-mode imaging. With this technique, the velocity with which the mitral flow moves toward the apex is normal (>55 cm/sec) or frequently exaggerated in constriction, whereas it is pathologically reduced in restriction.

Table 9.4 outlines the expected echocardiographic and Doppler findings in
constriction and restriction. It should be emphasized that no one finding will be 100% accurate and that a clinical diagnosis of either entity should be based on a combination of clinical and echocardiographic findings combined with other methods (e.g., computed tomography, magnetic resonance imaging) to visualize pericardial anatomy.

**FIGURE 9.58.** Composite imaging of a patient with a restrictive cardiomyopathy related to cardiac amyloid. Notice the biatrial enlargement and normal geometry of the left ventricle. In the upper right is a mitral Doppler inflow pattern revealing monophasic filling with a short deceleration time and at the lower right lateral annular velocities which are pathologically reduced to 4 cm/sec. The “bulls-eye” image of global longitudinal strain shows apical sparing, typical for cardiac amyloid.
# Table 9.4

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Constriction</th>
<th>Restriction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial size</td>
<td>Normal</td>
<td>Dilated</td>
</tr>
<tr>
<td>Pericardial appearance</td>
<td>Thick/bright</td>
<td>Normal</td>
</tr>
<tr>
<td>Septal motion</td>
<td>Abnormal</td>
<td>Normal</td>
</tr>
<tr>
<td>Septal position</td>
<td>Left shift with inspiration</td>
<td>Normal</td>
</tr>
<tr>
<td>Mitral E/A</td>
<td>Increased (≥2.0)</td>
<td>Increased (≥2.0)</td>
</tr>
<tr>
<td>Deceleration time</td>
<td>Short (≤160 ms)</td>
<td>Short (≤160 ms)</td>
</tr>
<tr>
<td>Lateral annular e”</td>
<td>Normal</td>
<td>Reduced (≤10 cm/sec)</td>
</tr>
<tr>
<td>Lateral/medial e’ ratio</td>
<td>Medial &gt; lateral</td>
<td>Medial &lt; lateral</td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
<td>Rare</td>
<td>Frequent</td>
</tr>
<tr>
<td>Left ventricular size/function</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Mitral/tricuspid regurgitation</td>
<td>Infrequent</td>
<td>Frequent (TR &gt; MR)</td>
</tr>
<tr>
<td>Isovolumic relaxation time</td>
<td>Varies with respiration</td>
<td>Stable with respiration</td>
</tr>
<tr>
<td>Respiratory variation of mitral E velocity</td>
<td>Exaggerated (≥25%)</td>
<td>Normal</td>
</tr>
<tr>
<td>Color M-mode mitral valve Vp</td>
<td>Increased (&gt;55 cm/sec)</td>
<td>Reduced</td>
</tr>
</tbody>
</table>

*The above table represents an outline of various parameters which can help in differentiating constrictive pericarditis from restrictive cardiomyopathy. It should be emphasized that in the majority of cases there may be discordant data and that the distinction should be based on the overall appearance and not any single factor. In*
complex instances such as combined constriction and restriction following radiation or either entity combined with primary valvular heart disease, many exceptions to these guidelines are anticipated.

MISCELLANEOUS PERICARDIAL DISORDERS

Postprocedural Effusions

Pericardial effusion is not uncommon after cardiac surgery and can range from small, self-limited and clinically inconsequential to larger effusions that cause hemodynamic compromise (Fig. 9.59). Postoperative effusions are most commonly localized to the posterior and lateral aspects of the heart and may be loculated. They may cause isolated, differential compression of one or more chambers, in distinction to a native pericardial effusion, which typically causes hydrodynamic compression of all cardiac chambers equally. Complicating their assessment is the postoperative status of the patient, which often interferes with transthoracic imaging and for whom transesophageal echocardiography may be necessary. It should also be emphasized that a postoperative pericardial effusion is by definition hemorrhagic and that often there will be components of intrapericardial hematoma present as well. Intrapericardial hematoma will have a density similar to that of the myocardium and other mediastinal structures, and a heightened awareness of the possible presence of hematoma within the pericardium is necessary. In evaluating a critically ill patient with a suspected postoperative pericardial effusion or hematoma, it is important to evaluate the size and geometry of all four cardiac chambers and attempt to identify the inflow from the pulmonary veins and superior and inferior vena cava. Loculated effusions and hematoma after cardiac surgery can result in isolated compression of one or more pulmonary veins or of vena caval inflow, either of which can compromise overall cardiac output. Identification of small, underfilled chambers that appear compressed may be indirect evidence of a compressive pericardial hematoma in this setting. This topic is also addressed in Chapter 23.

Perforation of a cardiac chamber is a well-recognized complication of catheterization and other interventional procedures. Virtually any of the four cardiac chambers, great vessels, or veins can be perforated by a catheter-based device. Depending on the site of perforation and the magnitude of leak
from the cardiac chamber to the pericardial space, the consequences may range from inconsequential to rapidly progressive life-threatening cardiac tamponade. In the majority of instances, the perforation will be to a pericardial space not already containing pericardial fluid and therefore relatively noncompliant. This results in a situation where relatively small amounts of pericardial fluid result in a rapid increase in intrapericardial pressure and subsequently in hemodynamic compromise. Because of the rapid elevation in pericardial pressure, several of the classic signs of cardiac tamponade may be absent. Hypotension will be a reliable indicator of hemodynamic compromise. With echocardiographic scanning, one may note reduction in cavity size of the right ventricle as well as an underfilled left ventricle (Fig. 9.60) and for reasons previously alluded to, respiratory interdependence of filling, flow, and forward output may not be apparent in this situation.

**FIGURE 9.59.** Parasternal transthoracic echocardiogram recorded in a patient with a compressive pericardial hematoma after bypass surgery. Note the small left ventricular cavity and the abnormal contour of the right ventricular free wall (RVFW). In this mid-diastolic frame, the right ventricular free wall is compressed
toward the ventricular septum (arrows), compromising filling of the right ventricle.

FIGURE 9.60. Subcostal two-dimensional echocardiogram recorded in a patient with acute hemodynamic collapse at the time of pacemaker insertion. The preprocedure echocardiogram had not shown a pericardial effusion and it showed normal sizes of the right ventricle and left ventricle. In this subcostal view, note the small pericardial effusion (long arrow) and in this end-diastolic frame the pathologically compressed right ventricle is small left ventricular internal dimension indicative of global underfilling of the four cardiac chambers.
Echocardiography-Guided Pericardiocentesis

Echocardiography plays several valuable roles with respect to therapeutic pericardiocentesis. Obviously, the first role is in determining the presence and distribution of a pericardial effusion and the presence of hemodynamic compromise. If pericardiocentesis is contemplated, multiple echocardiographic imaging windows should be used to determine the distribution of the fluid. Specifically, the distribution and depth from the surface of the chest at which contact with the fluid is anticipated by the pericardiocentesis needle should be determined (Fig. 9.61). Some laboratories perform continuous echocardiographic guidance of pericardiocentesis and attempt to visualize the pericardiocentesis needle as it enters the pericardial cavity (Fig. 9.62). Although this may be helpful to avoid cardiac damage in a relatively small effusion, it plays little incremental role in larger pericardial effusions, which are usually the target for a therapeutic pericardiocentesis. If the location of a pericardiocentesis needle is in question, agitated saline can be injected to confirm the location of the needle in the pericardial space (Fig. 9.63).

After pericardiocentesis, two-dimensional echocardiography can be used to determine the completeness of fluid removal, to monitor for reaccumulation, and assess for complications of attempted drainage (Fig. 9.64). A syndrome of acute right heart dilation is occasionally seen after large-volume pericardiocentesis. This probably occurs when a large intravascular volume that had been sequestered outside the cardiac chambers
is suddenly allowed unlimited entry into the right heart. This can result in acute right heart dilation with clinical evidence of mild right heart failure (Fig. 9.65). This syndrome is typically self-limited.

FIGURE 9.61. Echocardiogram recorded from the subcostal position in a patient with a moderate pericardial effusion. Note the 2-cm distance between the pericardium and right ventricular free wall (arrows), implying a significant distance between the pericardium and the heart, which may confer a decreased risk of pericardiocentesis if approached from the subcostal position.

Video 9-61
FIGURE 9.62. Apical four-chamber view recorded in a patient with a large pericardial effusion and cardiac tamponade. Ultrasound guidance is being used as a needle is placed into the pericardial space. The needle is seen as a bright echo density (arrow) lateral to the right ventricular free wall.
FIGURE 9.63. Parasternal long-axis view recorded in a patient with a large pericardial effusion undergoing pericardiocentesis. In A, note the large, clear free space consistent with pericardial effusion. In B, agitated saline has been injected into the pericardial space resulting in a cloud of contrast targets, thus confirming the location of the pericardiocentesis needle.

FIGURE 9.64. Transthoracic echocardiogram recorded in a patient with a large
pericardial effusion urgently drained, and with subsequent hemodynamic deterioration while a pericardial drain is in place. In the parasternal short-axis view (A), note the large loculated effusion anterolateral to the heart with consolidated components (arrows). B is a parasternal long-axis view, again showing an anterior pericardial effusion with consolidated components representing thrombus. Note the location of the pericardial drain (arrow) within the pericardial thrombus, explaining the failure to fully drain the pericardial effusion. ▶

Video 9-64a

Video 9-64b
FIGURE 9.65. Apical four-chamber view recorded immediately after drainage of a large pericardial effusion. Note the acute dilation of the right heart and reduced right ventricular systolic function. This four-chamber view was recorded in the same patient presented in Figure 9.5.
FIGURE 9.66. Apical four-chamber view recorded in a patient with acquired absence of the pericardium related to a penetrating chest injury. Note the highly unusual elongated shape of the right ventricle, denoted by *small arrows*. The apical portion of the right ventricle wraps around the apex of the left ventricle, and its more proximal portion prolapses beyond the plane of the mitral annulus (*longer arrow*). This is a result of herniation of the right ventricle through a pericardial rent occurring as a result of trauma. CT scans of the same patient are presented in Figure 9.68.
FIGURE 9.67. Cardiac CT recorded in the same patient presented in Figure 9.66. A reveals a continuous stripe of pericardial calcification over the apex (arrows) of the left and right ventricles in a patient with calcific pericarditis related to prior trauma. B was recorded at a slightly inferior location and reveals discontinuity (arrows) of the pericardial calcium, related to acquired absence of pericardium in that area and the herniation of the right ventricle through the pericardial rent.

FIGURE 9.68. Subcostal images recorded in a patient incidentally noted to have an abnormal cardiac contour or on chest x-ray. Note the large oval-shaped extracardiac space adjacent to the right atrioventricular groove (arrows). The
Absence of the Pericardium

Congenital absence of the pericardium can occur in either a partial or, less commonly, a complete form. It is often asymptomatic; however, in the partial form, the left atrial appendage or, less commonly, the right atrial appendage may herniate through the pericardial defect and become strangulated, resulting in symptoms. Because of the lack of pericardial constraint on cardiac chamber size and shape, there may be an abnormal contour of the cardiac silhouette on a chest radiograph and mild degrees of right atrial and right ventricular dilation. Abnormal and frequently paradoxical ventricular septal motion has also been reported. On occasion trauma or partial pericardiectomy may allow herniation of a cardiac structure beyond the pericardial space. Figure 9.66 was recorded in a patient several years following a stab wound to the chest and illustrates herniation of the right ventricle through a tear in the pericardium which has calcified. Note the anatomical detail of the calcified pericardium afforded by the cardiac CT in this same patient (Fig. 9.67).
FIGURE 9.69. Transesophageal echocardiogram recorded in the same patient presented in Figure 9.68. The central image is a bicaval view depicting the left and right atria. Note the echolucent, oval-shaped mass adjacent to the right atrium (arrows). The panel at the lower right was recorded after injection of intravenous saline and demonstrates opacification of the right atrium and superior vena cava but absence of contrast within the echo-free space adjacent to the right atrium, demonstrating lack of continuity with the right heart. Video 9-69a
**Pericardial Cysts**

Pericardial cysts are benign developmental anomalies that most commonly occur near the costophrenic angle. They appear as a loculated echo-free space adjacent to the cardiac border, most commonly near the right atrium (Fig. 9.68). They may distort the normal shape of the atrium. The diagnosis is best confirmed by computed tomography or magnetic resonance imaging (Fig. 9.69). Additional echocardiographic evaluation should include contrast echocardiography to exclude an anomalous systemic vein that may also present as an unusually located echo-free structure. Color flow and pulsed Doppler interrogation at low-velocity settings can be used to ensure that there is no phasic flow within the structure.

**Suggested Readings**

**GENERAL PRINCIPLES**

**EFFUSION AND TAMponade**
Armstrong WF, Schilt BF, Helper DJ, Dillon JC, Feigenbaum H. Diastolic collapse of the right


**Constrictive Pericarditis**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
The aortic valve is a remarkable structure—compliant enough to open fully in less than 25 ms, strong enough to withstand a 70 to 80 mm Hg pressure gradient without leaking, and durable enough to open and close over three billion times over the course of a lifetime. It is composed of three cusps of equal size, each of which is surrounded by a sinus. The cusps are separated by three commissures and supported by a fibrous annulus. Each cusp is crescent shaped and capable of opening fully to allow unimpeded forward flow, then closing tightly to prevent regurgitation. The free edge of each cusp curves upward from the commissure and forms a slight thickening at the tip or midpoint, called the node of Arantius (Fig. 10.1). When the valve closes, the three nodes meet in the center, allowing coaptation to occur along three lines that radiate out from this center point. Overlap of valve tissue along the lines of closure produces a tight seal and prevents backflow during diastole. When viewed from a conventional echocardiographic short-axis projection, these three lines of closure are recorded as a Y shape.

Behind each cusp is its associated sinus of Valsalva. The sinuses represent outpouchings in the aortic root directly behind each cusp. They function to support the cusps during systole and provide a reservoir of blood to augment coronary artery flow during diastole. The sinus and its corresponding cusp share the same name. The left and right coronary arteries arise from the left and right sinuses, respectively, and are associated with the left and right aortic cusps. The third, or noncoronary sinus, is posterior and rightward, just above the base of the interatrial septum, and is associated with the noncoronary aortic cusp. At the superior margin of the sinuses, the aortic root narrows at the sinotubular junction.

Diseases of the aortic valve may be either congenital or acquired and may
produce either stenosis or regurgitation or a combination of the two. The most common causes of acquired aortic valve disease in adults are degenerative, rheumatic, and infective. Diseases of the aorta may also affect aortic valve function. Subaortic obstruction may also occur, either due to hypertrophic cardiomyopathy (see Chapter 18) or membranous and fibromuscular subaortic stenosis (see Chapter 19).

**BICUSPID AORTIC VALVE**

Bicuspid aortic valve (BAV) is one of the most common congenital conditions, occurring in 1% to 2% of the population, with a significant male predominance. Normally, the developing valve tissue divides into three equal-sized cusps, with a commissure separating each adjacent pair. BAV is most often due to a failure of the developing valve to divide properly, leading to partial or complete fusion of two adjacent cusps and absence of the associated commissure. As such, a range of morphology is possible (Fig. 10.2). In most cases, the fusion results in two unequally sized cusps, with an elliptical orifice. The fused cusps are joined by a fibrous raphe where the commissure would normally exist. Most often, the right and left cusps are involved. Less commonly, two cusps of equal size and shape are present, without the associated raphe. The orientation of the two cusps may be either vertical or horizontal, depending on which two cusps fail to separate.

Given the spectrum of morphologies, it is not surprising that the natural history of BAV is also quite variable. In most patients, BAV leads to either valvular stenosis or regurgitation, with stenosis being more common. Rarely, individuals with BAV reach their 80s or 90s without significant valvular dysfunction. Most patients, however, develop symptomatic valve disease in early adulthood. Endocarditis and aortic dissection are other potential sequelae of BAV.
FIGURE 10.1. A normal aortic valve. Both images were recorded during diastole. 
A: The long-axis view demonstrates the appearance of a typical normal aortic valve in the closed position. B: The same valve is demonstrated from the short-axis view. Note that, because of shadowing and lateral resolution, the coaptation line between the left and noncoronary cusps is not visualized. [Video 10-1]
FIGURE 10.2. Two examples of bicuspid aortic valve are shown. On top (A), the systolic (sys) and diastolic (dias) panels demonstrate a vertically oriented coaptation line between the two equal-sized cusps and an oval-shaped systolic orifice. In the bottom panels (B), partial fusion of two cusps with calcification (arrow) is seen at 5 o'clock position, resulting in functionally bicuspid valve.
Echocardiography is both sensitive and specific for the diagnosis of BAV. In the long-axis view, the aortic cusps dome during systole, an important clue to the presence of BAV (Fig. 10.3). However, the doming may be subtle or partially obscured by the aortic walls. In addition, the preserved mobility of the basal and midportion of the two cusps can be deceiving leading to a missed diagnosis. To avoid this, a properly oriented short-axis view provides the best opportunity to establish a diagnosis of BAV (see Fig. 10.2). During diastole, the normal “Mercedes-Benz” appearance of the three closed leaflets is replaced by a linear, slit-like commissure, separating the two abnormal cusps. Recording such an appearance generally establishes the diagnosis although both false-positive and negatives are possible. Image quality and proper short-axis plane alignment are essential for a correct diagnosis. In addition, the variability in morphology can also create challenges, especially when partial fusion of two cusps (with an incomplete commissure) is present. Confirming the presence of a BAV is most challenging in older patients with heavily calcified leaflets and reduced cusp excursion, where distinguishing degenerative (tricuspid) aortic stenosis from BAV may be impossible. When transthoracic echocardiography is inadequate, especially due to image quality, transesophageal echocardiography is often confirmatory (Fig. 10.4).

BAV is often associated with connective tissue abnormalities that affect the ascending aorta. In most patients, both aortic annulus and aortic root dimensions are larger than normal and the risk of ascending aortic aneurysm formation and/or dissection is increased. Effacement of the sinotubular
junction is a characteristic finding. Aortic coarctation is another commonly associated congenital anomaly.

Echocardiography is extremely useful for assessment of the proximal aorta in such patients. From the long-axis plane, the dimensions of the aortic annulus, sinuses, sinotubular junction, and proximal ascending aorta should be obtained (Fig. 10.5). Although any portion of the ascending aorta can be diluted, typically in BAV, the sinuses are spared and the dilation begins above the sinotubular junction and extends to a variable degree toward the arch. If dissection is a concern, careful interrogation of the proximal aorta using a combination of two-dimensional imaging and color flow may permit visualization of the dissection flap (Fig. 10.6).

**FIGURE 10.3.** In the long axis, a bicuspid aortic valve often demonstrates mobility but will appear to dome in systole (arrows).
FIGURE 10.4. Two examples of bicuspid aortic valve recorded with transesophageal echocardiography are shown. In A, diastolic frames show thickening and asymmetry of coaptation. The short-axis view demonstrates two cusps. In B, an eccentric jet of aortic regurgitation is shown.
Because of the importance of aortopathy in patients with BAV, MRI and CT play an important role in evaluation (Fig. 10.7). Although transthoracic echocardiography can evaluate the proximal ascending aorta in most adults, transesophageal echo, MRI, or CT are often necessary for a complete assessment. In addition, these imaging modalities provide detailed evaluation of the aortic valve anatomy, especially important when ultrasound image quality is technically difficult.

**AORTIC STENOSIS**

Although obstruction to left ventricular outflow can occur at multiple levels, valvular aortic stenosis is most common. Congenitally abnormal valves may be stenotic at birth or may develop both stenosis and regurgitation over time. Typically, such valves are bicuspid, as described in the previous section. They demonstrate systolic “doming” and tend to become functionally abnormal during adolescence or early adulthood (Fig. 10.3). This form of aortic valve disease was described above and is covered further in Chapter 19.

Many cases of aortic stenosis are acquired, that is, the valves are normal at birth but become gradually dysfunctional over time. This includes both rheumatic and degenerative causes of aortic stenosis. In recent years, with the decline in the incidence of rheumatic heart disease, most adults with AS have either BAV or degenerative disease. The probability of one versus the other depends on the age of the patient, with BAV predominating through young adulthood and degenerative AS becoming more likely above age 60 years. The goals of the echocardiographic evaluation of this condition include establishing a diagnosis, quantifying severity, assessing left ventricular function, and excluding other conditions that may coexist with aortic stenosis.

Recent valvular heart disease guidelines have emphasized the utility of a classification scheme with four stages (A to D). This is especially helpful in managing and planning interventions in patients with valve disease. The staging system takes into account: (1) risk factors for developing stenosis; (2) presence or absence of symptoms; (3) severity of disease; (4) response of the left and right ventricle; and (5) impact on the systemic and pulmonary
circulations. The stages of aortic stenosis are summarized in Table 10.1. As is very evident, this classification of the stages of disease relies heavily on echocardiographic findings, but in the context of clinical management.

FIGURE 10.5. Aortic root dilation is commonly associated with bicuspid aortic valve. In A, a transthoracic echocardiogram shows a calcified aortic valve with moderate dilation and effacement of the sinotubular junction. In B, a transesophageal echocardiogram shows systolic doming of the valve with marked dilation of the sinuses and proximal aorta.

FIGURE 10.6. An example of aortic dissection (type A) occurring in a patient with a bicuspid valve and aortopathy is provided. In A, the aortic cusps are indicated
by the *small arrowheads*, and the dissection flap by the *larger arrows*. In B, during diastole, the flap herniates through the aortic valve into the left ventricular outflow track. C shows the aortic regurgitation jet (*arrow*) in the diastolic frame and the connection between true and false lumen (*arrow*) in the systolic frame.

Video 10-6a

Video 10-6b
FIGURE 10.7. MRI from a patient with a bicuspid aortic valve. In A, contrast imaging shows the doming of the valve and the systolic jet into the Ao indicated by the arrowhead. The sinuses and proximal aorta are well visualized using MRI. In B, a short-axis view shows the two cusps with a slit-like orifice in between.

**Table 10.1** STAGES OF AORTIC STENOSIS

<table>
<thead>
<tr>
<th>Stage</th>
<th>Definition</th>
<th>Symptoms</th>
<th>Valve Anatomy</th>
<th>Hemodynamics</th>
<th>Cons</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>At risk for AS</td>
<td>None</td>
<td>Bicuspid AV AV sclerosis</td>
<td>$V_{\text{max}} &lt;2 \text{ m/s}$</td>
<td>None</td>
</tr>
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<td>Column</td>
<td>Condition</td>
<td>Symptoms</td>
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<td>Pressure Gradient</td>
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<td>-----------</td>
<td>----------</td>
<td>---------------</td>
<td>-------------------</td>
<td>-----------------------</td>
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<tr>
<td><strong>B</strong></td>
<td>Progressive AS</td>
<td>None</td>
<td>Mild to moderate calcification Rheumatic changes/commissural fusion</td>
<td>Mild AS (mean PG &lt;20 mm Hg) Moderate AS (mean PG 20–39 mm Hg)</td>
<td>Mild diastolic dysfunction Normal EF</td>
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<tr>
<td><strong>C1</strong></td>
<td>Asymptomatic severe AS</td>
<td>None</td>
<td>Severe leaflet calcification Reduced cusp mobility</td>
<td>Mean PG &gt;40 mm Hg AV area ≤1.0 cm² Very severe AS (Vmax &gt;5 m/s or mean PG ≥60 mm Hg)</td>
<td>LV diastolic dysfunction Mild LVH Normal EF</td>
</tr>
<tr>
<td><strong>C2</strong></td>
<td>Asymptomatic severe AS with LV dysfunction</td>
<td>None</td>
<td>Severe leaflet calcification Reduced cusp mobility</td>
<td>Mean PG &gt;40 mm Hg AV area ≤1.0 cm²</td>
<td>EF &lt;50%</td>
</tr>
<tr>
<td><strong>D1</strong></td>
<td>Symptomatic severe high-gradient AS</td>
<td>Dypnea, exercise intolerance, exertional angina, syncope/presyncope</td>
<td>Severe leaflet calcification Reduced cusp mobility</td>
<td>Mean PG &gt;40 mm Hg AV area ≤1.0 cm²</td>
<td>LV diastolic dysfunction LVH Pulmonary hypertension</td>
</tr>
<tr>
<td><strong>D2</strong></td>
<td>Symptomatic severe low-flow/low gradient AS with reduced EF</td>
<td>Dyspnea, heart failure, angina, syncope</td>
<td>Severe leaflet calcification and severely reduced leaflet motion</td>
<td>AV area ≤1.0 cm² with resting Vmax &lt;4 m/s or mean PG &lt;40 mm Hg DSE shows AVA &lt;1.0 cm² with Vmax ≥4 m/s</td>
<td>LV diastolic dysfunction LVH EF &lt;50%</td>
</tr>
<tr>
<td><strong>D3</strong></td>
<td>Symptomatic severe low-gradient AS with normal EF (paradoxical low-flow severe AS)</td>
<td>Dyspnea, heart failure, angina, syncope</td>
<td>Severe leaflet calcification and severely reduced leaflet motion</td>
<td>AV area ≤1.0 cm² with Vmax &lt;4 m/s or mean PG &lt;40 mm Hg Stroke volume index &lt;35 mL/m² measured when patient is normotensive</td>
<td>Increased relative wall thickness Small LV chamber low stroke volume Restrictive diastolic filling EF ≥50%</td>
</tr>
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</table>

AV, aortic valve; AS, aortic stenosis; EF, ejection fraction; LV, left ventricle; LVH, left ventricular hypertrophy; PG, pressure gradient.

Appropriate Use Criteria, which were updated in 2011, provide guidance in the proper application of echocardiography to patients with known or suspected aortic valve disease (Table 10.2). Echocardiography is considered “appropriate” when used for the initial evaluation of known or suspected aortic stenosis, for the routine annual evaluation of asymptomatic severe aortic stenosis, and for the reevaluation of aortic stenosis if there is a change in clinical status. It is considered “rarely appropriate” to perform echocardiography for the routine annual reevaluation of asymptomatic mild aortic stenosis, unless there is a change in clinical status or to guide therapy. Absent a change in clinical status, a routine surveillance echocardiogram is considered appropriate every 3 years for mild aortic valve disease and yearly for moderate or severe disease.

These are reasonable recommendations based on available evidence, known natural history data, and expert consensus panel opinion. They underscore several important factors including: (1) the proper timing of reevaluation; (2) the expected rate of progression of disease; and (3) the importance of symptoms in patient management. Appropriate Use Criteria cannot provide guidance for all possible clinical scenarios and individual judgment must be used to manage patients. Current criteria do not, for example, take into account the important confounding effects of left ventricular dysfunction or coexisting coronary disease.

**Role of 2D Echocardiography**

The *qualitative* diagnosis of aortic stenosis relies heavily on two-dimensional echocardiography. By observing the opening and closing of the valve, the presence or absence of valvular stenosis can be determined. However, the *quantitative* assessment of aortic stenosis, that is, severity, requires measurement of the transvalvular gradient, and thus relies on Doppler (discussed below).

In normal subjects, the aortic valve cusps appear thin and delicate and consist of three approximately equal-sized cusps (Fig. 10.1). In the long-axis view, the cusps open rapidly in systole and appear as linear parallel lines close to the walls of the aorta (Fig. 10.8). With the onset of diastole, they
come together and are recorded as a faint linear density within the plane of the aortic annulus. Because the velocity of valve motion during opening and closing is high relative to the frame rate of most echocardiographic systems, the normal aortic valve is usually visualized either fully opened or closed but rarely in any intermediate position. In the basal short-axis view, the three aortic cusps can be visualized within the annulus during diastole (Fig. 10.9). The three lines of coaptation can be recorded, normally forming a Y (sometimes referred to as an inverted Mercedes-Benz sign). With the onset of systole, the cusps open out of the imaging plane, providing a view of the aortic annulus. The short-axis perspective is most helpful to determine the number of cusps and whether fusion of one or more commissures is present.

With acquired valvular aortic stenosis, the cusps become thickened and restricted (Fig. 10.10). Their position during systole is no longer parallel to the aortic walls, and the edges are often seen to point toward the center of the aorta. In severe cases, a near total lack of mobility may be present and the anatomy may become so distorted that identification of the individual cusps is impossible. Unfortunately, attempts to quantify the degree of stenosis based on two-dimensional echocardiographic findings have been unsuccessful. However, useful qualitative information is almost always present. For example, thickened cusps that display preserved mobility define aortic sclerosis (typically associated with a peak Doppler velocity of \( \leq 2.5 \) m/s). Conversely, heavily calcified cusps with little or no mobility suggest severe stenosis. If one cusp demonstrates full excursion, critical aortic stenosis can usually be excluded. Figure 10.11 is an example of mild aortic stenosis. Although the diagnosis of aortic stenosis is apparent by two-dimensional imaging, the degree of severity can only be estimated. In the example, the cusps are thickened and exhibit restricted mobility. However, Doppler examination revealed only mild stenosis with a maximal pressure gradient of approximately 28 mm Hg. In this example, based solely on two-dimensional appearance, overestimation of severity would be likely. Figure 10.12 is of a patient with heart failure and moderate left ventricular dysfunction. Also note that the aortic valve is severely calcified with markedly restricted systolic mobility.
### Table 10.2
**APPROPRIATE USE CRITERIA FOR AORTIC VALVE DISEASE**

<table>
<thead>
<tr>
<th>Indication</th>
<th>Murmur</th>
<th>Score (1–9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>34</td>
<td>Initial evaluation when there is a reasonable suspicion of valvular or structural heart disease</td>
<td>A (9)</td>
</tr>
<tr>
<td>37</td>
<td>Reevaluation of known valvular heart disease with a change in clinical status or cardiac examination or to guide therapy</td>
<td>A (9)</td>
</tr>
<tr>
<td>36</td>
<td>Reevaluation in a patient without valvular disease on prior echo and no change in clinical status or cardiac examination</td>
<td>I (1)</td>
</tr>
<tr>
<td>39</td>
<td>Routine surveillance (&gt;3 yrs) of mild valvular stenosis without a change in clinical status or cardiac examination</td>
<td>A (7)</td>
</tr>
<tr>
<td>41</td>
<td>Routine surveillance (&gt;1 yr) of moderate or severe valvular stenosis without a change in clinical status or cardiac examination</td>
<td>A (8)</td>
</tr>
<tr>
<td>38</td>
<td>Routine surveillance (&lt;3 yrs) of mild valvular stenosis without a change in clinical status or cardiac examination</td>
<td>I (3)</td>
</tr>
<tr>
<td>40</td>
<td>Routine surveillance (&lt;1 yr) of moderate or severe valvular stenosis without a change in clinical status or cardiac examination</td>
<td>I (3)</td>
</tr>
<tr>
<td>46</td>
<td>Routine surveillance (&gt;1 yr) of moderate or severe valvular regurgitation without a change in clinical status or cardiac examination</td>
<td>A (8)</td>
</tr>
<tr>
<td>42</td>
<td>Routine surveillance of trace valvular regurgitation</td>
<td>I (1)</td>
</tr>
<tr>
<td>43</td>
<td>Routine surveillance (&lt;3 yrs) of mild valvular regurgitation without a change in clinical status or cardiac examination</td>
<td>I (2)</td>
</tr>
<tr>
<td>44</td>
<td>Routine surveillance (&gt;3 yrs) of mild valvular regurgitation without a change in clinical status or cardiac examination</td>
<td>U (4)</td>
</tr>
<tr>
<td>45</td>
<td>Routine surveillance (&lt;1 yr) of moderate or severe valvular regurgitation without a change in clinical status or cardiac examination</td>
<td>U (6)</td>
</tr>
<tr>
<td>63</td>
<td>Evaluation of the ascending aorta in the setting of known or suspected connective tissue disease or genetic condition that predisposes to aortic aneurysm or dissection</td>
<td>A (9)</td>
</tr>
<tr>
<td>64</td>
<td>Reevaluation of known ascending aortic dilation or history of aortic dissection to establish a baseline rate of expansion or when the rate of expansion is excessive</td>
<td>A (9)</td>
</tr>
<tr>
<td>65</td>
<td>Reevaluation of known ascending aortic dilation or history of aortic dissection with a change in clinical status or cardiac examination or when findings may alter management or therapy</td>
<td>A (9)</td>
</tr>
</tbody>
</table>

**Native valvular stenosis**

**Native valvular regurgitation**

**TTE for evaluation of aortic disease (e.g., BAV)**

**TEE as initial or supplemental test**
Evaluation of valvular structure and function to assess suitability for and assist in planning of an intervention  

A (9)

To diagnose infective endocarditis with a moderate or high pretest probability  

A (9)

To diagnose infective endocarditis with a low pretest probability  

I (3)

**Chronic valvular disease—asymptomatic with stress echo**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>181</td>
<td>Moderate aortic stenosis</td>
</tr>
<tr>
<td>182</td>
<td>Severe aortic stenosis</td>
</tr>
<tr>
<td>187</td>
<td>Moderate aortic regurgitation</td>
</tr>
<tr>
<td>188</td>
<td>Severe aortic regurgitation with LV size and function not meeting surgical criteria</td>
</tr>
</tbody>
</table>

**Chronic valvular disease—symptomatic with stress echo**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>193</td>
<td>Evaluation of equivocal aortic stenosis, evidence of low cardiac output or LV dysfunction (with dobutamine only)</td>
</tr>
<tr>
<td>192</td>
<td>Severe aortic stenosis</td>
</tr>
</tbody>
</table>


Transesophageal echocardiography is excellent for determining the morphology of abnormal aortic valves. From a short-axis view at the level of the valve orifice, direct planimetry of the valve area is possible in some patients (Fig. 10.13). Limitations of this approach include the irregular, three-dimensional (3D) nature of the orifice and the shadowing effect of a calcified valve and root. As a result, the technical challenges of this approach are considerable and it is not routinely performed.

3D echocardiography may provide some advantages in this regard, specifically by offering more accurate visualization of the stenotic orifice (Fig. 10.14). Several studies have now confirmed the feasibility of this approach. However, the shadowing effect of calcified cusps remains a limitation. In addition, the challenges of precise planimetry of a very small area, where even small absolute errors maybe significant, must be taken into account.

The simultaneous assessment of left ventricular function is important because of its prognostic and management implications. In addition, reduced left ventricular function alters the relationship between transvalvular pressure
gradient and aortic valve area, thereby complicating the quantitative determination of severity. Other related factors that must be evaluated include the presence and extent of proximal aorta dilation, coexisting mitral valve disease, a measurement of pulmonary artery pressure, and coexisting coronary artery disease.

**Doppler Assessment of Aortic Stenosis**

The Doppler assessment of aortic stenosis begins with the determination of the maximal jet velocity through the stenotic valve from which the peak instantaneous gradient is derived using the simplified Bernoulli equation. This approach has been validated both in vitro and in clinical situations. It has proved to be a practical, noninvasive method for determining the pressure gradient across the aortic valve, correlating well with simultaneous measurements obtained by invasive means.

**FIGURE 10.8.** A normal aortic valve is shown during diastole in the closed position (A) and during systole in the open position (B). Note how the cusps open fully during systole, parallel to the walls of the aorta.
An accurate Doppler assessment of aortic stenosis depends on one’s ability to record the maximal jet velocity through the stenotic orifice (Fig. 10.15). As blood accelerates through the valve, peak velocity coincides temporally with the maximal pressure gradient. Peak velocity usually occurs in mid systole. As aortic stenosis worsens, velocity tends to peak later in systole and the shape becomes more rounded and less peaked. Late-peaking jets are also characteristic of dynamic subaortic stenosis, as occurs in hypertrophic cardiomyopathy (Fig. 10.16). Multiple windows, including the apical five chamber, suprasternal, and right parasternal, should be used in an attempt to align the Doppler beam with the direction of flow of the stenotic jet. Failure to achieve parallel alignment will result in underestimation of true velocity. Both imaging and nonimaging continuous-wave transducers should be used. Because the direction of jet flow is difficult to predict from two-dimensional imaging, color Doppler imaging may be used to improve alignment. Careful manipulation of the transducer position to achieve optimal alignment is recommended. In practice, a thorough and patient interrogation using all available echocardiographic windows is undertaken to record the highest-velocity signal possible. The highest jet velocity obtained, regardless of location, should then be used for calculation of the gradient. By carefully adjusting patient position and instrument gain, both the full envelope and the peak velocity of the stenotic jet can be obtained. Figure 10.17 illustrates the importance of using all available echocardiographic views. Data from two patients are shown. In panel A, the peak velocity of the aortic jet is 4.3 m/s.
from the apex, but 5.2 m/s from the right parasternal window (panel B). The mean gradient would be underestimated by 20 mm Hg if the lower velocity had been used. In the second patient, the opposite occurs, with a much higher velocity obtained from the apical window (panel D) compared to the right parasternal border (panel C). In this case, the mean gradient was recorded as 49 mm Hg from the right parasternal view, but 84 mm Hg from the apex.

From the Doppler recording, the peak instantaneous and mean pressure gradient can be determined from the simplified Bernoulli equation (Fig. 10.18). The maximal gradient is derived from the equation:

\[
\Delta P \text{ (in mm Hg)} = 4v^2 \quad \text{[Eq. 10.1]}
\]

where \( v \) equals the maximal jet velocity expressed in meters per second. This represents a significant simplification of the complete Bernoulli equation. For example, it assumes that the distal velocity is sufficiently greater than the proximal velocity that the latter can be ignored. However, in cases where the proximal velocity is greater than 1.5 m/s and the distal velocity is only modestly elevated (less than 3.5 m/s), the proximal velocity cannot be ignored and the more complete form of the equation should be used:

\[
\Delta P = 4(v_{\text{max}}^2 - v_{\text{proximal}}^2) \quad \text{[Eq. 10.2]}
\]

Situations in which this is relevant include severe aortic regurgitation (due to high forward stroke volume) or when stenoses appear in series, such as combined valvular and subvalvular stenosis.

The mean pressure gradient is most often obtained by planimetry of the Doppler envelope, which allows the computer to integrate the instantaneous velocity data and provide a mean value. It should be emphasized that mean gradient cannot be obtained by squaring the mean velocity. Because of the nearly linear relationship between mean gradient and maximal gradient, the mean pressure gradient can also be estimated from the formula:

\[
\Delta P_{\text{mean}} = \frac{\Delta P_{\text{max}}}{1.45} + 2 \text{ mm Hg} \quad \text{[Eq. 10.3]}
\]

Stated differently, Equation 10.3 suggests that mean gradient is approximately two-thirds of the peak instantaneous gradient. Both mean and peak gradients should be reported.
The accuracy of the Bernoulli equation to quantify aortic stenosis pressure gradients is well established. Selective studies that have validated the modified Bernoulli equation against invasive hemodynamic measurements are shown in Table 10.3. In general, Doppler gradients tend to be slightly higher than corresponding values obtained in the catheterization laboratory. Such differences are not due to the inaccuracy of either technique but most likely the result of pressure recovery, which is discussed in detail in Chapter 8. In the setting of native valve aortic stenosis, some recovery of pressure downstream from the vena contracta can be expected. This occurs as the jet expands and decelerates downstream from the vena contracta resulting in a lower net pressure gradient compared to peak pressure gradient. The net gradient is measured in the catheterization laboratory, typically as the difference in pressure between the left ventricle and ascending aorta. The peak pressure gradient is derived from continuous-wave Doppler by measuring the highest velocity within the vena contracta at the level of the orifice. In most cases, pressure recovery has a negligible effect on the accuracy of gradient calculation. Situations in which pressure recovery may be more significant include small aortic root (e.g., less than 3.0 cm in diameter), domed congenital aortic stenosis, and with certain types of prosthetic valves. In such cases, Doppler will record a higher pressure gradient within the vena contracta, while the catheter-derived pressure will likely be obtained further downstream and will record a lower gradient. These methodologic differences provide a plausible explanation for the slightly higher gradients derived by Doppler versus catheterization techniques.
FIGURE 10.9. A normal tricuspid aortic valve is shown with and without color Doppler. A: The short-axis view demonstrates the three cusps during diastole. B: A diastolic frame with color flow imaging demonstrates trivial aortic regurgitation. C: The valve is shown during systole demonstrating the orifice in an open
position. **D:** Color flow imaging during systole demonstrates the flow through the valve.

**FIGURE 10.10.** A two-dimensional echocardiogram from a patient with severe aortic stenosis. **A:** The long-axis view reveals an echogenic and very immobile aortic valve. **B:** The corresponding short-axis view suggests a high degree of calcification of the valve and minimal mobility during systole.
**FIGURE 10.11.** In the left panel, the long-axis view shows a thickened, sclerotic aortic valve with preserved mobility. In the right panel, Doppler reveals a peak velocity of 2.6 m/s and a mean pressure gradient of 17 mm Hg, consistent with mild stenosis.

**Video 10-11**

**FIGURE 10.12.** An example of severe aortic stenosis in the setting of left ventricular dysfunction. Parasternal (left) and apical (right) long-axis views are provided. The valve is calcified and immobile. A qualitative diagnosis of aortic stenosis is possible, but no quantitative information is available.
FIGURE 10.13. A transesophageal echocardiogram demonstrates the method of direct planimetry of the aortic valve orifice. By carefully adjusting the level of the short-axis plane, the orifice can be visualized in most patients. In this example, severe stenosis was confirmed. AVA, aortic valve area.
FIGURE 10.14. A three-dimensional transesophageal echocardiogram from a patient with degenerative aortic stenosis. In A, the two-dimensional image shows calcification of the cusps and restricted systolic opening. In panel B, the irregularly shaped orifice (arrow) is shown in a short-axis projection.
FIGURE 10.15. Continuous-wave Doppler from the apical four-chamber view in a patient with degenerative aortic stenosis is shown. The peak velocity is 4.3 m/s, corresponding to a maximal gradient of 74 mm Hg.
FIGURE 10.16. A late-peaking gradient from a patient with hypertrophic cardiomyopathy. This obstruction occurs at the level of the left ventricular outflow tract and results in a gradient of approximately 50 mm Hg. Note the contour of the jet with acceleration of flow in mid and late systole.
Despite the generally excellent agreement between Doppler and invasive methods, errors can occur. When discrepancies in measurements happen, several possibilities should be considered. First, the technical quality of the Doppler data should be examined. A technically poor recording may fail to display the highest-velocity signals, resulting in underestimation of the true gradient. An inability to align the interrogation angle parallel to flow also results in underestimation. This relationship is demonstrated in Figure 10.19. The various curves plot the relationship between jet velocity and calculated peak gradient (using the Bernoulli equation), assuming different values for angle $\theta$. Note that for low-velocity jets ($<3$ m/s), the magnitude of the error introduced by angle misalignment is relatively modest. For patients with
severe aortic stenosis, errors in alignment cause substantial underestimation of true gradient. Also note that errors less than 20 degrees result in a relatively insignificant degree of underestimation. However, as the intercept angle increases beyond 20 degrees, the magnitude of error increases rapidly.

Because the Doppler technique measures velocity over time, Doppler-derived data always represent *instantaneous* gradients. It is customary in the cardiac catheterization laboratory to report the peak-to-peak gradient, which is often less than the peak instantaneous gradient. This is illustrated in Figure 10.18. It is well known that peak-to-peak gradients are contrived and never exist in time. Failure to recognize the differences in the reported data often leads to miscommunication of clinical information. This can be partially avoided through the use of mean gradients, which correlate better between catheterization and echocardiographic data. Finally, bear in mind that valve gradients are dynamic measurements that vary with heart rate, loading conditions, blood pressure, and inotropic state. Figure 10.20 is an example of varying jet velocities from a patient with an arrhythmia. Note how each recorded beat yields a different peak instantaneous gradient, ranging from approximately 35 to more than 100 mm Hg. If two tests are performed on different days, the results may be expected to vary. It is therefore not surprising that the best correlation between invasive hemodynamics and Doppler is achieved in studies in which the tests are performed simultaneously. When catheterization and Doppler results differ, both tests may be correct but may reflect variations in gradient over time.

### Table 10.3

<table>
<thead>
<tr>
<th>References</th>
<th>Maximal Pressure Gradient</th>
<th>Aortic Valve Area</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>r Value</td>
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<tr>
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<td>35</td>
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<td>Simpson et al. (1985)</td>
<td>33</td>
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<td>Currie et al. (1985)</td>
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<td>Yeager et al. (1986)a</td>
<td>52</td>
<td>0.87</td>
</tr>
<tr>
<td>Study</td>
<td>n</td>
<td>$\Delta P_{peak}$</td>
</tr>
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<td>-----------------------------</td>
<td>-----</td>
<td>-------------------</td>
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<tr>
<td>Currie et al. (1986)</td>
<td>62</td>
<td>0.95</td>
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<tr>
<td>Teirstein et al. (1986)$^a$</td>
<td>31</td>
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<td>Harrison et al. (1988)</td>
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<td>Oh et al. (1988)$^a$</td>
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<td>Tribouilloy et al. (1994)$^c$</td>
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<td>Kim et al. (1997)$^c$</td>
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</table>

$^a$Data are for mean rather than peak gradient.

$^b$All patients with severe aortic regurgitation.

$^c$Echo-valve area by planimetry using transesophageal echocardiography.

**FIGURE 10.18.** **Left:** This schematic demonstrates the relationship between the pressure gradient across the stenotic aortic valve and the velocity tracing obtained using Doppler. The differences between peak-to-peak and peak instantaneous gradients are illustrated. The maximal flow velocity obtained with Doppler corresponds temporally with the peak instantaneous gradient. **Right:** A
Doppler tracing from a patient with severe aortic stenosis demonstrates a peak gradient of 99 mm Hg and a mean gradient of 69 mm Hg.

**FIGURE 10.19.** The effect of incident angle (θ) on recorded velocity. When the angle is 0 degrees (*uppermost curve*), the Bernoulli equation provides an accurate measure of gradient. As θ increases, an increasing degree of underestimation occurs. See text for details.

Overestimation of the true pressure gradient is less common but can occur. This is usually the result of mistaken identity of the recorded signal. For example, the mitral regurgitation jet has a contour similar to that of a jet of severe aortic stenosis. Because of the similarities in location and direction of the two jets, mistaken identity can occur. To avoid this, the two jets should be recorded by sweeping the transducer back and forth to clearly indicate to the interpreter which jet is which. Another helpful clue involves the timing of the two jets (*Fig. 10.21*). Mitral regurgitation is longer in duration, beginning during isovolumic contraction and extending into isovolumic relaxation.
In most cases, a complete echocardiographic assessment of aortic stenosis includes a determination of aortic valve area using the continuity equation. Based on the principle of conservation of mass, the continuity equation states that the stroke volume proximal to the aortic valve (within the left ventricular outflow tract) must equal the stroke volume through the stenotic orifice. Because stroke volume is the product of the cross-sectional area (CSA) and time velocity integral (TVI), the continuity equation can be arranged to yield:

**FIGURE 10.20.** Doppler recording of aortic stenosis from a patient with an arrhythmia. Note the variability in velocity, depending on the stroke volume and the preceding R-R interval. See text for details.
FIGURE 10.21. The jets of aortic stenosis (AS) and mitral regurgitation (MR) can sometimes be confused. A helpful clue to differentiate between the two involves the timing of flow. **A:** Aortic flow begins after the period of isovolumic contraction. The vertical line provides a reference mark relative to the QRS of the electrocardiogram. Note the gap between the line and the onset of flow. **B:** The same line coincides with the onset of mitral regurgitation. This is because mitral
Regurgitation occurs during isovolumic contraction. In addition, mitral regurgitation flow extends later in systole compared with aortic stenosis (during isovolumic relaxation).

\[
\text{AV area} = \text{CSA}_{\text{OT}} \times \frac{\text{TVI}_{\text{OT}}}{\text{TVI}_{\text{AS}}} \quad \text{[Eq. 10.4]}
\]

This is illustrated in Figure 10.22. To calculate aortic valve area, the following three measurements must be performed: (1) the CSA of the left ventricular outflow tract (OT); (2) TVI of the outflow tract; and (3) TVI of the aortic stenosis jet (AS).

To measure the CSA of the outflow tract, the diameter of the outflow tract is generally measured from the parasternal long-axis view and the shape is assumed to be circular. The equation used is simply

\[
\text{Area} = \pi r^2 \quad \text{[Eq. 10.5]}
\]

where \( r \) is one-half of the measured diameter (in centimeters). The importance of performing this measurement accurately cannot be overemphasized. Because the radius is squared to determine area, small errors in measuring this linear dimension will be compounded in the final formula. The smaller the annulus, the greater is the percentage error introduced by any given mismeasurement. Potential factors that may contribute to errors include image quality, annular calcification (which obscures the true dimension) and failure to measure the true diameter. Importantly, the increased use of CT angiography in recent years for the sizing of transcatheter aortic valve implants has demonstrated that the outflow tract is often noncircular, relatively flattened in the anterior–posterior direction and elongated in the medial-lateral plane (Fig. 10.23). Since two-dimensional echocardiography typically relies on the long-axis view for this measurement, some degree of underestimation of outflow tract area can occur. As can be seen in the illustration, underestimation (by echo) of the LVOT dimension will result in underestimation of both the outflow tract and aortic valve areas, thus suggesting that the severity of stenosis is worse than it actually is. When available, direct planimetry of outflow tract using either CT angiography or 3D echocardiography avoids this potential source of error. This should yield a very accurate outflow tract area, provided the planimetry is performed at the same level that the pulsed Doppler velocity is recorded.
FIGURE 10.22. This illustration demonstrates how aortic valve area is calculated. In A, pulsed Doppler of aortic outflow tract area indicates a time velocity integral (TVI) of 26 cm. In B, the outflow tract (OT) diameter is used to calculate the area, in this case, 3.46 cm$^2$. In C, continuous wave Doppler records the aortic stenosis jet with peak velocity of 4.7 m/s and a TVI of 127 cm. The valve area is therefore 0.7 cm$^2$. 
FIGURE 10.23. A CT scan of the left ventricular outflow tract, from a patient with aortic stenosis, is shown. Because the area is not circular, measuring the LVOT diameter from the parasternal long-axis view to calculate area (yellow arrow) would result in underestimation of the true area, which can be determined by planimetry of the CT image. In this example, the underestimation of LVOT area would be approximately 20%.

The TVI of the outflow tract is measured from the apical window using pulsed Doppler imaging and positioning the sample volume just proximal to the stenotic valve. In this position, the flow is still laminar and has not yet begun to accelerate through the valve. Then, from the same transducer position, continuous-wave Doppler imaging should be used to record the jet velocity envelope. Using planimetry, both envelopes can be traced so that the TVI of each can be derived. If the units used for the measurement of the outflow tract diameter are centimeters, the value of the aortic valve area will be in cm\(^2\). A simplified form of the continuity equation, in which maximal velocity of the outflow tract flow and valve jet are used in place of the respective TVIs, is possible because flow duration across the two sites is the same. Thus, the simplified continuity equation is:

\[
\text{AV area} = \frac{\text{TVI}_{\text{OT}} \times \text{Area}_{\text{OT}}}{\text{TVI}_{\text{AV}}}
\]

“Small” Area\(_{\text{OT}}\) → “Small” AV area

Circle: 3.8 cm\(^2\)
Oval: 4.9 cm\(^2\)
FIGURE 10.24. Aortic valve area (AVA) is calculated in a patient with aortic stenosis and severe left ventricular dysfunction. On the left, the outflow tract diameter is measured from the parasternal long-axis view. In the middle frame, the LVOT flow is recorded and TVI is determined. The jet velocity and TVI are provided in the right panel. From these 3 measurements, the aortic valve area is shown to be 0.6 cm$^2$. See text for details. CSA, cross-sectional area; TVI, time velocity integral.

\[ \text{CSA}_{\text{OT}} = 3.14 \times 1^2 = 3.14 \text{ cm}^2 \]
\[ \text{TVI}_{\text{OT}} = 11 \text{ cm} \]
\[ \text{TVI}_{\text{AS}} = 59 \text{ cm} \]
\[ \text{AVA} = \frac{3.14 \text{ cm}^2 \times 11 \text{ cm}}{59 \text{ cm}} \]
\[ \text{AVA} = 0.6 \text{ cm}^2 \]

**Video 10-24**

\[ \text{AV area} = \text{CSA}_{\text{OT}} \times \frac{V_{\text{OT}}}{V_{\text{AS}}} \quad [\text{Eq. 10.6}] \]

Somewhat technically easier to obtain, this equation yields valve areas that are similar to those obtained using the full equation (Equation 10.4).

As with the Bernoulli equation, this approach has also been validated in a
variety of clinical and in vitro settings. Some of the studies validating the use of the continuity equation to measure aortic valve area are presented in Table 10.3. These and other studies demonstrate that the continuity equation provides an accurate and reproducible assessment of the severity of aortic stenosis. However, at very low-flow rates, the correlation is not as good, with a consistent overestimation of severity of stenosis by the Gorlin equation. In addition to the challenge of properly measuring the area of the outflow tract, other potential sources of error should also be considered. It is essential that the outflow tract area and flow assessment be measured at the same level. Because the area of the outflow tract is usually measured from the parasternal long-axis view, some conventions are necessary to ensure that this is the case. In practice, the sample volume is positioned in the outflow tract from the apical window and then gradually advanced toward the aortic valve until the flow begins to accelerate. At this point, the peak velocity rises and turbulence is apparent. Then, the sample volume is gradually withdrawn toward the apex until the signal becomes laminar and without evidence of acceleration. This is the point at which the Doppler envelope should be measured.

The continuity equation has two important advantages compared to the Bernoulli equation for the assessment of aortic stenosis. First, coexisting aortic regurgitation may increase the measured transvalvular pressure gradient because of the increase in stroke volume through the valve during systole. The continuity equation, on the other hand, is not affected by the presence of aortic regurgitation. More importantly, left ventricular dysfunction may lead to reduced stroke volume and a low measured gradient even in the presence of severe valve stenosis. This concept is demonstrated in Figure 10.24, which is recorded from a patient with aortic stenosis and left ventricular dysfunction. The aortic jet velocity is only 2.9 m/s, which by the Bernoulli equation yields a peak pressure gradient of approximately 33 mm Hg. However, because the flow is reduced (as evidenced by the left ventricular outflow tract TVI of 11 cm), the calculated aortic valve area is 0.6 cm². In this example, the severity of the aortic stenosis would have been significantly underestimated if the pressure gradient alone had been reported rather than the aortic valve area. Another case of severe left ventricular dysfunction is shown in Figure 10.25. In this patient, the mean gradient was only 20 mm Hg, but the valve area was severely reduced at 0.77 cm². Note that the heavily calcified valve and aortic annulus makes precise
measurement of the outflow tract diameter difficult. These two examples demonstrate the importance of the continuity equation. In both, if gradient alone had been relied upon, underestimation of aortic stenosis severity would have occurred. The continuity equation is relatively unaffected by low flow and will allow an accurate determination of valve area whether the stroke volume is normal or reduced. Although an accurate measurement of the pressure gradient is sufficient to make clinical decisions in many cases, a determination of aortic valve area is especially important in patients with significant aortic regurgitation and/or reduced left ventricular function.

The interplay among velocity, stroke volume, and aortic valve area is illustrated graphically in Figure 10.26. The two curves compare the relationship between pressure gradient and aortic valve area at different flow rates, indicated by the different outflow tract velocities (1.2 and 0.8 m/s). Each curve plots the correlation between gradient and valve area for a given level of flow (or stroke volume). At point A, a patient has moderate aortic stenosis, with a peak gradient of 56 mm Hg and a corresponding valve area of 1.0 cm$^2$. This is in the setting of normal left ventricular function, with a peak left ventricular outflow tract velocity of 1.2 m/s. Moving from point A to point B is the result of a sudden decrease in stroke volume (e.g., following a myocardial infarction). The associated decline in stroke volume is evident by the change in left ventricular outflow tract velocity to 0.8 m/s. Because aortic stenosis severity is not affected, the peak gradient decreases to 23 mm Hg and the aortic valve area remains at 1.0 cm$^2$. At this level of left ventricular dysfunction, progression of aortic stenosis well into the severe range (point C, a new valve area of 0.7 cm$^2$) would be required to restore the peak gradient back to the original value of 56 mm Hg. Figure 10.27 illustrates an elderly patient with severe aortic stenosis and normal ejection fraction who declined surgical intervention. A repeat echocardiogram 3 years later demonstrates the development of left ventricular systolic dysfunction. The mean gradient at the time of initial evaluation was 46 mm Hg and the valve area was 0.6 cm$^2$, consistent with severe aortic stenosis. The patient presented 3 years later with heart failure. Left ventricular function had declined significantly, now with an ejection fraction of 35%. Although the mean gradient had not changed significantly (45 mm Hg), the stroke volume had declined from approximately 76 to 50 mL. The calculated aortic valve area had decreased to 0.44 cm$^2$. 
FIGURE 10.25. A patient with calcific aortic stenosis in the setting of severe left ventricular dysfunction. This case demonstrates the importance of using the continuity equation to estimate aortic valve area. See text for details.

AVA = $3.14 \times \frac{15}{61} = .77 \text{ cm}^2$

Video 10-25
FIGURE 10.26. The relationship among pressure gradient, aortic valve area, and stroke volume is demonstrated graphically. See text for details.
FIGURE 10.27. Serial evaluation of a patient who initially had severe, but asymptomatic aortic stenosis. Images from his initial evaluation (A to C) and 3 years later (D to F) are shown. See text for details.
Video 10-27a

coming soon

Video 10-27d

coming soon
FIGURE 10.28. Short-axis views at the level of the aortic valve illustrate the effect of flow on valve motion. These are taken from a patient with severe left ventricular dysfunction and decreased stroke volume. A: The aortic valve is shown during diastole in the closed position. B: Recorded during midsystole, a minimal degree of cusp opening is the result of decreased flow through the valve. The valve is not stenotic, but the relative immobility is the result of a reduced stroke volume.

Other Approaches to Quantifying Stenosis

The quantitative approaches described previously, the Bernoulli and the continuity equations, provide sufficient information in most instances. Thus, other parameters, although available, are used infrequently. Aortic valve resistance is a relatively flow-independent measure of stenosis severity that depends on the ratio of mean pressure gradient and mean flow rate and is
calculated as

$$\text{Resistance} = (\Delta P_{\text{mean}}/Q_{\text{mean}}) \times 1,333 \quad [\text{Eq. 10.7}]$$

The relationship between mean resistance and valve area is given by the formula:

$$\text{Resistance} = \frac{28\sqrt{\text{Gradient}_{\text{mean}}}}{\text{AV area}} \quad [\text{Eq. 10.8}]$$

Several investigators have demonstrated a close relationship between aortic valve resistance and aortic valve area. The advantages of this method over the continuity equation, however, have not been established.

A novel approach to aortic stenosis severity involves the calculation of left ventricular stroke work loss (SWL). SWL is calculated as:

$$\text{SWL} (%) = \frac{100 \times \Delta P_{\text{mean}}}{\Delta P_{\text{mean}} + \text{SBP}} \quad [\text{Eq. 10.9}]$$

where SBP is systolic blood pressure, $\Delta P_{\text{mean}}$ is the mean aortic valve gradient, and SWL is expressed as a percentage. This is based on the concept that the left ventricle expends work during systole to keep the aortic valve open and to eject blood into the aorta. Thus, it accounts for the stiffness of the aortic valve leaflets and is less dependent on flow compared with other parameters. Figure 10.28 is an example of an aortic valve that opens minimally, not because of aortic stenosis but because of low stroke volume. The illustration underscores the relationship between flow and valve motion.

A relatively simple calculation, SWL requires only Doppler determination of the mean aortic valve gradient and measurement of systolic blood pressure. With a normal aortic valve, relatively little work is needed to maintain the aortic leaflets in the open position during systole, and the amount of work performed calculated from left ventricular pressures compared with aortic pressures is very similar. In the setting of aortic stenosis, some of the total work performed must be expended on opening the stiff valve leaflets, resulting in a loss or wasting of some amount of total work. Left ventricular SWL is then calculated as the difference between left ventricular work and effective work. One study compared various hemodynamic measures of aortic stenosis severity for their ability to predict
symptoms and outcome and concluded that SWL was among the best predictors of symptom status and clinical end points. A cutoff value more than 25% effectively discriminated between patients experiencing a good and poor outcome. Again, although conceptually attractive, the calculation of SWL has limited practical application.

**Defining the Severity of Aortic Stenosis**

From the previous section it is clear that there are many parameters that can be used to measure the severity of valve stenosis. Even so, defining severity in an individual patient must take into account several confounding factors. In normal adults, aortic valve area is usually between 3.0 and 4.0 cm² (see Table 10.4). Clinically significant aortic stenosis generally requires the valve area to be reduced to less than one-fourth of normal or between 0.75 and 1.0 cm². As disease progresses and valve area diminishes toward the severe range, small changes in area may be associated with significant changes in hemodynamics. Thus, as severity worsens, the challenges of accurate measuring are compounded and minimal errors in measurement become increasingly important clinically. The relationship between valve area and severity is further influenced by patient size—for example, an aortic valve area of 0.9 cm² may be “severe” in a large patient but only “moderate” in a smaller person. There is also an inconsistent relationship between valve area and symptoms, another very important factor in clinical decision making.

<table>
<thead>
<tr>
<th>Table 10.4 DEFINING SEVERITY OF AORTIC STENOSIS</th>
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<tbody>
<tr>
<td><strong>Stenosis</strong></td>
</tr>
<tr>
<td>Mean gradient (mm Hg)</td>
</tr>
<tr>
<td>Peak gradient (mm Hg)</td>
</tr>
<tr>
<td>Valve area (cm²)</td>
</tr>
</tbody>
</table>

With this background in mind, defining severity has obvious limitations. The American College of Cardiology/American Heart Association Guidelines on valvular heart disease define severe aortic stenosis as a mean gradient ≥40 mm Hg or a valve area ≤1.0 cm². As discussed above, gradient is inherently flow-dependent so the correlation between pressure gradient and valve area will break down when flow (i.e., stroke volume) is reduced. However, even at normal flow rates, the correlation between gradient and valve area is inconsistent. In one large study (Minners et al. *Heart* 2010) the relationship between Doppler-derived mean gradient and valve area in patients with aortic stenosis and a normal stroke volume index was examined. Assuming normal cardiac output, ejection period and heart rate, a considerable amount of inconsistent grading resulted. For example, a valve area of 1.0 cm² corresponded to a mean gradient of 22 mm Hg (rather than 40 mm Hg), and a
mean gradient of 40 mm Hg corresponded to a valve area of 0.74 cm² (see Fig. 10.29). These discrepancies were only partially explained on the basis of a reduced left ventricular stroke volume index. Clearly, a single, universal definition of severity that can be applied to all patients does not exist. Clinicians must recognize these limitations and take into account all parameters, both echocardiographic and clinical, in order to properly classify disease severity and make appropriate clinical decisions.

**Classification of Aortic Stenosis**

The recently published AHA/ACC guidelines on valvular heart disease defines four stages of progression: (A) disease-free, but at risk for development; (B) progressive, including mild to moderate disease without symptoms; (C) severe but asymptomatic; and (D) severe and symptomatic (see Table 10.1). For aortic stenosis, stage C is further divided into: (C1) asymptomatic severe, with normal stroke volume; and (C2) asymptomatic severe, with LV dysfunction (EF <50%). Stage D—severe symptomatic—is subdivided into: (D1) high gradient AS; (D2) low-flow, low-gradient (reduced EF); and (D3) paradoxical low-flow (preserved EF).

The challenges of evaluating low-flow, low-gradient aortic stenosis (stage D2) have been previously covered. Because gradient is flow-dependent, patients with a low stroke volume (and ejection fraction) will have a lower-than-expected gradient for any level of valve area. Thus, gradient alone will underestimate severity. Furthermore, at very low-flow rates, valve opening may be reduced as a result of low flow rather than severe stenosis. Thus in patients with a low-flow rate, gradient will tend to underestimate severity of stenosis, while valve area may overestimate severity (see Figs. 10.24 and 10.25).

In such patients, it may be difficult to distinguish true severe valvular stenosis (with a low pressure gradient due to low stroke volume) from mild to moderate stenosis (with reduced aortic valve opening due to low flow, e.g., a patient with cardiomyopathy). Dobutamine echocardiography may be useful to make this distinction. Using a stepwise infusion of dobutamine from 5 to 20 μg/kg/min (in an effort to increase stroke volume across the stenotic valve) may allow differentiation of the two possibilities. In contrast to the typical dobutamine protocol, in this setting, longer dobutamine stages of 5
minutes are recommended to allow stabilization of hemodynamics before measurements are made. The test assumes that if the leaflets are relatively flexible (mild to moderate stenosis), the valve area will increase in response to an increasing stroke volume. Thus, an increase in valve area during infusion to >1.0 cm$^2$ is consistent with mild to moderate stenosis (Fig. 10.30). Alternatively, true severe aortic stenosis is associated with a fixed valve area that will not change with dobutamine infusion. In such patients, the dobutamine infusion will increase the maximal velocity of both the outflow tract and the jet proportionally and the ratio will remain unchanged. The test is most helpful in the presence of contractile reserve, defined as a >20% increase in stroke volume in response to dobutamine. Truly severe aortic stenosis is then defined as an increase in peak jet velocity of ≥4 m/s or a mean gradient of >30 to 40 mm Hg, provided that the valve area does not exceed 1.0 cm$^2$. In milder forms of stenosis, the increase in velocity of the outflow tract will be greater than that of the jet (due to the functional increase in valve area). In this case, the ratio of outflow tract to jet velocity will increase compared with baseline and the peak jet velocity will remain below 4 m/s. Another possible response to dobutamine is a failure of the left ventricle to augment, in which case neither the gradient nor the valve area changes significantly. This occurs in approximately one-third of cases and is usually the result of severe coronary disease or prior infarction or scarring. This response is associated with a poor overall prognosis and a very high surgical risk. The various responses to dobutamine infusion among patients with aortic stenosis are summarized in Table 10.5. In each of the three examples, a low outflow tract velocity (indicating a reduced stroke volume) and a peak jet velocity of 3.0 m/s (indicating a resting maximal gradient of 36 mm Hg) are present. In the first case, the left ventricular contractility augments with dobutamine, leading to an increase in stroke volume. This leads to an increase in the gradient to 64 mm Hg, consistent with severe stenosis. In the second case, contractile reserve is again present, stroke volume increases, but in this case, the gradient increases to a lesser degree and does not exceed 60 mm Hg. This suggests moderate aortic stenosis. The third case demonstrates lack of contractile reserve, with no significant change in either outflow tract nor jet velocity.
FIGURE 10.30. An example of moderate aortic stenosis in the setting of left ventricular dysfunction is shown. At baseline, the peak gradient is only 21 mm Hg, but because of the low stroke volume (SV) of 45 mL, the aortic valve area (AVA) is .9 cm$^2$, borderline severe. With dobutamine infusion, both the LVOT and the AS jet velocities increase, indicating contractile reserve. Although the peak pressure gradient (PG) rises to 41 mm Hg, the AVA also increases (to 1.1 cm$^2$) suggesting that the aortic stenosis is not severe.

In Figure 10.31, the initial aortic valve gradient is only 30 mm Hg and the low outflow tract velocity (0.6 m/s) is consistent with reduced stroke volume. With infusion of dobutamine, both the outflow tract and the jet velocities increase in a stepwise manner. Although stroke volume increases, the ratio between the outflow tract and the jet velocity does not change appreciably and the peak gradient rises to approximately 60 mm Hg. These findings support the diagnosis of severe aortic stenosis. Figure 10.32 is an example of a significant increase in valve gradient that occurs during dobutamine infusion in a patient with left ventricular dysfunction. In this case, the mean and peak gradients at rest were 31 and 48 mm Hg, respectively. With dobutamine infusion, the corresponding values increased to 50 and 90 mm Hg, confirming the severity of the stenosis.

Paradoxical low-flow, low-gradient (but with normal ejection fraction)
aortic stenosis (stage D3) is a more recently recognized entity. It is defined as an AV area <1.0 cm², a mean gradient <40 mm Hg, but with an ejection fraction >50%. In most cases, this is the result of a low stroke volume index (≤35 mL/m²), which is present despite the “normal” ejection fraction. To understand how this occurs, it is important to remember that the relationship between gradient and valve area depends on flow rate, rather than ejection fraction. Although in most patients a normal flow rate is associated with a normal ejection fraction, this is not always the case. For example, in a small, hypertrophied left ventricle, a normal ejection fraction may be present despite a significant reduction in stroke volume. The relationship between stroke volume and ejection fraction is illustrated in Figure 10.33. Depending on left ventricular size (and end-diastolic volume), the same ejection fraction can yield markedly different stroke volumes. And recall that it is stroke volume, not ejection fraction that determines the relationship between gradient and valve area. Another condition in which a normal ejection fraction is seen in the setting of low forward stroke volume is severe mitral regurgitation.

Paradoxical low-flow, low-gradient aortic stenosis occurs in 10% to 25% of aortic stenosis cases and is most commonly seen in the setting of older age, female gender, diabetes, and hypertension—the same conditions likely to result in a small, hypertrophied left ventricle. Diastolic dysfunction and reduced global longitudinal strain are typically present and contribute to both the abnormal hemodynamics and symptom development.

The echocardiographic assessment of these patients begins with a recognition of the discrepancy between gradient and valve area in a patient with a preserved ejection fraction. Such a combination raises two possibilities—paradoxical low-flow, low-gradient aortic stenosis, or moderate aortic stenosis (as reflected by the mean gradient <40 mm Hg) and an associated error in valve area determination (as reflected by the valve area <1.0 cm²). Differentiating between the two possibilities hinges on a careful and accurate determination of stroke volume (indexed to body surface area). As shown in Figure 10.34, pulsed Doppler recording of LVOT flow must be obtained just below the valve, before flow acceleration occurs. Then, the outflow tract dimensions should be taken at the same level. If the resulting stroke volume, indexed for body surface area, is ≤35 mL/m², the findings are consistent with paradoxical low-flow, low-gradient aortic stenosis. Another example is provided in Figure 10.35. In this patient, despite a mean gradient of only 18
mm Hg, the calculated aortic valve area was 0.6 cm². 

**Table 10.5**

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Low Dose</th>
<th>Mid Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVOT Velo</td>
<td>Jet Velo</td>
<td>Maximal</td>
<td>LVOT Velo</td>
</tr>
<tr>
<td>Velocity</td>
<td>Gradient</td>
<td>Gradient</td>
<td>Velocity</td>
</tr>
<tr>
<td>0.6</td>
<td>3.0</td>
<td>36</td>
<td>0.8</td>
</tr>
<tr>
<td>0.6</td>
<td>3.0</td>
<td>36</td>
<td>0.8</td>
</tr>
<tr>
<td>0.6</td>
<td>3.0</td>
<td>36</td>
<td>0.6</td>
</tr>
</tbody>
</table>

Values for velocity are in meters per second; for gradient, in millimeters of mercury.

AS, aortic stenosis.

To avoid errors in diagnosis of this entity, several steps are necessary. First, the patient’s blood pressure at the time of the echo examination should be recorded. The presence of untreated hypertension confounds the Doppler assessment of aortic stenosis. If blood pressure is elevated, it should be treated and the echocardiographic evaluation should be repeated. Next, the valve area must be checked to ensure that it is really less than 1.0 cm². If a reduced aortic valve area cannot be determined with confidence, the possibility of moderate stenosis severity should be considered. The stroke volume index, another potential source of error, should be rechecked and verified against other parameters of systolic function. If the ventricle is small and hypertrophied with reduced global longitudinal strain and Doppler evidence of impaired relaxation, the plausibility of a low stroke volume is confirmed. Alternatively, if the left ventricular volume is normal and left
ventricular hypertrophy is absent, the likelihood of a reduced stroke volume is much less. Finally, the overall clinical scenario should be examined. Most patients with this condition are symptomatic and have at least one or more of the risk factors described previously. If this is not the case, an alternative explanation for the echocardiographic findings should be sought.

**Natural History of Aortic Stenosis**

In addition to its pivotal role in diagnosis, echocardiography has contributed significantly to an understanding of the natural history of valvular aortic stenosis and its rate of progression. Because of the relatively long asymptomatic period, predicting the rate of progression to severe aortic stenosis is helpful in the timing of follow-up evaluation and the planning for surgical intervention. The definition of severe aortic stenosis varies, but echocardiographic criteria have been established. When the maximal aortic valve velocity exceeds 4 m/s, indicating a peak pressure gradient of greater than 64 mm Hg, the stenosis is considered severe. Some investigators have argued that the mean pressure gradient is a better predictor of severity, often using a cutoff of 40 mm Hg as the criterion for severe.

As can be seen in **Figure 10.36**, a precise correlation between maximal velocity or mean gradient and the aortic valve area is lacking and considerable overlap exists. Using an aortic valve area less than 1 cm$^2$ as the definition of severe stenosis, patients with mean Doppler gradients between 10 and 110 mm Hg would be included (**Fig. 10.36B**). Much of this range is accounted for on the basis of left ventricular dysfunction. Furthermore, significant overlap occurs in measured severity between symptomatic and asymptomatic individuals.

Several studies have contributed to our understanding of the rate of disease progression in adult patients with aortic stenosis. Despite individual variability, most patients demonstrate an average increase in mean pressure gradient of 0 to 10 mm Hg per year (mean, 7 mm Hg) with a corresponding decrease in aortic valve area of 0.12 ± 0.19 cm$^2$ per year. **Figure 10.37** illustrates progression of aortic stenosis over a 2-year period. In this example, the peak aortic gradient increases from 49 to 69 mm Hg. Progression can also occur in the absence of an increase in jet velocity if left ventricular function declines (see **Fig. 10.27**). To date, attempts to predict the determinants of
more rapid progression have been largely unsuccessful.

**Clinical Decision Making**

The management of patients with aortic stenosis must take into account the presence or absence of symptoms, the severity of the stenosis, the status of the left ventricle, and the existence of comorbidities. Recent advances in treatment of valvular aortic stenosis, notably transcutaneous approaches, have greatly expanded the options for therapy, including patients for whom surgery was not a possibility. This topic is covered more fully in Chapter 14.

Most asymptomatic adults with significant aortic stenosis are managed medically, and thus the role of echocardiography in this group focuses on measuring severity, rate of progression, and assessment of left ventricular function. However, it is now recognized that some asymptomatic patients with severe aortic stenosis (stage C) may be best served with valve replacement, either surgical or percutaneous. This decision is based in part on clinical factors, but also includes echocardiographic findings, such as ejection fraction, severity of disease, rate of progression, and degree of leaflet calcification. A left ventricular ejection fraction <50% in an asymptomatic patient with severe aortic stenosis remains the only class I indication for valve surgery in this subset. In addition, the most recent AHA/ACC Valvular Heart Disease Guidelines consider a peak jet velocity ≥5 m/s, a mean gradient ≥60 mm Hg, and markers of very severe aortic stenosis, as reasonable indications (class IIa) for valve replacement in the absence of symptoms. A summary of recommendations for intervening in patients with aortic stenosis from the 2014 AHA/ACC Valvular Heart Disease Guidelines in provided in Table 10.6.

It should also be recognized that a lack of symptoms reported by a patient with severe aortic stenosis may not be reliable. In this setting, exercise testing is considered a class IIa indication, primarily to assess exercise tolerance, ST-segment depression, or an abnormal blood pressure response (Fig. 10.38). However, the role of stress echocardiography (as opposed to a nonimaging treadmill test) remains controversial. Some studies have reported an increase in mean gradient during exercise of >20 mm Hg as a predictor of a poor outcome in asymptomatic patients. However, the echocardiographic Appropriate Use Criteria update from 2011 does not endorse exercise
echocardiography is this setting. Finally, exercise testing should never be done in symptomatic (stage D1) patients. The more recently recognized subset of patients with paradoxical low-flow, low-gradient aortic stenosis (stage D3) appears to represent a particularly high-risk group in whom valve replacement should be considered. Such patients are generally symptomatic and have multiple comorbidities.
FIGURE 10.31. Dobutamine stress echocardiography can be used to assess the severity of aortic stenosis in patients with left ventricular dysfunction. **Top:** Baseline two-dimensional echocardiogram demonstrating significant left ventricular dysfunction. **Bottom:** Doppler recordings of left ventricular outflow tract velocity (above) and aortic jet velocity (below) at rest, 20 μg/kg/min, and 30 μg/kg/min. See text for details.

Video 10-31
FIGURE 10.32. An elderly patient with ischemic cardiomyopathy and aortic stenosis. At baseline, left ventricular systolic function is reduced (A) and Doppler interrogation suggests moderate stenosis (B, mean and maximal gradients 30 and 48 mm Hg, respectively). In (C), during dobutamine infusion, increased contractility occurs with only a modest increase in heart rate. However, the mean and maximal gradients increase to 48 and 90 mm Hg, respectively. This indicates severe stenosis.
FIGURE 10.33. The relationship between stroke volume and ejection fraction (EF) depends on left ventricular volume, specifically, on end-diastolic volume. In the top panel, an ejection fraction of 55% in a patient with a normal left ventricular volume yields a stroke volume of 77 mL. In the lower panel, the same EF in a patient with a small, hypertrophied ventricle yields a much lower stroke volume (49 mL).

The indications and timing of intervention in patients with aortic stenosis are listed in Table 10.6. Based on these recommendations, it is clear that echocardiography plays a role before, during, and after such interventions. Being able to determine aortic stenosis severity over a range of flow states, irrespective of left ventricular ejection fraction, is critical to clinical decision making.
An example of paradoxical low-flow, low-gradient aortic stenosis is shown. In A, the parasternal long-axis view shows thickened LV walls with normal contractility and a small end-systolic cavity. Doppler of the left ventricular outflow tract (B) and aortic valve jet (C) allow the valve area to be calculated. Despite a peak aortic valve gradient of only 23 mm Hg, the valve area is 0.7 cm².

Video 10-34a

AORTIC REGURGITATION
Aortic regurgitation may be congenital or acquired and may be caused by either abnormalities of the aortic root or the valve itself. Some of the more common causes of aortic regurgitation are listed in Table 10.7. Long-standing hypertension may result in dilation of the aortic root and annulus, leading to valvular regurgitation. Other diseases of the aortic root often associated with aortic regurgitation include Marfan syndrome, syphilitic aortitis, annuloaortic ectasia, and aortic dissection. Often, dilation of the sinotubular junction is the underlying mechanism for these causes of aortic regurgitation. More commonly, aortic regurgitation is due to defects in the valve leaflets, including BAV, rheumatic heart disease, endocarditis, and degenerative calcific aortic valve disease. An unusual cause of aortic regurgitation is membranous subaortic stenosis. In these patients, the impact of the jet through the stenotic membrane damages the valve, leading to regurgitation (Fig. 10.39). Regardless of etiology, aortic regurgitation imposes a volume overload on the left ventricle and, eventually, a reduced forward stroke volume. Thus, the echocardiographic assessment of this condition includes establishing a diagnosis, determining an etiology, evaluating the effects of volume overload on the left ventricle, and a careful assessment of the aortic root. The stages of aortic regurgitation, as described in the recent AHA/ACC Valvular Heart Disease Guideline update (2014), are summarized in Table 10.8.

**Appropriate Use Criteria**

Recommendations for the appropriate use of echocardiography in aortic regurgitation, are provided in Table 10.2. An echocardiogram is considered appropriate for the initial evaluation of known or suspected aortic regurgitation, for annual reevaluation of an asymptomatic patient with moderate or severe aortic regurgitation, and for reevaluation of any patient with a change in clinical status or to guide therapy. It is considered inappropriate to perform echocardiography for the routine reevaluation of a patient with mild aortic regurgitation, no change in clinical status, and normal left ventricular size.

**M-Mode and Two-Dimensional Imaging**

As the aortic jet cascades across the anterior mitral leaflet, it creates a high-
frequency fluttering that requires the rapid sampling rate of M-mode echocardiography for detection. This was one of the earliest examples of the use of the M-mode technique to indirectly assess valve disease (Fig. 10.40). In acute aortic regurgitation, premature closure of the mitral valve (due to rapidly increasing left ventricular diastolic pressure) was also initially detected with this technique (Fig. 10.41). As with other forms of valve disease, however, the development of two-dimensional imaging and Doppler techniques largely supplanted the M-mode technique in this setting.

FIGURE 10.35. Paradoxical low-flow, low-gradient aortic stenosis often occurs in the setting of a small, hypertrophied left ventricle. This example had a stroke volume index of 20 mL/m². The valve area was 0.6 cm². The mean aortic valve gradient was only 18 mm Hg (C).

FIGURE 10.37. Doppler imaging is useful to document the rate of progression of aortic stenosis. A: A baseline study; B, C: recorded at 1- and 2-year intervals,
respectively. The series demonstrates a gradual increase in peak gradient across the valve.

Two-dimensional echocardiographic imaging focuses on a detailed evaluation of the aortic valve and root and an assessment of left ventricular size and function. Many of the causes of aortic regurgitation, including rheumatic, degenerative, and congenital, are established on the basis of two-dimensional echocardiographic findings. Very importantly, manifestations of endocarditis are accurately assessed with a combination of transthoracic and transesophageal echocardiography. Figure 10.42 is an example of abnormal mitral valve motion due to impingement on the anterior leaflet by a posteriorly directed aortic regurgitation jet. Note how the midportion of the leaflet is deformed during diastole.

Diseases that affect the aortic root can cause regurgitation by altering the geometry of aortic leaflet coaptation, primarily through dilation at the level of the sinotubular junction (Fig. 10.43). Conditions such as hypertension, Marfan syndrome, and cystic medial necrosis typically result in the combination of a dilated aortic root and some degree of aortic regurgitation (Fig. 10.44). In such conditions, the aortic regurgitation jet arises centrally and may vary over the full range of severity. Causes of acute aortic regurgitation that can be identified using two-dimensional echocardiography include aortic dissection (Fig. 10.45) and endocarditis (Fig. 10.46). Figure 10.45 is from a patient with BAV and ascending aortic aneurysm who developed type A dissection with only mild aortic regurgitation. Figure 10.47 is an example of aortic dilation at the level of the sinotubular junction that prevents the normal coaptation of the aortic cusps, resulting in severe, eccentric aortic regurgitation. Figure 10.48 is an example of paravalvular regurgitation occurring as a result of abscess formation in a patient with a stentless aortic prosthesis. Two-dimensional echocardiography is critically important in patients with aortic regurgitation to evaluate the left ventricle’s response to volume overload. Over an extended period, chronic aortic regurgitation leads to dilation of the left ventricle and a characteristic change to a more spherical shape. Left ventricular systolic function is typically preserved and left ventricular mass increases, although the increase in wall thickness is often quite modest. Hyperdynamic interventricular septal motion occurs as a result of left ventricular volume overload due to unequal filling.
and stroke volume of the ventricles. The enlarging left ventricle remains compliant and is able to accept the simultaneous filling through the mitral and aortic valves throughout diastole without a significant increase in pressure. Eventually, left ventricular function begins to deteriorate, although this generally does not occur until a significant increase in end-systolic volume is present. Figure 10.49 is taken from a patient with long-standing aortic regurgitation. The left ventricular end-diastolic dimension was 6.2 cm. Also note the globular shape of the chamber. The reduction in left ventricular function should be viewed as a late and sometimes irreversible change in the natural history of the disease. The implications of these changes on clinical decision making will be discussed later.

**Establishing a Diagnosis of Aortic Regurgitation**

By directly visualizing the aortic valve, two-dimensional echocardiography can frequently identify an anatomic condition that would predispose to the development of aortic regurgitation. Although such indirect indicators may provide a clue to the presence of aortic regurgitation, the specific diagnosis requires Doppler techniques. In some cases, even when aortic regurgitation is severe, two-dimensional imaging will be surprisingly unremarkable, even suggesting that the valve is “anatomically” normal. In such cases, Doppler imaging will be the most important, and sometimes the only, clue to a diagnosis.

The jet of aortic regurgitation can be recorded with pulsed, continuous-wave, or color flow Doppler imaging. All three methods are highly sensitive for the detection of regurgitation and should be viewed as complementary in the evaluation of individual patients (Figs. 10.50 and 10.51). Pulsed Doppler echocardiography relies on the demonstration of turbulent flow during diastole in the left ventricular outflow tract on the ventricular side of the aortic valve (Fig. 10.52). Because the velocity of the aortic regurgitation jet is high, aliasing occurs inevitably, but simply the presence of turbulence will usually establish the diagnosis. The method is highly sensitive but requires a methodical and careful search for the regurgitant jet, using multiple views and echocardiographic windows. There can be false-positive findings, sometimes in the setting of mitral stenosis or a prosthetic mitral valve, where turbulent diastolic flow may be mistaken for aortic regurgitation. Because the
aortic regurgitation jet is invariably high-velocity, continuous-wave Doppler imaging is necessary for the full contour of the envelope to be recorded (Fig. 10.53). The density of the jet, a qualitative indication of the volume of regurgitation, can also be assessed. Density is a function of the number of blood cells being sampled and will generally increase as the regurgitant volume increases. The velocity of the regurgitation jet and particularly the rate of deceleration of retrograde flow can be measured (Fig. 10.54). Continuous-wave Doppler imaging is especially helpful when there is confusion about whether a flow disturbance is due to aortic regurgitation or mitral stenosis (Fig. 10.55). The velocity and contour of the jet will generally allow this distinction to be established.

FIGURE 10.38. Peak exercise ECG from a patient with severe asymptomatic aortic stenosis. Although the patient demonstrated good exercise tolerance, there was ST-segment depression and a decline in blood pressure at peak exercise.

Table 10.6  RECOMMENDATIONS FOR TIMING OF INTERVENTION IN AORTIC STENOSIS
Recommendations

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
<th>Level of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVR is recommended for severe high-gradient AS in patients with symptoms by history or on exercise testing (stage D1)</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>AVR is recommended in asymptomatic patients with severe AS and LVEF &lt;50% (stage C2)</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>AVR is recommended for severe AS when undergoing other cardiac surgeries (stage C or D)</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>AVR is reasonable for asymptomatic patients with very severe AS (jet velocity ≥5 m/s) and low surgical risk (stage C1)</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>AVR is reasonable for asymptomatic patients with severe AS and decreased exercise tolerance or an exercise fall in BP (stage C1)</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>AVR is reasonable in symptomatic patients who have low-flow, low-gradient severe AS with a reduced LVEF with a dobutamine stress study showing an increase in jet velocity ≥4.0 m/s, or mean gradient &gt;40 mm Hg, with a valve area ≤1.0 cm² at any dobutamine stage (stage D2)</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>AVR is reasonable in symptomatic patients who have low-flow, low-gradient severe AS who are normotensive with an EF ≥50% if clinical, hemodynamic, and anatomic data support AS as the most likely cause of symptoms (stage D3)</td>
<td>IIa</td>
<td>C</td>
</tr>
<tr>
<td>AVR is reasonable for patients with moderate AS who are undergoing other cardiac surgeries (stage B)</td>
<td>IIa</td>
<td>C</td>
</tr>
<tr>
<td>AVR may be considered for asymptomatic patients with severe AS and rapid disease progression who are at low surgical risk (stage C1)</td>
<td>IIb</td>
<td>C</td>
</tr>
</tbody>
</table>

AVR, aortic valve replacement; AS, aortic stenosis; BP, blood pressure; EF, ejection fraction.


Table 10.7 CAUSES OF AORTIC REGURGITATION

<table>
<thead>
<tr>
<th>Affecting the Aortic Leaflets</th>
<th>Involved the Ascending Aorta</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Congenital</strong></td>
<td><strong>Acquired</strong></td>
</tr>
<tr>
<td>Bicuspid, unicuspid, or quadricuspid AV</td>
<td>AV sclerosis/calcification</td>
</tr>
<tr>
<td>Leaflet fenestration</td>
<td>Rheumatic disease</td>
</tr>
<tr>
<td>Subarterial VSD</td>
<td>Infective endocarditis</td>
</tr>
<tr>
<td><strong>Congenital</strong></td>
<td><strong>Acquired</strong></td>
</tr>
<tr>
<td>Annuloaortic ectasia</td>
<td>Hypertension</td>
</tr>
<tr>
<td>Loeys–Deitz syndrome</td>
<td>Aortic dissection, type A</td>
</tr>
<tr>
<td>Marfan syndrome</td>
<td>Idiopathic aortic root dilation</td>
</tr>
</tbody>
</table>
Membranous subaortic stenosis  | Antiphospholipid syndrome  | Ehlers–Danlos syndrome  | Syphilitic aortitis  
--- | --- | --- | ---  
Chest irradiation  |  | Lupus erythematosus  | Trauma  

AV, aortic valve; VSD, ventricular septal defect.

**FIGURE 10.39.** An unusual cause of aortic regurgitation is the presence of a subaortic membrane. **A:** A parasternal long-axis view demonstrates narrowing just below the aortic valve (arrow) due to the membrane. **B:** Doppler imaging demonstrates a peak gradient of approximately 50 mm Hg, at the level of the subaortic membrane. **C:** A mild degree of aortic regurgitation (arrow) recorded using color flow imaging.

**Video 10-39**

**Table 10.8** STAGES OF AORTIC REGURGITATION

<table>
<thead>
<tr>
<th>Stage</th>
<th>Definition</th>
<th>Symptoms</th>
<th>Valve Anatomy</th>
<th>Hemodynamics</th>
<th>Consequences</th>
</tr>
</thead>
</table>

coming soon
<table>
<thead>
<tr>
<th>Column A</th>
<th>Column B</th>
<th>Column C</th>
<th>Column D</th>
</tr>
</thead>
<tbody>
<tr>
<td>At risk for AR</td>
<td>Progressive AR</td>
<td>Asymptomatic severe AR</td>
<td>Symptomatic severe AR</td>
</tr>
<tr>
<td>None</td>
<td>None</td>
<td>None; exercise testing reasonable to confirm</td>
<td>Exertional dyspnea, angina, HF symptoms</td>
</tr>
<tr>
<td>Bicuspid AV, AV sclerosis, diseases of the sinuses or ascending aorta, rheumatic heart disease, previous IE</td>
<td>Mild to moderate calcification Previous IE Rheumatic changes Dilated sinuses</td>
<td>Calcified valve Bicuspid valve Previous IE, abnormal cusp closure, perforation Rheumatic changes Dilated sinuses or ascending aorta</td>
<td>Leaflet calcification Bicuspid valve Previous IE, abnormal cusp closure, perforation Dilated sinuses or ascending aorta Rheumatic changes</td>
</tr>
<tr>
<td>AR severity: None or trace</td>
<td>None</td>
<td>Jet width &gt;65% of LVOT Vena contracta &gt;0.6 cm Holodiastolic flow reversal in the DA RV &gt;60 mL RF &gt;50% ERO &gt;0.3 cm² Evidence of LV dilation (if chronic AR)</td>
<td>Jet width &gt;65% of LVOT Vena contracta &gt;0.6 cm Holodiastolic flow reversal in the DA RV &gt;60 mL RF &gt;50% ERO &gt;0.3 cm² Evidence of LV dilation (if chronic AR)</td>
</tr>
<tr>
<td>Stage C1: Normal LVEF (≥50%) and mild–moderate LV dilation</td>
<td>Stage C2: Abnormal LV systolic function and EF &lt;50% or severe LV dilation</td>
<td>May occur with normal LV systolic function, mild–moderate systolic dysfunction, or severe systolic dysfunction (EF &lt;40%) Moderate to severe LV dilation</td>
<td></td>
</tr>
</tbody>
</table>
FIGURE 10.40. An M-mode echocardiogram from a patient with aortic regurgitation demonstrates fine fluttering of the anterior mitral leaflet as a result of the jet. PW, posterior wall.
FIGURE 10.41. An M-mode recording from a patient with acute and severe aortic regurgitation demonstrates both fluttering (FL) of the anterior mitral leaflet and premature closure (C’) of the mitral valve, the result of rapidly increasing diastolic left ventricular pressure.
FIGURE 10.42. A two-dimensional short-axis echocardiogram is shown from a patient with significant aortic regurgitation and a posteriorly directed jet. The arrows indicate the effect of the jet on the anterior mitral leaflet. The midportion of the leaflet is deformed during diastole as a result.

By far, the most commonly used technique to assess aortic regurgitation is color flow imaging. This technique has a reported sensitivity of greater than 95% and a specificity of nearly 100% for establishing the diagnosis. In fact, using current generation scanners, minor degrees of aortic regurgitation may be detected using color flow imaging in many otherwise normal individuals. Most cases involve “trivial” or “mild” regurgitation, and the prevalence increases with advancing age. Among normal subjects younger than 40 years of age, significant aortic regurgitation is rare, occurring in less than 1%. The reported frequency in older individuals is much higher, however, occurring in between 10% and 20% of subjects older than 60 years of age. In very elderly individuals, for example, those older than 80 years of age, some degree of aortic regurgitation can be detected using color Doppler imaging in the vast
majority.

Color flow imaging will demonstrate a turbulent jet in the left ventricular outflow tract of nearly all patients with clinical evidence of aortic regurgitation. The jet usually persists throughout diastole, and its dimensions provide useful information regarding severity. False-negative findings are rare but may occur in the setting of very high heart rate, in which diastole is short in duration and the frame rate of the ultrasound instrument permits only a few diastolic frames to be displayed. For example, in acute, severe aortic regurgitation, when the heart rate is often high and the duration of the regurgitant flow may be short, color Doppler may underestimate both the presence and severity of disease. In this setting, continuous-wave Doppler, by virtue of its higher sampling rate, is often useful.
FIGURE 10.43. A common cause of aortic regurgitation is aortic root dilation. In this example, dilation and effacement of the sinotubular junction are present (A). Color Doppler demonstrates severe aortic regurgitation (B). From the short-axis view (C), failure of complete coaptation the three aortic cusps is seen. Video 10-43a

Video 10-43a

coming soon

Video 10-43b

coming soon
Evaluating the Severity of Aortic Regurgitation

The severity of aortic regurgitation can be judged using several different criteria. The size or extent of the regurgitant jet within the left ventricle, the effective regurgitant orifice area (ROA), and the volume or fraction of regurgitant flow are distinct, but obviously interrelated, measures of severity. Although the effective ROA may be the most hemodynamically important parameter, it is quite challenging to derive in patients with aortic regurgitation. By far, the most commonly used approach relies on the relationship between the size of the regurgitant jet, visualized by color flow imaging, and the regurgitant volume. The jet should be recorded in multiple imaging planes to provide a 3D assessment of its dimensions. It is now generally believed that the length of the jet conveys unreliable information about overall severity. In any given plane, the area of the jet can be estimated or measured by planimetry. Figure 10.56 is recorded from a patient with mild regurgitation. The jet originates posteriorly and is narrow at the orifice. Both the area and the length of the color jet are small. Figure 10.57 shows three examples of aortic regurgitation recorded with color flow imaging, demonstrating the differences in appearance of the regurgitant jet in mild, moderate, and severe disease. Again, this approach has several limitations and correlates only modestly with other measures of severity.
FIGURE 10.44. Aortic regurgitation can result from dilation of the aortic root. A: A severely dilated aortic root in a patient with a prosthetic aortic valve. B: A similar degree of dilation is demonstrated from a patient with Marfan syndrome. In both cases, significant aortic regurgitation was present.
FIGURE 10.45. Type A aortic dissection causing severe aortic regurgitation in a patient with bicuspid aortic valve. During diastole, the dissection flap herniates through the aortic valve into the left ventricular outflow track.
FIGURE 10.46. Endocarditis can cause aortic regurgitation through a variety of mechanisms. A: A long-axis view demonstrates a long, thin mass attached to the aortic valve, extending into the outflow tract (arrows). B: Color Doppler imaging demonstrates mild aortic regurgitation.

FIGURE 10.47. Dilation of the aorta at the sinotubular junction prevents normal...
Aortic cusp coaptation (A) resulting in severe, eccentric aortic regurgitation (B, white arrows).

Video 10-47a

Video 10-47b

A related approach relies on the visualization of the regurgitant jet at its origin (i.e., immediately downstream from the valve) as an indicator of regurgitant orifice size (Fig. 10.58). From the parasternal long-axis view, the “height” of the jet just below the valve can be measured using electronic calipers. This dimension can also be expressed as a percentage of left ventricular outflow tract dimension to provide an estimate of severity. In the three examples in Figure 10.57 note the differences in jet height/outflow tract dimension ratio. Figure 10.58 illustrates a jet height that occupies more than
70% of the left ventricular outflow tract dimension. The greater the percentage is of the left ventricular outflow tract that is filled by the jet at its origin, the more severe the regurgitation. A jet that occupies more than 60% of the left ventricular outflow tract (either height or area) usually indicates severe aortic regurgitation. A similar approach uses the short-axis view with the imaging plane positioned immediately proximal to the aortic valve (Fig. 10.59). The outflow tract is directly visualized as a circular space, and the regurgitant jet is visualized as a two-dimensional shape within this circle.

There are several limitations to the use of color flow mapping as a direct indicator of severity. Eccentric jets may become entrained along a wall of the left ventricle, which tends to alter their appearance and hence the perception of severity (Fig. 10.60). It must be remembered that the jet is inherently 3D so that no one imaging plane conveys complete information about its shape and extent. The apparent size of the jet is very instrument dependent. Changes in gain, color scale, transducer frequency, and wall filters will affect the jet appearance, independent of severity. For example, the width of an aortic regurgitant jet is often greater from an apical view compared with a parasternal view. This is because the jet’s width recorded from a parasternal projection depends on axial resolution, whereas the same dimension recorded apically will rely more on lateral resolution, resulting in the appearance of a wider jet. Alternatively, image quality and/or the 3D shape of the jet may create the opposite effect. Figure 10.61 is an example of aortic regurgitation that appears mild in the apical four-chamber view but moderate in the parasternal long-axis projection. The example merely points out the limitations of color flow imaging in assessing regurgitation severity and underscores the fact that no single view conveys all the necessary information for measuring severity. Finally, there is evidence that the ROA in patients with chronic aortic regurgitation changes (and usually decreases) during diastole. This finding has implications for techniques such as color Doppler and may explain the temporal variability in jet size in many patients. A gradual decrease in ROA would also account for the tendency of color Doppler to overestimate severity because the visualized jet area would reflect peak rather than mean orifice area.
FIGURE 10.48. Images were recorded from a patient with a Medtronic Freestyle aortic prosthesis who developed an aortic root abscess. **A:** The transesophageal echocardiogram demonstrates a complex echo-free space (*arrow*) surrounding the aortic valve. **B:** The same abnormality is demonstrated from the short-axis view (*arrow*). **C:** Color Doppler imaging demonstrates flow within the abscess cavity and evidence of paravalvular regurgitation (*arrows*). [Video 10-48](coming soon)
FIGURE 10.49. **A:** The effect of chronic, severe aortic regurgitation on the LV. The volume overload imposed by the regurgitation eventually results in left ventricular enlargement and dysfunction. The chamber assumes a more spherical shape. **B:** Color Doppler imaging demonstrates the aortic regurgitation jet (*black arrow*). See text for details.

Video 10-49

FIGURE 10.50. An example of mild aortic regurgitation demonstrated with color Doppler (*arrow*) from the parasternal long-axis view.
Continuous-wave Doppler imaging can also be used to estimate severity. The simplest approach compares the density or darkness of the envelope of the antegrade aortic flow and the regurgitant jet. The larger the regurgitant volume is, the darker the regurgitant jet is on continuous-wave imaging, since density is proportional to the number of blood cells within the beam. The shape of the envelope also contains information. The velocity of the jet is simply a reflection of the pressure gradient between the aorta and the left ventricle throughout diastole (Fig. 10.62). This can be thought of as the driving force for the regurgitant flow. In early diastole, the gradient is highest and the velocity will be in the range of 4 to 6 m/s, depending on the blood pressure. As diastole progresses, the gradient diminishes as aortic pressure decreases and left ventricular pressure increases.

With mild aortic regurgitation, a compliant left ventricle allows a slow and modest increase in left ventricular pressure and aortic diastolic pressure is maintained. Thus, the velocity of the regurgitant jet remains relatively high throughout diastole and the envelope appears flat. With more severe aortic regurgitation, the combination of increasing left ventricular pressure and more rapidly decreasing aortic pressure leads to a more rapid deceleration of the regurgitant jet velocity resulting in a steeper slope of the Doppler envelope (Fig. 10.63). The deceleration of jet velocity can be described as either the slope or the pressure half-time of the jet. These parameters have been correlated with other measures of severity, and a reasonable agreement has been demonstrated. A pressure half-time less than 250 ms or a slope
greater than 400 cm/s\(^2\) is an indicator of severe aortic regurgitation. However, other factors, including aortic compliance, blood pressure, and left ventricular size and compliance will also affect these measures. As is discussed later, a rapid rate of deceleration of the aortic regurgitation jet is more an indicator of acuity rather than severity.

**FIGURE 10.51.** Grading the severity of aortic regurgitation should be based on multiple criteria, including color Doppler (A and D), continuous-wave Doppler (B), and pulsed Doppler to assess retrograde flow in the descending aorta (C). In this example, the lesion appears moderate by color Doppler, from both the parasternal and apical views. The flat deceleration slope on the continuous-wave Doppler recording (B) and the absence of diastolic flow reversal in the descending aorta
A final nonquantitative approach using pulsed Doppler imaging assesses diastolic flow reversal in the descending aorta. Two examples of this are shown in Figure 10.64. As aortic regurgitation becomes worse, a greater degree of flow reversal occurs and retrograde velocities can be recorded throughout diastole. Again, this parameter is dependent on vessel compliance and the location of the sample volume but does provide a simple and practical marker of severity. The presence of holodiastolic flow reversal in the descending aorta and an end-diastolic velocity >20 cm/s have been correlated with severe aortic regurgitation.

Several more quantitative approaches are also available to assess aortic regurgitation. Because the four valves of the heart exist in series, the flow or stroke volume at any point must be equal. In the setting of aortic regurgitation, the total stroke volume through the aortic valve in systole must equal the forward stroke volume (which can be determined at another nonregurgitant valve) plus the regurgitant volume (Fig. 10.65). As described previously, stroke volume is simply the product of the CSA and TVI. If the mitral valve is competent, forward stroke volume is typically measured at this location. Then, total stroke volume across the aortic valve is determined. This value will include both forward and regurgitant volumes. Hence, the regurgitant volume is the difference between the forward flow across the aortic and mitral valves (Fig. 10.66). This approach has been validated in
several laboratories. Both the regurgitant stroke volume and the regurgitant fraction can be quantified. As a reference, a regurgitant fraction greater than 50% or a regurgitant volume greater than 60 mL indicates severe aortic regurgitation. In the example provided in Figure 10.66, stroke volume is calculated as 112 cc across the aortic valve and 69 cc across the mitral valve. The difference is the result of significant aortic regurgitation. Based on these values, the regurgitant volume is approximately 43 cc and the regurgitant fraction is 38%.

**FIGURE 10.52.** Using pulsed Doppler, the high-velocity jet of aortic regurgitation recorded in the left ventricular outflow tract demonstrates aliasing.
FIGURE 10.53. Continuous-wave Doppler recording of an aortic regurgitant jet from the apical window. The velocity and contour of the jet are best appreciated using this technique.
FIGURE 10.54. Two patients with aortic regurgitation are shown. In A, color Doppler suggests mild aortic regurgitation (arrow) and continuous-wave Doppler shows a faint regurgitant signal with a flat deceleration slope (B). The second patient demonstrates more severe aortic regurgitation, with a wider vena contracta (C, arrows) and a more dense continuous-wave Doppler signal with a steeper slope (D).
FIGURE 10.55. Because of the proximity of the aortic regurgitant jet to mitral inflow, the two flow patterns can sometimes be recorded simultaneously. In this example, severe aortic regurgitation is superimposed on the mitral inflow pattern (arrows).
FIGURE 10.56. A transesophageal echocardiogram demonstrates mild aortic regurgitation. The jet originates posteriorly.
FIGURE 10.57. Three examples of aortic regurgitation are provided, all taken from the parasternal long-axis view using color Doppler imaging. Mild (A), moderate (B), and severe (C) aortic regurgitation.

Video 10-57

FIGURE 10.58. A: The schematic demonstrates how the dimensions of the color jet of aortic regurgitation can be used to estimate severity. B: The jet height just below the aortic valve (black arrows) can be measured and compared with the dimension of the left ventricular outflow tract. This is a useful measure of severity.
Proximal isovelocity surface area, in theory, can be applied to any regurgitant valve to measure regurgitant area and volume. However, because of the technical challenges of visualizing the isovelocity shells that converge on the aortic regurgitant orifice, this technique has limited application to the aortic valve. Finally, an interesting approach to the quantification of severity of aortic regurgitation involves the concept of conservation of momentum. Momentum, the product of volumetric flow rate and velocity, is constant at any point within the regurgitant jet. Thus, as the jet expands in diastole to include a greater volume of blood, the velocity must decrease proportionately. Because flow is the product of the CSA and velocity, through substitution,

\[
\text{Momentum} = \text{Flow} \ (Q) \times \nu \text{ or } [\text{Eq. 10.10}]
\]

\[
\text{Momentum} = \text{Area} \times \nu^2 \quad [\text{Eq. 10.11}]
\]

To measure the ROA, momentum is determined at two points, one of which is at the regurgitant orifice. Because momentum is conserved, just like
mass, a form of the continuity equation is employed to yield

**FIGURE 10.60.** A very eccentric jet from a patient with bacterial endocarditis involving the aortic valve is shown. Because eccentric jets become entrained along the anterior mitral leaflet, severity may be underestimated. [Video 10-60](coming soon)
This is an attractive concept based on sound theoretical principles. By measuring the jet area and velocity at two points (one of which is within the regurgitant orifice), the ROA can be determined. The measurements are reasonably straightforward and reproducible, and in vitro studies have demonstrated the accuracy of this approach. However, jet momentum calculation remains a research tool and is difficult to apply clinically. A summary of the various approaches to measuring the severity of aortic regurgitation is provided in Table 10.9. It should be evident that no single measure of regurgitation severity is sufficient for clinical decision making. Each provides clues to severity but is imperfect and cannot be relied on in isolation. Instead, the clinician/echocardiographer must take into account all available data so that a comprehensive assessment of severity can be obtained.

**Acute Versus Chronic Aortic Regurgitation**

Several important differences exist between acute and chronic aortic regurgitation. The most common causes of acute regurgitation are endocarditis of the aortic valve (leading to disruption or destruction of the aortic leaflets) and aortic dissection (leading to annular and/or aortic root dilation or impingement of the dissection flap on the valve itself). Less commonly, chest trauma can result in this condition.

A primary difference between acute and chronic aortic regurgitation involves the response of the left ventricle. Over time, the left ventricle has a remarkable capacity to dilate, remaining compliant and accommodating even a large regurgitant volume while maintaining nearly normal diastolic filling pressures. This is not possible with acute aortic regurgitation in which the volume overload is poorly tolerated (due to the normal left ventricular size and the constraining effects of the pericardium) so that left ventricular diastolic pressure increases rapidly. The shape of the regurgitant jet envelope on continuous-wave Doppler imaging and especially the rate of deceleration of flow are perhaps the most useful hemodynamic markers to distinguish between the two (Fig. 10.67). In this example, the aortic regurgitation was the result of leaflet destruction from staphylococcal endocarditis. In acute aortic
regurgitation, the rapid increase in left ventricular diastolic pressure may also lead to premature closure of the mitral valve, which can be recorded using M-mode imaging (Fig. 10.41). Thus, echocardiography is critical to establish the cause of aortic regurgitation and to distinguish acute from chronic disease.

**FIGURE 10.61.** A: The parasternal long-axis view records the aortic regurgitant jet with color Doppler imaging. The height of the jet relative to the dimension of the left ventricular outflow tract suggests that the regurgitation is moderate. B: Taken from the same patient, the apical four-chamber view suggests mild aortic regurgitation. See text for details.

**Video 10-61**
FIGURE 10.62. This schematic illustrates how hemodynamic changes are reflected in the Doppler velocity tracing. **Left:** Mild aortic regurgitation (AR) is associated with a fairly flat contour of the regurgitant jet. **Right:** As severity increases, the slope of the jet becomes steeper. These changes are the result of the instantaneous pressure gradient between the aorta and the left ventricle during diastole. See text for details.
Assessing the Left Ventricle

In most patients, chronic aortic regurgitation is slowly progressive and is associated with a long asymptomatic period before surgical intervention is necessary. Because left ventricular dysfunction may precede the onset of symptoms, the longitudinal evaluation of patients with chronic significant aortic regurgitation focuses on the left ventricle. Several clinical studies, initially using M-mode echocardiography and later two-dimensional imaging, have demonstrated the value of serial studies in detecting the earliest signs of left ventricular decompensation in asymptomatic patients. These longitudinal series have confirmed that chronic aortic regurgitation is a slowly progressive condition and that patients with more severe disease progress more rapidly than those with mild or moderate regurgitation. However, more rapid and unexpected progression is possible. In Figure 10.68, a patient with mixed connective tissue disease is shown. In the first study, mild aortic regurgitation was present. Two years later, the regurgitation had become severe.

A variety of measures have been proposed to aid in clinical decision making. End-diastolic and end-systolic minor-axis left ventricular dimensions, ejection fraction, fractional shortening, and end-systolic wall stress have all been shown to predict outcome in patients with severe aortic regurgitation. When patients are initially evaluated, left ventricular systolic dysfunction thought secondary to aortic regurgitation is often an indication for surgical intervention. Among patients with preserved systolic function, an increase in chamber size, particularly the end-systolic dimension or volume, is generally regarded as an early manifestation of decompensation and frequently an indication for aortic valve replacement. Thus, the echocardiographic evaluation of these patients must pay particular attention to evidence of systolic dysfunction or progressive chamber enlargement. These parameters, together with the symptom status of the patient and his or her exercise capacity, provide most of the information needed for management decisions in aortic regurgitation. The indications for surgical intervention in patients with aortic regurgitation are summarized in Table 10.10.
FIGURE 10.63. Continuous-wave Doppler imaging of the aortic regurgitation (AR) jet permits quantitation of both slope and pressure half-time ($P_{1/2}t$). **Top:** An example of mild aortic regurgitation. The slope is relatively flat and the ($P_{1/2}t$) is long. **Bottom:** An example of severe aortic regurgitation demonstrates a much steeper slope and shorter $P_{1/2}t$.

FIGURE 10.64. Pulsed Doppler recording within the descending aorta (Desc Ao) from two patients (panels A and B) demonstrate flow reversal throughout diastole (arrows), suggesting severe aortic regurgitation. See text for details.
FIGURE 10.65. Stroke volume can be measured through any valve within the heart. This schematic demonstrates how stroke volume can be calculated at the level of the aortic valve (#1) and mitral valve (#2). The difference in stroke volume represents the regurgitant volume. In addition, the regurgitant fraction can be calculated. See text for details. CSA, cross-sectional area; TVI, time velocity integral.

- \[ \text{CSA}_{AV} = \pi r^2 = 0.785 \times D^2 \]
- \[ \text{SV}_{AV} = \text{CSA} \times \text{TVI} \]

- \[ \text{CSA}_{MV} = \pi r^2 = 0.785 \times D^2 \]
- \[ \text{SV}_{MV} = \text{CSA} \times \text{TVI} \]

Regurgitant volume = \( \text{SV}_{AV} - \text{SV}_{MV} \)
Regurgitant fraction = \( \text{RV}/\text{SV}_{AV} \times 100\% \)
FIGURE 10.66. An example of how regurgitant volume and regurgitant fraction can be quantified. In panels A and B, aortic stroke volume (SV) is calculated from the outflow tract area and pulsed Doppler recording. In C and D, the same calculation is performed for mitral SV. In this example, the aortic SV is 112 cc, while the mitral SV is 69 cc. The difference is the regurgitant volume (43 cc). See text for details. CSA, cross-sectional area; TVI, time velocity integral.

Aortic flow:
\[ CSA_{AV} = 3.1 \text{ cm}^2 \]
\[ TVI_{AV} = 36 \text{ cm} \]
\[ SV_{AV} = 112 \text{ cc} \]

Mitral flow:
\[ CSA_{MV} = 5.3 \text{ cm}^2 \]
\[ TVI_{MV} = 13 \text{ cm} \]
\[ SV_{MV} = 69 \text{ cc} \]

Regurgitant volume:
\[ 112 - 69 = 43 \text{ cc} \]

Regurgitant fraction:
\[ \frac{43}{112} = 38\% \]

FIGURE 10.67. An example of acute aortic regurgitation in a patient with endocarditis involving the aortic valve. A: Color Doppler imaging demonstrates severe aortic regurgitation. There is also evidence of diastolic mitral regurgitation (arrow) due to high diastolic left ventricular pressure. B: Continuous-wave Doppler imaging is consistent with severe regurgitation, based on the slope of the jet.
### Table 10.9  ESTIMATING THE SEVERITY OF AORTIC REGURGITATION

<table>
<thead>
<tr>
<th>Modality</th>
<th>Parameter</th>
<th>Criteria for Severe</th>
<th>Example of Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color flow</td>
<td>Jet area</td>
<td>&gt;60% LVOT area</td>
<td>Instrument (gain) dependent, eccentric jet, temporal variability</td>
</tr>
<tr>
<td></td>
<td>Jet height</td>
<td>&gt;60% LVOT height</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PISA</td>
<td>Effective regurgitation orifice area &gt;0.3 cm²</td>
<td>Multiple measurements, technically challenging</td>
</tr>
<tr>
<td></td>
<td>Vena contracta width</td>
<td>&gt;0.6 cm</td>
<td>Width may vary in different views</td>
</tr>
<tr>
<td>CW Doppler</td>
<td>Signal density $P^{1/2}t$</td>
<td>Nonquantitative &lt;250 ms</td>
<td>Affected by other factors, e.g., blood pressure, LV compliance acuity</td>
</tr>
<tr>
<td></td>
<td>Slope</td>
<td>&gt;400 cm/s²</td>
<td></td>
</tr>
<tr>
<td>Pulsed Doppler imaging</td>
<td>Regurgitant volume</td>
<td>&gt;60 mL</td>
<td>Requires multiple measurements, assumes no regurgitation at reference valve; limited quantitative information; affected by sample volume location</td>
</tr>
<tr>
<td></td>
<td>Regurgitant fraction</td>
<td>&gt;50%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Descending aortic flow reversal</td>
<td>Holodiastolic retrograde flow</td>
<td></td>
</tr>
<tr>
<td>2D</td>
<td>LV end-diastolic diameter</td>
<td>&gt;7 cm</td>
<td>Nonspecific, affected by multiple factors</td>
</tr>
</tbody>
</table>
Echocardiography
diastolic diastolic dimension LV end-
LV end-systolic systolic dimension >4.5 cm

CW, continuous wave; PISA, proximal isovelocity surface area; P1/2t, pressure half-time; 2D, two-dimensional.

FIGURE 10.68. Progression of severity of aortic regurgitation can be assessed using echocardiography. A: Mild aortic regurgitation (arrow). B: The same patient is evaluated 2 years later. During the interim, the severity of regurgitation (arrow) has increased dramatically. See text for details.  

Video 10-68
### Table 10.10  RECOMMENDATIONS FOR TIMING OF INTERVENTION IN AORTIC REGURGITATION

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
<th>Level of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVR is recommended for symptomatic patients with severe AR regardless of LV systolic function (stage D)</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>AVR is recommended for asymptomatic patients with chronic severe AR and LV systolic dysfunction (LVEF &lt;50%) (stage C2)</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>AVR is recommended for patients with severe AR who are undergoing other cardiac surgery (stage C or D)</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>AVR is reasonable for asymptomatic patients with severe AR and normal LV systolic function (EF ≥50%) but with severe LV dilation (LVESD &gt;50 mm) (stage C2)</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>AVR is reasonable in patients with moderate AR who are undergoing other cardiac surgery (stage B)</td>
<td>IIa</td>
<td>C</td>
</tr>
<tr>
<td>AVR may be considered for asymptomatic patients with severe AR and normal LV systolic function (EF ≥50%) but with progressive severe LV dilation (LVEDD &gt;65 mm) and low surgical risk (stage C1)</td>
<td>IIb</td>
<td>C</td>
</tr>
</tbody>
</table>

AR, aortic regurgitation; AVR, aortic valve replacement; EF, ejection fraction; LVEDD, left ventricular end-diastolic dimension; LVESD, left ventricular end-systolic dimension.

FIGURE 10.69. A parasternal long-axis view demonstrates an example of Lambl excrescence (arrow). [Video 10-69] coming soon
MISCELLANEOUS ABNORMALITIES OF THE AORTIC VALVE

Lambl excrescences are thin, delicate filamentous strands that arise from the ventricular edge of aortic cusps. Considered normal variants, these structures are seen increasingly with advancing age and improved image quality (Fig. 10.69). As such, they may represent a form of degenerative change of the valve that occurs over time. They can occasionally be multiple. An important goal in the evaluation of such structures is to distinguish a Lambl excrescence from pathologic entities, especially vegetations. This can be difficult and generally requires some consideration of the clinical setting. For example, if a patient has fever and positive blood cultures, a small aortic valve mass likely represents a vegetation. If the patient is afebrile and asymptomatic, the possibility of a Lambl excrescence should be strongly considered. Tumors affecting the aortic valve, such as fibroelastoma, are rare and are discussed in Chapter 21. An example of a papillary fibroelastoma involving the aortic valve is shown in Figure 10.70. This patient presented with an acute anterior myocardial infarction. Although the tumor appears small on transesophageal echocardiography, coronary angiography confirmed embolic disease to the left coronary artery.

FIGURE 10.70. A transesophageal long-axis view (A) and short-axis view (B) of the aortic valve from a patient with an embolic event to the left coronary artery are shown. The small, mobile mass attached to the aortic valve is a papillary fibroelastoma (arrow).
Video 10-70a

Video 10-70b

Suggested Readings


Sprigings DC, Chambers JB, Cochrane T, Allen J, Jackson G. Ventricular stroke work loss: validation


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Chapter 11
Mitral Valve Disease

The mitral valve was the first of the four cardiac valves to be evaluated with echocardiography. This was due to the relatively large excursion of the mitral valve leaflets, which made them an easier target for early M-mode techniques. The relatively high prevalence of rheumatic heart disease at the time also created the need for noninvasive assessment of a relatively common form of valvular heart disease. Modern two-dimensional and Doppler techniques have made echocardiography an essential tool in the management of patients with known and suspected mitral valve disease of all types. Three-dimensional echocardiography plays a unique and incremental role in mitral valve disease, often exceeding its benefit in other valve diseases.

Both primary and secondary mitral valve disease can be a major contributor to cardiovascular symptoms. Table 11.1 outlines the primary and secondary causes of mitral valve disease. These include congenital lesions such as congenital mitral stenosis and cleft valves and acquired valve disease such as rheumatic heart disease. Other forms of acquired heart disease, typically presenting later in life, include functional mitral regurgitation related to myocardial disease and degenerative lesions of the mitral valve and apparatus.

The most recent guidelines for management of patients with valvular heart disease recommend a system of staging similar to that utilized for patients with congestive heart failure. The stages of valvular heart disease describe the clinical status of patients with anatomic valvular abnormalities and are broken into four categories. Stage A is a patient who is at risk of developing clinical valvular heart disease. Patients in this subgroup are those with an anatomical abnormality of one of four cardiac valves but with no significant hemodynamic sequelae and no symptoms related to valvular heart disease.
Stage B represents patients with progressive valvular heart disease, defined as those who have an associated hemodynamic abnormality not (currently) resulting in either symptoms or abnormalities of cardiac physiology, other than the valve lesion itself. Stage C represents those with severe valvular abnormalities who are asymptomatic. These individuals will have secondary sequelae of valvular heart disease, such as chamber dilation. Stage D represents patients with severe valvular heart disease who are symptomatic. Table 11.2 outlines the stages of valvular heart disease and provides an illustrative example of how it would be employed in patients with mitral valve prolapse.

| Table 11.1 | ETIOLOGY OF MITRAL VALVE DISEASE |

<table>
<thead>
<tr>
<th>Diseases directly affecting the mitral apparatus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rheumatic heart disease</td>
</tr>
<tr>
<td>Congenital mitral stenosis</td>
</tr>
<tr>
<td>Congenital cleft mitral valve</td>
</tr>
<tr>
<td>Infectious endocarditis</td>
</tr>
<tr>
<td>Marantic endocarditis</td>
</tr>
<tr>
<td>Libman–Sacks endocarditis</td>
</tr>
<tr>
<td>Hypereosinophilic heart disease</td>
</tr>
<tr>
<td>Diet–drug valvulopathy</td>
</tr>
<tr>
<td>Mitral annular calcification</td>
</tr>
<tr>
<td>Degenerative</td>
</tr>
<tr>
<td>Infiltrative</td>
</tr>
<tr>
<td>Carcinoid</td>
</tr>
<tr>
<td>Myocardial ischemia or infarction/papillary muscle rupture</td>
</tr>
<tr>
<td>Tumors of the mitral valve</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Indirect effect on mitral valve function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dilated cardiomyopathy</td>
</tr>
<tr>
<td>Ischemic left ventricular dysfunction</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Bundle branch dyssynchrony</td>
</tr>
<tr>
<td>Left atrial myxoma</td>
</tr>
</tbody>
</table>
Echocardiography is the primary diagnostic tool for evaluating patients with all forms of known or suspected mitral valve disease. The published “Appropriateness Criteria for the Utilization of Transthoracic and Transesophageal Echocardiography” have defined multiple indications for the utilization of transthoracic and transesophageal echocardiography in patients with known or suspected mitral valve disease (Table 11.3). The range of patients evaluated for suspected mitral valve disease is substantial and includes those with murmurs of uncertain significance, as well as patients with congestive heart failure, ischemic heart disease, and dilated and hypertrophic cardiomyopathies as well as those with systemic diseases known to result in valvular heart disease.

**ANATOMY OF THE MITRAL VALVE**

The mitral valve is best thought of as an apparatus consisting of the mitral annulus, leaflets, chordae tendineae, papillary muscles, and the underlying ventricular wall (Fig. 11.1). Pathologic changes in any component of the mitral valve apparatus can result in mitral valve dysfunction. A classic form of mitral valve disease is rheumatic heart disease, which involves predominantly the leaflets and chordae. Other forms of mitral valve disease involve different aspects of mitral apparatus. Table 11.4 outlines the impact of different disease states on the different components of the mitral apparatus and the degree to which they result in mitral regurgitation or stenosis.

The mitral annulus is a complex three-dimensional structure. It is part of the fibrous skeleton of the heart, which also includes the aortic annulus and the junction of the anterior mitral valve leaflet and aorta (annuloaortic fibrosa). Three-dimensional echocardiography has demonstrated the nonplanar nature of the mitral annulus and the implications of this complex geometry for the diagnosis of mitral valve prolapse as well as for the design of therapeutic interventions such as mitral annuloplasty rings. Figures 11.2 and 11.3 depict the anatomy of the mitral annulus and its relationship to mitral leaflet closure patterns.
### Valve Prolapse

<table>
<thead>
<tr>
<th>Definition</th>
<th>Anatomy</th>
<th>Hemodynamics</th>
<th>Consequences</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A</strong> At risk</td>
<td>Mild MVP</td>
<td>No MR jet, MR area &lt;20%, Vena contracta &lt;3 mm</td>
<td>None</td>
</tr>
<tr>
<td><strong>B</strong> Progressive</td>
<td>Severe MVP with normal coaptation</td>
<td>MR jet 20–40%, RV &lt;60 mL, ERO &lt;0.40 cm², Grade 1–2+ on angio</td>
<td>Mild LA enlargement, Normal LV</td>
</tr>
<tr>
<td><strong>C</strong> Asymptomatic—severe</td>
<td>Severe MVP, abnormal coaptation or flail</td>
<td>MR jet &gt;40%, VC &gt;7 mm, RV &gt;60 mL, ERO ≥0.40 cm², Angio grade 3–4+</td>
<td>≥moderate LAE, LV dilation, Rest or exercise PHTN</td>
</tr>
<tr>
<td><strong>D</strong> Symptomatic—severe</td>
<td>Severe MVP with abnormal coaptation or flail</td>
<td>MR jet &gt;40%, VC &gt;7 mm, RV &gt;60 mL, ERO ≥0.40 cm², Angio grade 3–4+</td>
<td>≥moderate LAE, LV dilation, PHTN</td>
</tr>
</tbody>
</table>

### Table 11.3 Appropriateness Criteria for Use of Echocardiography in Mitral Valve Disease

<table>
<thead>
<tr>
<th>Myocardial Ischemia/Infarction with TTE</th>
<th>Appropriateness Score (1–9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>23. Suspected complication of myocardial ischemia/infarction, including but not limited to acute mitral regurgitation, ventricular septal defect, free-wall rupture/tamponade, shock, right ventricular involvement, HF, or thrombus</td>
<td>A (9)</td>
</tr>
<tr>
<td>32. Severe deceleration injury or chest trauma when valve injury, pericardial effusion, or cardiac injury are possible or suspected</td>
<td>A (9)</td>
</tr>
</tbody>
</table>

**Murmur or click with TTE**

| 34. Initial evaluation when there is a reasonable suspicion of valvular or structural heart disease | A (9) |
| 35. Initial evaluation when there are no other symptoms or signs of valvular or structural heart disease | rA (2) |
| 36. Reevaluation in a patient without valvular disease on prior echocardiogram and no change in clinical status or cardiac examination | rA (1) |
| 37. Reevaluation of known valvular heart disease with a change in clinical status or cardiac examination or to guide therapy | A (9) |

**Native valvular stenosis with TEE**

| 38. Routine surveillance (<3 yrs) of mild valvular stenosis without a change in clinical status or cardiac examination | rA (3) |
|   | Routine surveillance (≥3 yrs) of mild valvular stenosis without a change in clinical status or cardiac examination | A (7) |
|   | Routine surveillance (<1 yr) of moderate or severe valvular stenosis without a change in clinical status or cardiac examination | rA (3) |
|   | Routine surveillance (≥1 yr) of moderate or severe valvular stenosis without a change in clinical status or cardiac examination | A (8) |
| **Native valvular regurgitation with TEE** |   |   |
| 39. | Routine surveillance of trace valvular regurgitation | rA (1) |
| 40. | Routine surveillance (<3 yrs) of mild valvular regurgitation without a change in clinical status or cardiac examination | rA (2) |
| 41. | Routine surveillance (≥3 yrs) of mild valvular regurgitation without a change in clinical status or cardiac examination | U (4) |
| 42. | Routine surveillance (<1 yr) of moderate or severe valvular regurgitation without a change in clinical status or cardiac examination | U (6) |
| 43. | Routine surveillance (≥1 yr) of moderate or severe valvular regurgitation without a change in clinical status or cardiac examination | A (8) |
| **TEE as initial or supplemental test—valvular disease** |   |   |
| 44. | Evaluation of valvular structure and function to assess suitability for, and assist in planning of, an intervention | A (9) |
| **Chronic valvular disease—asymptomatic with stress echocardiography** |   |   |
| 177. | Mild mitral stenosis | rA (2) |
| 178. | Moderate mitral stenosis | U (5) |
| 179. | Severe mitral stenosis | A (7) |
| 183. | Mild mitral regurgitation | rA (2) |
| 184. | Moderate mitral regurgitation | U (5) |
| 185. | Severe mitral regurgitation; LV size and function not meeting surgical criteria | A (7) |
| **Chronic valvular disease—symptomatic with stress echocardiography** |   |   |
| 189. | Mild mitral stenosis | U (5) |
| 190. | Moderate mitral stenosis | A (7) |
| 191. | Severe mitral stenosis | rA (3) |
| 194. | Mild mitral regurgitation | U (4) |
| 195. | Moderate mitral regurgitation | A (7) |
| 196. | Severe mitral regurgitation; severe LV enlargement, or LV systolic dysfunction | rA (3) |

Anatomically, there are two major papillary muscles, each of which may have more than one head. The anterolateral papillary muscle provides chordae to the anterolateral half of both mitral leaflets. The posteromedial papillary muscle provides chordae to the posteromedial aspect of both leaflets. There is substantial variability in the exact number of chordae and
percentage of chords that are devoted to the anterior and posterior leaflets from each papillary muscle, but in general both papillary muscles provide chordal attachments to a portion of each of the leaflets. The posteromedial papillary muscle typically is perfused by the right coronary artery, and the anterolateral papillary muscle has a dual blood supply. Because of the dual blood supply of the anterolateral papillary muscle, it is less susceptible to ischemic injury than the posteromedial papillary muscle.

Figure 11.4 schematically depicts the detailed anatomy of the anterior and posterior mitral valve leaflets and different viewing perspectives. Each leaflet can be described as having three separate scallops termed anterior 1 through 3 (A1, A2, A3) and posterior 1 through 3 (P1, P2, P3). By definition, the A1 and P1 scallops are most anterolaterally located, nearest the left atrial appendage. The A3 and P3 scallops are more inferomedial in location. When viewed surgically from within the left atrium, the A1 and P1 scallops will be at the left of the surgeon’s field of view, whereas when viewed in an echocardiographic imaging plane, they will be to the right and inferior when viewed by transesophageal echocardiography. Rotation of the echocardiographic image 180 degrees results in an echocardiographic image which duplicates the “surgeon’s view.” This is especially useful when imaging the mitral valve with three dimensional imaging. Depending on the depth of the probe insertion and the angle of rotation, imaging planes can be obtained that will simultaneously view two or three scallops with two-dimensional imaging. Typically, when viewing the left ventricle in a longitudinal plane (120 degrees), the imaging plane intersects the A2/P2 boundary. Confusion may arise when imaging the mitral valve in a view orthogonal to this (60 degrees). In this view, the P1, A2, and A3 scallops may be simultaneously visualized. Because of this imaging plane, confusion may arise between a flail P3 and A3 scallop in this view. This may have relevance for feasibility of repair depending on available surgical expertise.

Three-dimensional echocardiography, especially when performed from a transesophageal route, is of proven value for evaluating mitral valve anatomy. From the left atrial or “surgeon’s view,” all six scallops of both leaflets can usually be simultaneously visualized. Scrupulous attention to technical detail is essential to avoid artifactual dropout. With this view, the coaptation points are easily visualized, and prolapsing or flail segments can be directly visualized, often with resolution such that even isolated chordal
rupture can be identified. Analysis programs are available which can create a schematic plane of the mitral annulus and the mitral leaflet tissue. This allows not only localization of prolapse or flail segments but quantitation of the actual volume of flail or prolapsing tissue. Anatomically, functional mitral regurgitation can be visualized with three-dimensional views and typically appears as areas of noncoaptation, from which the actual regurgitant orifice can be visualized and quantified by planimetry. The three-dimensional quantitation of the tenting area is also available with these techniques.

<table>
<thead>
<tr>
<th>Table 11.4</th>
<th>ANATOMIC CORRELATES OF DISEASE OF THE MITRAL VALVE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MS</td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td>✓</td>
</tr>
<tr>
<td>Congenital mitral stenosis</td>
<td>✓</td>
</tr>
<tr>
<td>Cleft mitral valve</td>
<td>✓</td>
</tr>
<tr>
<td>Bacterial endocarditis</td>
<td>✓</td>
</tr>
<tr>
<td>Coronary artery disease—myocardial infarction</td>
<td>✓</td>
</tr>
<tr>
<td>Diet—drug valvulopathy</td>
<td>✓</td>
</tr>
<tr>
<td>Mitral annular calcification</td>
<td>±</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>✓</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>✓</td>
</tr>
<tr>
<td>Myxoma</td>
<td>✓</td>
</tr>
<tr>
<td>Radiation</td>
<td>±</td>
</tr>
<tr>
<td>Infiltrative</td>
<td>✓</td>
</tr>
<tr>
<td>Carcinoid</td>
<td>✓</td>
</tr>
<tr>
<td>Papilloma</td>
<td>±</td>
</tr>
<tr>
<td>Metastatic disease</td>
<td>±</td>
</tr>
</tbody>
</table>

MS, mitral stenosis; MR, mitral regurgitation; ✓, common and primary involvement; ±, infrequent or late-stage involvement; *, rare abscess formation.
FIGURE 11.2. Schematic representation of a hypothetical planar mitral valve annulus (A) and the more accurate three-dimensional geometry of the annulus (B). In each set of schematics, the plane of the annulus is depicted as a dotted line and either a normal mitral valve or a mitral valve with mitral valve prolapse, depicted as viewed from orthogonal planes. A: Note that a planar annulus results in the same appearance of the mitral valve when viewed from two perspectives 90 degrees apart. Normal mitral closure is noted on the right and the bottom of each schematic and mitral valve prolapse at the top and left. Note that the normal valve closes with the belly of the leaflet slightly behind the plane of the annulus, irrespective of the viewing perspective, and that the prolapse valve bows to a substantially greater degree. B: Because of its complex three-dimensional shape, the annulus may be either concave or convex toward the apex of the left ventricle depending on the viewing perspective. With a normal closure pattern in the lower annular schematic, note that the leaflet does not protrude above the plane of the annulus. The schematic to the right represents the identical closing geometry of
the mitral valve, which now appears to prolapse behind the plane of the annulus because of its geometry in that perspective. The upper and leftward schematics depict the appearance of mitral valve prolapse as it relates to the saddle-shape geometry. In each instance, the geometry of the prolapse schematic is identical. Note the substantially greater degree to which prolapse is apparent on the left in (B) versus in (A), which is related to the different contour of the annulus when viewed from the orthogonal views. MVP, mitral valve prolapse.

**FIGURE 11.3.** Three-dimensional reconstruction of the mitral annulus from a transesophageal echocardiogram. Using this technique discrete points are identified, after which the annulus is tracked in three dimensions. Notice the nonplanar elliptical shape of the annulus. The anterior (A), posterior (P), anterolateral (AL), and posteromedial (PM) aspects of the annulus are as noted. The small circle at the anterior aspect denotes the plane of the aortic valve. The panel at the right of the figure provides detailed measurements of multiple annular dimensions. (This figure was recorded in a patient with prolapse of the middle posterior scallop as can be seen by the red encoded areas.)
FIGURE 11.4. Schematic representation of the mitral valve from multiple perspectives. **Bottom:** The view of the mitral valve in a surgical approach from inside the left atrium. **Top:** The mitral valve as viewed from a traditional transthoracic parasternal short-axis view. **Middle:** The mitral valve is seen from a transesophageal approach at the mid gastric level. In each instance, the proximal aorta is as noted in the schematic, as is the left atrial appendage. The three distinct scallops of the anterior and posterior leaflets (A1, A2, A3, P1, P2, P3) are also schematized. L, left coronary sinus; N, noncoronary sinus; R, right coronary sinus.

FIGURE 11.5. Expanded view of the mitral valve as seen from a transthoracic echocardiographic approach. This image corresponds to the top image of Figure 11.4. The imaging plane of a traditional transverse (0 degrees) plane and parasternal long-axis view (or 120-degree transesophageal echocardiographic view) are as noted. Note that when imaged from a 60-degree imaging plane (commissural view) with a transesophageal echocardiographic probe, the imaging plane will intersect the P1, A2, and P3 intersection. A1, A2, A3, anterior scallops 1 through 3; L, left coronary sinus; N, noncoronary sinus; P1, P2, P3, posterior scallops 1 through 3; PLAX, transthoracic parasternal long-axis plane; R, right coronary sinus; TEE, transesophageal echocardiography.
Figure 11.5 schematizes the relationship between the anterior and posterior leaflets to transesophageal echocardiographic imaging planes. Figures 11.6 through 11.9 depict normal transthoracic and transesophageal echocardiographic images recorded in various imaging planes outlining the relationship of the echocardiographic image to the anatomic mitral valve. Because of the curved “C shape” of the closed mitral valve, confusion may arise when dealing with a flail mitral valve leaflet. The “C-shaped” coaptation results in image planes in which alternating portions of the anterior and posterior leaflets may be visualized simultaneously (see the 60-degree plane in Figs. 11.5 and 11.9). It is not uncommon to detect multiple regurgitation jets in this view. Coaptation of the mitral leaflets is not isolated to the mitral valve tips but is the result of overlap of several millimeters of tissue (the zona coapta) (Figs. 11.8 and 11.10). Because of this, the closing force of the anatomically intact mitral valve increases with systolic pressure as the leaflets are forced to coapt along a longer portion of their terminal length. Any disease process that reduces the ability of the mitral valve to coapt along a several millimeter length results in inefficient or incomplete coaptation and subsequent regurgitation. It should be emphasized that disease processes occurring anywhere along the length of the mitral apparatus (from the annulus to the base of the papillary muscle and underlying ventricular wall) can result in malfunction of the mitral valve.
FIGURE 11.6. Parasternal long-axis view recorded in a structurally normal heart. The central image is recorded at end-systole and demonstrates the normal closure pattern of the mitral valve. The inset at the lower left is an expanded view of the closed mitral valve demonstrating the coaptation zone which extends for approximately 5 mm (arrows).

Video 11-6
FIGURE 11.7. Parasternal short-axis view (A) and transesophageal short-axis view from a transgastric position (B) recorded in normal patients. The positions of the aorta and left atrial appendage are as noted by the schematics. In each of these examples, recorded in diastole, the anterior (A) and posterior (P) leaflets of the mitral valve are clearly visualized and the three distinct regions (1 to 3) can be seen. For each imaging format, notice that the A1/P1 coaptation point is closest to the left atrial appendage and the A3/P3 coaptation closest to the ventricular septum. M, medial; L, lateral.

Video 11-7b

Mitral anatomy and motion are commonly defined by the Carpentier classification. This classification classifies the leaflets as either normal or abnormal, and furthermore defines their motion with respect to the mechanism of mitral regurgitation. Carpentier class I is defined as normal leaflet motion in which mitral regurgitation is functional due to annular dilation or perforation of a mitral leaflet. Class II is defined as leaflets with prolapse or loss of support and excess motion. Carpentier class III is defined as restricted leaflet motion and furthermore subdivided into IIIa in which motion is abnormal in diastole and IIIb in which it is abnormal in systole. Figure 11.11 depicts a variety of different abnormal mitral valve motion patterns and pathology, and where applicable the Carpentier classification for the pathology is noted on the illustration.
FIGURE 11.8. Apical four-chamber view recorded in systole in a normal patient. In this image, the normal closure pattern of the anterior and posterior leaflets of the mitral valve is clearly demonstrated. At the upper right, the closure pattern has been expanded. Note that the anterior and posterior mitral valve leaflets do not close tip to tip but rather along a 4-mm length (the zona coapta [ZC]).
FIGURE 11.9. Transesophageal echocardiogram recorded at 66 degrees. In this view, the P1, A2, and P3 scallops are clearly visualized (A). B: Note the two separate mitral regurgitation jets (arrows) arising from the P1-A2 and P3-A2 commissures. A1, A2, A3, anterior scallops 1 through 3; P1, P2, P3, posterior scallops 1 through 3. [Video 11-9a, Video 11-9b]
FIGURE 11.10. Anatomic rendering of the normal mitral valve in a closed position. Again note that the chordae attach not only to the leaflet tips but to the belly of the leaflet as well. Also note that the normal mitral valve does not close in a tip-to-tip manner but that there is an overlap of the leaflets as they close (the zona coapta). (Artwork by Amanda Almon and Travis Vermilye.)
Video 11-10
FIGURE 11.11.  Schematic drawings demonstrate a normal mitral valve closure pattern (upper left) and multiple different pathologic closure patterns. In each example, the annulus (small black dot) and proximal ventricular wall are denoted. At the point of the intended coaptation, the open circle denotes the regurgitant orifice and the arrow denotes the direction of the regurgitant flow. The dotted lines denote the mitral valve chordae. The designation “C-I” refers to the appropriate
Carpentier classification of pathology.

**PHYSIOLOGY OF MITRAL VALVE DISEASE**

Physiologic abnormalities of mitral valve disease can be classified as stenosis, regurgitation, and a combination of the two. A classic form of mitral valve disease is rheumatic mitral stenosis in which predominately the leaflet tips and chordae are involved. This results in obstruction of flow from the left atrium to the left ventricle and a diastolic transvalvular gradient. This increases left atrial pressure, which is transmitted to the pulmonary veins and pulmonary capillary bed. This elevated pressure then translates to an increased driving force for transudation of fluid into the alveoli and development of pulmonary congestion. Typically, with normal plasma oncotic pressure, fluid extravasation into the alveoli occurs at a pulmonary capillary pressure of approximately 24 mm Hg. Extravasation of fluid into the alveoli interrupts pulmonary gas exchange and results in dyspnea, initially with exercise but subsequently at rest, and may lead to secondary pulmonary hypertension. Development of pulmonary hypertension in the presence of increased pulmonary venous pressure is initially due to increased pulmonary vasoreactivity. Over time, fixed anatomic changes occur in the pulmonary vascular bed. Chronically elevated left atrial pressure results in secondary dilation of the left atrium. Over time, this results in progressive fibrosis in atrial myocardium with a subsequent decrease in atrial contractility, stasis of blood, and the potential for atrial fibrillation and thrombus formation.

**MITRAL STENOSIS**

In adults, the etiology of mitral stenosis is most often rheumatic heart disease. Often adult patients with rheumatic mitral stenosis have no recognized history of rheumatic fever, but the morphology of the valve permits establishment of a diagnosis of antecedent rheumatic fever. Mitral annular calcification may progress to the point that it obstructs mitral valve inflow and physiologically mimics rheumatic mitral stenosis. A less common etiology of mitral stenosis is congenital mitral stenosis. Tumors such as left atrial myxoma have been described as mimicking mitral stenosis, but
presentation as occult mitral stenosis by a myxoma is exceptionally rare.

**Two-Dimensional Echocardiography in Rheumatic Mitral Stenosis**

The classic echocardiographic and anatomical features of rheumatic mitral stenosis are thickening and fusion of the mitral valve commissural edges and chordae with relative sparing of the body of the leaflets. This results in characteristic abnormalities of mitral leaflet opening. Normally, the anterior and posterior leaflets open with a pattern that involves maximal excursion at the leaflet tips with substantially greater excursion of the anterior leaflet. In mitral stenosis, due to commissural and chordal fusion, the leaflets open with a “doming” motion. The anterior leaflet has also been described as having a “hockey stick” appearance in diastole. This results in reduction of the orifice area and conversion of the mitral leaflets from a tubular channel to a funnel-shaped orifice. As a result, the limiting factor in flow from the left atrium to the left ventricle is the orifice of the mitral valve and chordae at their junction. The degree of chordal thickening and mitral valve commissural fusion is highly variable. Over time, there is progressive fibrosis at the initial site of fusion as well as throughout the more distal chordae and more proximal leaflets. Eventually, this results in stiffening and calcification of these structures. Figures 11.12 through 11.17 were recorded in patients with rheumatic mitral valve involvement. Note in Figure 11.12 the mild diffuse thickening of the mitral leaflets with restricted motion at the tips resulting in doming of the mitral valve (arrows). The orientation of the more proximal anterior leaflet and distal tips takes on a “hockey stick appearance.” In Figure 11.13 recorded from a transesophageal echocardiogram in the same patient presented in Figure 11.12, note the relatively symmetric, oval-shaped orifice (X) and the diffuse thickening of multiple chordae (arrows). Figures 11.14 through 11.16 depict variable degrees of valvular thickening, fibrosis, and leaflet mobility.
FIGURE 11.12. Transthoracic parasternal long-axis echocardiogram recorded in a patient with rheumatic mitral stenosis. In this image, recorded in early diastole, note the doming motion of the anterior mitral valve leaflet with restriction of motion at the tips. The belly of the leaflet (arrows) is pliable, and there is little or no fibrosis, calcification, or thickening of the leaflets. Also note the secondary dilation of the left atrium. In the real-time image, note the relatively fixed position of the leaflet tips with all motion of the leaflet occurring at the mid and proximal portions of the leaflets. 

Video 11-12

coming soon
While mitral stenosis is the hallmark of classic rheumatic heart disease, it should be appreciated that rheumatic disease can involve any of the four cardiac valves. The mitral valve is by far the most commonly affected, followed by the aortic valve (Fig. 11.17). While stenosis is the classic finding of rheumatic mitral valve disease, concurrent regurgitation is not uncommon. Disease of the tricuspid valve can be either due to primary rheumatic involvement, or more commonly functional and secondary to pulmonary hypertension. Concurrent aortic valve disease or mitral regurgitation has significant implications regarding assessment of the severity of mitral stenosis by Doppler techniques.

**Congenital Mitral Stenosis**

Congenital mitral stenosis is infrequently encountered in adult practice. There are two forms of congenital mitral stenosis. The first is the “parachute” mitral valve. It typically occurs in conjunction with a single papillary muscle to which all chordae of an otherwise normal valve attach. This limits the mobility of the leaflets and results in restriction of inflow to a variable degree. The second type of congenital mitral stenosis is due to an anatomic abnormality of the valve and chordae resulting in a combination of reduced mobility and an intrinsic reduction in the anatomic orifice due to abnormal leaflet morphology. A double-outlet mitral valve is a final variation of congenital mitral stenosis. This is discussed further in Chapter 19.
FIGURE 11.13. Short-axis view of the left ventricle recorded from the lower esophageal position from transesophageal echocardiogram in a patient with rheumatic mitral stenosis. This image was recorded in diastole and nicely demonstrates the restricted oval-shaped orifice (X). Also note the thickening of the chordal apparatus (small arrows). This image was recorded in the same patient presented in Figure 11.12. [Video 11-13]
FIGURE 11.14. Parasternal long-axis (A) and short-axis (B) transthoracic echocardiograms recorded in a patient with rheumatic mitral stenosis. A: Note the marked thickening of the chordae throughout their entire length, from the mitral leaflet to the papillary muscles (arrows). In the short-axis view (B), the slit-like orifice of the mitral valve is visualized.

Video 11-14a

Video 11-14b

M-Mode Echocardiography

M-mode echocardiography was one of the early tools used for the evaluation of rheumatic mitral valve disease. The hallmark of rheumatic heart disease on M-mode echocardiography was increased echogenicity of the leaflets with decreased excursion and reduced separation of the anterior and posterior leaflets. This was accompanied by a reduced diastolic (E-F) slope of mitral
closure (Fig. 11.18). The E-F slope, measured in millimeters per second, is inversely correlated with the severity of mitral stenosis and improved (i.e., became steeper) after successful open commissurotomy. The E-F slope ultimately proved to be nonspecific and was noted in situations in which left ventricular filling was impaired such as in diastolic dysfunction. The E-F slope is of more historical than clinical value today. Additional features of mitral stenosis noted on M-mode echocardiography included “paradoxical” anterior diastolic motion of the posterior mitral valve leaflet. This occurred because tethering at the tips resulted in an obligatory anterior motion of the posterior leaflet tips that tethered to the larger anterior leaflet.

FIGURE 11.15. Apical four-chamber view recorded in a patient with rheumatic mitral stenosis. This image was recorded in diastole and confirms the restriction of motion of both mitral leaflets. The inset is the transmural Doppler demonstrating a mean pressure gradient of 9 mm Hg.
Video 11-15

coming soon

Video 11-16

coming soon
FIGURE 11.16. Apical four-chamber view recorded in a patient with rheumatic mitral stenosis and heavy fibrosis and calcification, predominantly of the posterior leaflet (downward-pointing arrows). Note the markedly dilated left atrium. Continuous-wave Doppler confirms a mean pressure gradient of 11 mm Hg.
FIGURE 11.17. Transesophageal echocardiogram recorded in a patient with rheumatic heart disease with involvement of both the mitral and aortic valves. The central illustration was recorded in diastole. Notice the diffuse thickening and doming motion of the mitral valve. The aortic valve cusps are also noted to be thickened. Incidental note is made of significant spontaneous echo contrast in the left atrium. The inset at the upper right was recorded at mid systole. Notice the restricted motion of the aortic valve consistent with aortic stenosis. [Video 11-17]
FIGURE 11.18. M-mode echocardiogram recorded in a patient with rheumatic mitral stenosis. Note the marked thickening of the mitral valve leaflets and the flat E-F slope during diastole. The posterior leaflet appears to move anteriorly in diastole as well (arrows).

**Transesophageal and Three-Dimensional Echocardiography**

Transesophageal echocardiography is often essential for the complete evaluation of patients with rheumatic mitral stenosis (Figs. 11.19 to 11.24). It allows a refined assessment of mitral valve and chordal anatomy and obviously allows assessment of the presence or absence of secondary findings, such as thrombus in the left atrial appendage. When viewed exclusively from a left atrial aspect, the degree of chordal involvement may be understated. Transgastric planes are often necessary to provide detailed visualization of the chordal apparatus. It is important to recognize that the cardiac physiology in a sedated, fasting patient may be different from that in a nonfasting, awake patient and that there may be discrepancies between the transmitral valvular gradients obtained at transesophageal echocardiography and those obtained in an ambulatory, awake patient, with typically higher gradients noted in the latter.
FIGURE 11.19. Transesophageal echocardiogram recorded in a patient with rheumatic mitral stenosis. In this four-chamber equivalent view, note the dilated left atrium, as well as the diffuse thickening of the mitral valve leaflets and, in this mid-diastolic frame, the restricted motion of the leaflets and the narrowed orifice (arrow). The inset at the upper right is a three-dimensional echocardiogram recorded from this patient from a left atrial view. Note the restricted but fairly symmetric, oval-shaped mitral orifice (dotted lines), from which a mitral valve area of 0.8 cm$^2$ could be calculated. In the lower right is the transmitral Doppler demonstrating a mean transmitral gradient of 14 mm Hg.
Video 11-19 3D

coming soon

Video 11-19
FIGURE 11.20. Transesophageal echocardiogram recorded from a 126-degree imaging angle from behind the left atrium. Note the doming of the mitral valve in diastole and the color flow convergence zone within the left atrium (arrows) as flow accelerates toward the restricted orifice. The continuous-wave Doppler through the restricted orifice is also presented revealing mean transvalvular gradients of 10 and 6.5 mm Hg for the shorter and longer cycles in this patient with atrial fibrillation. MPG, mean pressure gradient.

Video 11-20
FIGURE 11.21. Transesophageal echocardiogram recorded in a patient with classic rheumatic mitral stenosis. Note the dilated left atrium and the thickening at the tips of the mitral valve. The lower right inset is the three-dimensional echocardiogram recorded from this patient from a perspective of within the left atrium. Note the slightly irregular mitral valve orifice (black arrows).
FIGURE 11.22. Transthoracic three-dimensional echocardiogram recorded in a patient with rheumatic mitral stenosis. On the left are three extracted images, including a four-chamber (4C), two-chamber (2C), and short-axis (Sx) views oriented at the level of the restricted mitral valve orifice. The main image shows the three-dimensional image of the mitral valve orifice (arrows).

Video 11-22

Three-dimensional echocardiography plays an incremental role in
assessment of mitral valve anatomy and mitral stenosis and has seen the greatest impact when used from a transesophageal approach. With three-dimensional echocardiography, it is often possible to directly visualize the true anatomical mitral valve orifice of the stenotic valve, as the imaging plane can be adjusted offline to correspond to the true limiting orifice. Concurrent mitral regurgitation can also be accurately assessed.

**FIGURE 11.23.** Transesophageal echocardiogram recorded in a patient with severe mitral stenosis and marked diffuse thickening of both the anterior and posterior mitral leaflets (*arrows*). The smaller image at the upper right is the three-dimensional image recorded from the left atrial perspective demonstrating a slit-like mitral valve orifice (*arrows*). In the lower left is the three-dimensional image of the mitral valve orifice recorded from the perspective of the left ventricle. The actual orifice is noted by the small “X.” Note the marked irregular thickening of the anterior mitral leaflet and chordae (*arrows*).
FIGURE 11.24. Transesophageal echocardiogram recorded in a patient with rheumatic mitral valve disease and combined stenosis and regurgitation. Note the dilated left atrium and in this mid-systolic frame the mitral regurgitation jet (arrows) which appears to arise from two separate points. The image at the upper left is a three-dimensional echocardiogram from the left atrial perspective recorded at mid diastole revealing an irregular stenotic mitral orifice (black arrows). The image at the upper right is a three-dimensional image recorded at mid systole, again from the left atrial perspective. Note the failure of complete coaptation and the slit-like regurgitant orifice (black arrows).
Anatomic Determination of Severity

M-mode, two-dimensional, and three-dimensional echocardiography have all been used for the determination of the severity of mitral stenosis. As noted previously, M-mode echocardiography relied on determination of leaflet thickness and the E-F slope as indirect measures of leaflet restriction. Although previously used for serial follow-up, M-mode echocardiography provided no quantitative data regarding the actual restrictive orifice.

Using two-dimensional echocardiography from a parasternal short-axis view, it is possible to visualize the actual restrictive orifice of the stenotic mitral valve at its limiting orifice (Fig. 11.25). In patients with relatively symmetric involvement, the orifice area can accurately be planimetered. This measurement correlates well with that determined from hemodynamic data. There are several technical factors that must be accounted for in determining orifice area from this approach. First, one should recognize that, in mitral stenosis, the mitral valve represents a funnel-shaped structure that tapers to its limiting orifice at the tips, and careful scanning must be performed to ensure that the image is frozen and planimetered at the mitral valve tips and not more proximally where the orifice area would be overstated (Fig. 11.26). Second, instrumentation gain and other settings affect accurate visualization of the limiting orifice. Increased gain will result in a “blooming” of the echoes, which then overstates their boundary and thereby diminishes the visualized orifice. When appropriately recorded, the measured orifice area correlates very well with that determined by hemodynamics. After commissurotomy, the orifice often becomes more irregular and the area of the commissural opening may be difficult to planimeter accurately. Three-dimensional echocardiography allows more precise localization of the actual flow limiting orifice, especially if the orifice is not aligned with the long axis of the left ventricle. As such it may provide a more accurate assessment of the stenotic orifice (Figs. 11.19, 11.21 to 11.24).

Doppler Echocardiographic Determination of Severity

Multiple Doppler parameters are useful for assessing the severity of mitral stenosis (Fig. 11.27). Doppler echocardiography can be used to determine the
left atrial to left ventricular transvalvular gradient, which is the single most important factor in determining the physiologic significance of mitral stenosis. If one understands the hemodynamic and physiologic principles noted previously, then the overall hemodynamic effect of mitral stenosis can be derived from the transthoracic echocardiogram. It should be recognized that the transmitral gradient plus the anticipated left ventricular diastolic pressure equals the left atrial pressure. Left atrial, pulmonary venous, and pulmonary capillary pressures are all similar and represent the hydrostatic driving pressure leading to pulmonary congestion. The pressure gradient is dependent on volume status, forward flow volume, and heart rate, which affects filling time. Determination of the pressure gradient, and its overall relevance to left atrial pressure should play an equal role to determination of anatomical mitral valve area in management.

FIGURE 11.25. Parasternal short-axis views recorded in patients with rheumatic mitral stenosis. In each instance, note the restricted mitral valve orifice. A: The orifice can be planimetered as 1.3 cm². In this example, note the localized thickening of the chordae at the anterolateral border of the mitral orifice (arrows). B: Recorded in a patient with more severe stenosis. The mitral orifice has been planimetered at 0.7 cm². Also note the diffuse nature of thickening around the mitral orifice. MVO, mitral valve orifice.
In most patients, the mitral Doppler inflow is easily recorded from the transthoracic apical view (Fig. 11.28). It can often be recorded in individuals in whom two-dimensional scanning provides suboptimal anatomic definition of the mitral valve. The transmitral gradient should be recorded using continuous-wave Doppler imaging aligned as parallel as possible to the direction of flow. If pulsed-wave Doppler imaging is used, it is essential that the sample volume be placed at the level of the restrictive orifice and not further back near the annulus. Placement of the sample volume near the annulus will result in systematic underestimation of the transmitral gradient. In general, rheumatic mitral stenosis results in a central stenotic orifice with flow directed from the left atrium toward the apex of the left ventricle. As
such, traditional two- and four-chamber viewing planes usually suffice for measurement. If necessary, color flow imaging can be used to determine the direction of flow and further refine this assessment. The peak and mean pressure gradient can be obtained online by electronic planimetry of the spectral profile (Fig. 11.28). Atrial fibrillation with an irregular heart rate poses additional problems. Depending on heart rate, there may be dramatic variation in diastolic filling time and mean transvalvular gradient. Multiple cycles should be averaged to provide an accurate assessment of severity (Figs. 11.20 and 11.29).

**FIGURE 11.26.** Series of parasternal short-axis views recorded in a patient with rheumatic mitral stenosis. A: Recorded at the actual restrictive orifice, and the mitral valve area (MVA) can be planimetered at 0.9 cm$^2$. B–D: The three additional views were recorded progressively closer to the annulus and show a progressive increase in the planimetered mitral orifice depending on the position at which the “orifice” is planimetered.
FIGURE 11.27. Schematic representation of mitral valve inflow depicting different parameters that can be extracted for determination of the severity of mitral stenosis. In the schematic, note the relatively flat decay of pressure from the E point. Parameters that can be measured include integration of the overall pressure gradient beneath the spectral display to calculate the mean pressure gradient (MPG) as well as calculation of mitral valve area (MVA) from the pressure half-time method. For the pressure half-time method, the time required for the pressure to decay from its peak value (16 mm Hg in this example) to one-half of that value (8 mm Hg) is determined. The velocity at which the gradient has declined to one-half its peak can be calculated as $0.7 \times V_{\text{MAX}}$. This value (400 ms in this example) is then entered into the equation $\text{MVA} = \frac{220}{P_{t_{1/2}}}$. In the schematic, the MVA calculates to 0.6 cm$^2$. PPG, peak pressure gradient.
FIGURE 11.28. Transmitral gradient recorded in a patient with rheumatic mitral stenosis. In the left-hand complex, the deceleration of mitral inflow has been measured, from which a pressure half-time of 225 ms, corresponding to a mitral valve area of 0.98 cm$^2$, has been calculated. In the right-hand cycle, the transmitral gradient has been measured to be 5 mm Hg.
FIGURE 11.29. Transmitral continuous-wave Doppler image recorded in a patient with mitral stenosis in atrial fibrillation with an irregular ventricular response. A: Note the marked variation in diastolic filling time and the obvious variation in the spectral profile. B: Recorded in the same patient, revealing three different diastolic filling profiles. Note the marked variation in the mean pressure gradient, dependent on diastolic filling time.

An additional feature is the rapidity with which the instantaneous pressure gradient decays over time. It was recognized relatively early in the
hemodynamic laboratory that individuals in whom the pressure gradient persisted to the end of diastole had more severe stenosis than those individuals in whom the pressure gradient declined to near zero at end-diastole. A measure of the rate of decay of the mitral valve gradient is pressure half-time ($P_{1/2}t$), or the time in milliseconds at which the initial instantaneous pressure gradient declines to one-half of its maximum value. The mathematical calculation of $P_{1/2}t$ is depicted in Figure 11.27 and an example of its utilization is presented in Figure 11.28. Empirically, $P_{1/2}t$ is related to the mitral valve area by the formula: mitral valve area = $220/P_{1/2}t$. There are several technical factors that should be noted. First, the initial validation was done in a very small number of patients with anatomical or hemodynamic correlations. Second, the $P_{1/2}t$ calculation represents the “pressure decay” between the left atrium and left ventricle, and will be affected by any factor that changes either left atrial driving pressure or left ventricular compliance and pressure. Situations in which the latter can be altered include left ventricular hypertrophy or concurrent aortic insufficiency, in which there is competitive filling of the left ventricle. Concurrent aortic insufficiency results in overestimation of mitral valve area by the $P_{1/2}t$ method (Fig. 11.30). In many instances, the mitral stenosis signal does not have a uniform slope but may have an early rapid decay followed by a more gradual decay, giving a “ski slope” appearance. In this instance, caution is advised, but the more accurate reflector of area will be derived from the flatter portion of the spectral envelope. In general, the derived anatomic area from the $P_{1/2}t$ calculation is often less valuable for patient management than determination of pressure gradients and anatomically measured valve areas.

Although the mean pressure gradient is directly related to the average area of the restrictive orifice and cardiac output, the peak instantaneous early pressure gradient between the left atrium and left ventricle is also related to the early transmural flow volume. Early flow volume is dependent on cardiac output and also affected by high early left atrial volumes, as may be seen with concurrent mitral regurgitation or high-output states. In the presence of mitral regurgitation or high cardiac output, there is a disproportionate increase in the early transvalvular velocity and gradient compared with the mean mitral valve gradient. On occasion, this exaggerated early pressure gradient, compared with the mean pressure gradient, can be a clue to the presence of
concurrent mitral regurgitation in situations in which the mitral regurgitation may not be directly visualized. This observation may be of particular value in patients with highly eccentric regurgitation jets or paravalvular regurgitation in a mitral prosthesis.

**FIGURE 11.30.** Impact of aortic insufficiency on calculation of mitral valve area by the pressure half-time method. In the central image note the stenotic area of approximately 0.8 cm². At the upper left is a recording of the transmitral Doppler velocity recorded at the tips of the mitral valve from which a mitral valve area of 1.07 cm² is calculated by the pressure half-time method. The image at the upper right is recorded in an apical long-axis view confirming the presence of moderate aortic insufficiency. In this instance the aortic insufficiency results in a more rapid equalization of transmitral velocities for any given level of stenosis and therefore results in the pressure half-time method systematically understating the severity of mitral stenosis.
Video 11-30 AI

coming soon

Video 11-30

coming soon
FIGURE 11.31. Echocardiogram recorded in a patient with rheumatic heart disease and mixed mitral stenosis and regurgitation. In the central illustration note the mild to moderate mitral regurgitation jet. At the upper right inset note the three-dimensional echocardiographic depiction of the actual stenotic mitral orifice which can be quantified at 1.18 cm². The inset at the upper left is continuous-wave Doppler through the stenotic mitral orifice where a mean pressure gradient of 21 mm Hg is noted. This level of pressure gradient is substantially greater than what would be anticipated from a mitral orifice of 1.18 cm² and is related to the concurrent mitral regurgitation which results in a high transmural flow volume thus accentuating the gradient for any level of stenosis.
It is not uncommon to encounter patients with a combination of both mitral stenosis and regurgitation. The clinician should recognize that the transmitral gradient is dependent both on anatomical restriction of the orifice area, as well as the volume of flow. In the presence of concurrent mitral regurgitation, the diastolic transmitral gradient will be increased for any given valve area compared to that seen in pure isolated mitral stenosis (Fig. 11.31). This may result in a discrepancy between the severity of symptoms and the measured orifice area. In these instances, it is critically important that the clinician incorporate all available data, including the presence of secondary pulmonary hypertension and the behavior of the transmitral gradient at variable heart rates. While the current valvular heart disease guidelines provide specific recommendations for management of isolated mitral stenosis and mitral regurgitation, less data are available to provide the clinician guidance with respect to mixed stenotic and regurgitant states.

**Exercise Gradients**

By measuring the transmitral gradient with exercise, valuable information can be obtained regarding the physiologic impact of mitral stenosis. When high transmitral gradients are measured at rest, clinical dilemmas regarding the clinical relevance of mitral stenosis are uncommon. Occasional patients are encountered in whom there is a moderate resting gradient of 6 to 8 mm Hg but who have substantial clinical impairment. Limited exercise such as 30 to 60 seconds of leg lifts increases the heart rate, which shortens diastolic filling time and therefore results in an increase in the transmitral gradient. Transmitral gradients can then be compared with values obtained at rest. Figures 11.32 and 11.33 are examples in which transmitral gradients were recorded at rest and again after 30 seconds of leg lifts. The gradient measured at rest is unimpressive but increased dramatically with limited exercise. Keeping in mind the physiologic relationship between this transvalvular gradient and pulmonary capillary pressures, one can then surmise valuable information regarding the physiologic abnormalities present in this patient after limited exercise and establish a link between the mitral valve disease and symptoms. Finally, Doppler of the tricuspid regurgitation jet can be used
to assess for exercise-induced pulmonary hypertension.

**FIGURE 11.32.** Transmitral gradient recorded at rest (*upper panel*) and immediately following exercise (*lower panel*) in a patient with rheumatic mitral valve disease. At rest, note the mean pressure gradient of 8 mm Hg at a heart rate of 70 beats per minute. In the lower panel, note the increase of the gradient to a mean gradient of 22 mm Hg at a heart rate of 139.

**Secondary Features of Mitral Stenosis**
Chronic elevation in left atrial pressure results in left atrial dilation and eventual fibrosis of the atrial myocardium. Over time, this results in decreased atrial contraction and serves as a substrate for the development of atrial fibrillation. Atrial fibrillation may be either intermittent or persistent. In the presence of atrial fibrillation there is a loss of organized mechanical activity of the left atrium. This increases the tendency to form thrombus. Dilation of the left atrium occurs both in the atrial body and in the left atrial appendage. The combination of atrial and atrial appendage dilation with decreasing mechanical function results in stasis of blood with an enhanced propensity to thrombus formation, most commonly in the left atrial appendage. The tendency to develop stasis and clot is markedly increased in the presence of atrial fibrillation. Using either high-resolution transthoracic imaging or more often transesophageal imaging, it is not uncommon to see varying degrees of stasis of the blood in the atrium of patients with mitral stenosis. This typically appears as a swirling mass of echoes in the body of the left atrium, referred to as spontaneous echo contrast, and is often maximal in the left atrial appendage. Figures 11.34 and 11.35 were recorded in patients with mitral stenosis and varying degrees of spontaneous echo contrast and thrombus formation within the left atrium and left atrial appendage. Current opinion suggests that dense spontaneous echo contrast and stasis of blood are precursors to thrombus formation and are markers of a patient with increased thromboembolic risk, especially if seen in the presence of atrial fibrillation. Using pulsed Doppler, it is common to see reduced atrial appendage velocities in this setting (Fig. 11.36).

When evaluating a patient for a possible left atrial appendage thrombus, it is important to recognize the range in anatomic variability of the atrial appendage. Traditionally, the left atrial appendage has been considered a single-lobe structure with varying degrees of trabeculation due to pectinate muscles (Fig. 11.37). It is well recognized that the left atrial appendage has multiple lobes in a substantial percentage (>30%) of patients (Figs. 11.38 and 11.39). This raises several concerns when evaluating patients for a left atrial appendage thrombus. The first is that all lobes of the appendage must be identified and examined. The second issue is the need to recognize the septation tissue between appendage lobes as normal tissue and not as protruding thrombus.
FIGURE 11.33. Doppler mitral valve inflow and tricuspid regurgitation velocity recorded in a patient with mitral stenosis at rest (upper panels) and immediately following exercise (lower panels). Note the mean resting gradient of 6 mm Hg across the mitral valve. The tricuspid regurgitation profile recorded at rest reveals a right ventricle-to-right atrial pressure gradient of 34 mm Hg. For the mitral inflow pattern recorded immediately following exercise note the increase in the mean transmural gradient to 32 mm Hg at a heart rate of 126. The tricuspid regurgitation velocity recorded confirms an elevation of the right ventricle-to-right atrial pressure gradient of 57 mm Hg consistent with exercise-induced elevation and pulmonary artery pressures.
FIGURE 11.34. Transesophageal echocardiogram recorded in a patient with mitral stenosis and atrial fibrillation. A: Note the spontaneous contrast of the body of the left atrium (arrows). B: Note the aneurysmal atrial septum (arrows) which is filled with dense spontaneous contrast versus soft thrombus (arrows).
As a part of surgical correction of mitral valve disease and/or a maze procedure, the left atrial appendage may be surgically resected or ligated in an effort to reduce the likelihood of cardioembolic complications. This can either be performed by actual surgical amputation or ligation. Recent data suggest that in more than half of such procedures, the left atrial appendage may not remain fully closed and either a residual stump or partial opening into a left atrial appendage persists (Fig. 11.40). As such, the degree to which this procedure has reduced the potential for left atrial thrombus formation may be uncertain but can be confirmed with transesophageal echocardiography.

**FIGURE 11.35.** Transesophageal echocardiogram recorded in a patient with rheumatic mitral stenosis and atrial fibrillation. In this instance there is a firm thrombus filling the majority of the left atrial appendage. The black arrows denote the outer boundary of the left atrial appendage which is filled by a firm thrombus (white arrow).
FIGURE 11.36. Pulsed Doppler image recorded from the left atrial appendage in a patient with mitral stenosis and atrial fibrillation. Note the high-frequency, low-velocity signals (<20 cm/s), indicative of reduced mechanical transport in the left atrial appendage.
FIGURE 11.37. Transesophageal echocardiogram recorded in a patient with a normal left atrial appendage. Note the prominent pectinate muscles within the body of the left atrium (arrows). [Video 11-37]
FIGURE 11.38. Transesophageal echocardiogram recorded in a patient with complex left atrial appendage anatomy. In the central figure, recorded at 85-degree imaging plane there is a normal-appearing single lobe left atrial appendage (arrow). The panel at the lower left is recorded at 142 degrees where a distinct side lobe (upward-pointing arrow) is visualized. The body of the left atrial appendage is noted by the longer downward-pointing arrow. [Video 11-38A coming soon]
The fibrillatory mechanical activity of the atrium can be appreciated by either two-dimensional visualization or M-mode echocardiography of the left atrial wall. In addition, Doppler echocardiography at the mouth of the atrial appendage reveals indirect evidence of the reduction in mechanical force due to atrial fibrillation. In Figure 11.36, note the high-frequency but low-velocity signals recorded by pulsed Doppler imaging at the mouth of the left atrial appendage. This represents a marked reduction in velocity and volume of flow out of the left atrial appendage compared with velocities seen in normal sinus rhythm and is the anatomic/physiologic basis for stasis and formation of clot. Patients with atrial fibrillation and relatively intact atrial appendage transport function as documented by preserved emptying velocities (>50 cm/s) are less likely to have spontaneous contrast (and presumably thrombosis) than are those with reduced atrial appendage velocities. See Chapter 22 for further discussion of atrial fibrillation.
FIGURE 11.39. Transesophageal echocardiogram recorded in a patient with mitral stenosis and atrial fibrillation. The central figure was recorded at 96 degrees and reveals a dilated left atrial appendage with normal geometry (downward-pointing arrow). In the inset at the lower right is an image recorded at 130 degrees revealing a side lobe off the main body of the left atrial appendage (arrows) within which is a definite thrombus (long arrow). Video 11-39A
Video 11-39B

coming soon
FIGURE 11.40. Transesophageal echocardiogram recorded in a patient with rheumatic mitral stenosis who had undergone open mitral commissurotomy and “closure” of the left atrial appendage (LAA). Notice in this example the partial closure of the LAA with persistent limited flow into the cavity of the LAA (arrow). PV, pulmonary vein.
Secondary Pulmonary Hypertension

An additional sequela of long-standing severe mitral stenosis is secondary pulmonary hypertension (Fig. 11.41). In the early phases this is related to reactive pulmonary vascular tone and is reversible with correction of mitral stenosis. In long-standing severe mitral stenosis, a fixed component to pulmonary vascular resistance occurs and pulmonary artery systolic hypertension may be only partially reversible following correction of mitral stenosis.
FIGURE 11.41. Apical four-chamber view recorded in a patient with severe mitral stenosis and secondary pulmonary hypertension as well as moderate to severe tricuspid regurgitation. The central illustration reveals moderate to severe tricuspid regurgitation and was recorded prior to mitral valve replacement. The Doppler recording at the upper left corresponds to this image and reveals a tricuspid regurgitation velocity of 3.9 cm/s corresponding to a pressure gradient of 59 mm Hg representing pulmonary hypertension. The Doppler at the lower right was recorded 3 months following mitral valve replacement. Note the reduction of the tricuspid regurgitation velocity 22.3 cm/s corresponding to a pressure gradient of 21 mm Hg representing resolution of the secondary pulmonary hypertension following mitral valve replacement.
Echocardiographic manifestations of secondary pulmonary hypertension in mitral stenosis are similar to those seen in pulmonary hypertension of any cause. Concurrent tricuspid regurgitation is often present either due to right ventricular dilation or less often due to rheumatic involvement of the tricuspid valve.

**Decision Making Regarding Intervention**

Medical management plays only a minor role in alleviating symptoms in mitral stenosis. Definitive therapy is directed at increasing the effective mitral orifice area. This can be accomplished by open surgical commissurotomy, percutaneous balloon valvotomy, or mitral valve replacement. Once a
decision has been made that the severity of mitral stenosis warrants intervention, two-dimensional echocardiography plays a valuable role in determining the most appropriate interventional or surgical technique. As a general rule, valves with heavy degrees of calcification, chordal shortening and fibrosis, and prominent subvalvular involvement are not good candidates for either surgical or interventional correction. The echocardiographic images in Figures 11.12, 11.13, and 11.15 were recorded in patients with relatively mild fibrosis of the valve for which balloon intervention would be feasible. Compare these figures with Figures 11.14 and 11.16, in which there are varying degrees of diffuse fibrosis and calcification of the mitral valve apparatus.

FIGURE 11.43. Transthoracic parasternal short-axis view recorded at mid diastole in a patient with rheumatic mitral stenosis and asymmetrical fusion of the commissures. Note the small, nearly circular regurgitant orifice located at the medial aspect of the mitral valve (left arrows) and the complete fusion of the lateral commissures (right arrows).
A mitral valve score has been developed to stratify the degree to which the valve is anatomically compromised. The components of the score are leaflet thickening, leaflet mobility, calcification, and subvalvular involvement (Fig. 11.42). Each of these is then graded as 0 (absent) to 4 (severe) and the scores summed to create a mitral stenosis score. There is a direct relationship between the stenosis score and the likelihood of successful balloon valvotomy, with higher scores mitigating against successful intervention. Individuals with a mitral valve score of ≤8 typically are excellent candidates for balloon valvotomy, and those with scores ≥12 are less likely to have a satisfactory result. Asymmetric calcification and subvalvular involvement represent a disproportionate contribution to the likelihood of technical failure at the time of balloon valvotomy (Fig. 11.43).

MITRAL REGURGITATION

Mitral regurgitation can occur due to primary disease of the mitral leaflets or secondary to abnormalities of other components of the mitral apparatus. Etiologies of mitral regurgitation are outlined in Table 11.1. Acute severe mitral regurgitation often results in acute pulmonary congestion, whereas chronic mitral regurgitation may be tolerated for decades. For most patients with acute severe mitral regurgitation, surgical correction will be required. By definition, mitral regurgitation occurs during systole, which, at usual heart rates, constitutes approximately 30% to 50% of the cardiac cycle. As such,
marked left atrial pressure elevation is not present consistently but only transiently. The transient nature of the atrial pressure increase seen in mitral regurgitation represents less of a drive to development of secondary pulmonary hypertension than does the chronic (although lower intensity) pressure elevation seen in severe mitral stenosis. Mitral regurgitation also results in a volume overload of the left ventricle which may be well tolerated for relatively long periods of time. This eventually results in a reduction in left ventricular myocardial contractile force, which may not be reversible even with correction of the mitral regurgitation.

It is important to define the mechanism of mitral regurgitation as either being due to an anatomical disturbance of the leaflets, or as a function of abnormalities of the underlying supporting papillary muscles and ventricular wall (primary vs. secondary). Therapy for primary mitral regurgitation should be directed at the anatomical abnormality, whereas abnormalities leading to functional mitral regurgitation require addressing the underlying functional disturbance. Current guidelines suggest that equivalent degrees of functional and anatomical regurgitation of moderate or more severity have a worse impact on prognosis if regurgitation is functional rather than anatomical.

**FIGURE 11.44.** Transesophageal echocardiogram recorded in a patient with faint, early systolic blue color Doppler encoding within the left atrium (arrow). This color signal represents overall posterior motion of the pre-existing blood pool in the left atrium, combined with backwash of flow forced to motion by the closing mitral valve leaflets. Also note the lack of any convergence zone, vena contracta.
or high-velocity color coding. This signal should not be mistaken for true mitral regurgitation. The side panel was recorded one frame later and shows absence of flow.

Video 11-44

Doppler Evaluation of Mitral Regurgitation

The full range of echocardiographic techniques should be used for complete evaluation of mitral regurgitation (Table 11.5). Color Doppler imaging is the primary echocardiographic tool for initial detection and quantitation of mitral regurgitation. There are several potential sources of color Doppler flow signal in the left atrium, and not all color Doppler signals appearing within the left atrium represent mitral regurgitation. These include normal posterior motion of the blood pool caused by mitral valve closure (Fig. 11.44), reverberation from aortic flow (Fig. 11.45) and normal pulmonary vein inflow (Fig. 11.46). Confusion of normal pulmonary vein inflow may be most problematic if an inappropriately low Nyquist limit is used, or if a patient has a high flow state resulting in increased pulmonary vein inflow velocities. Atrial blood pool motion of overall low velocity visualized because of inappropriate gain and Nyquist limits may also mimic mitral regurgitation. On occasion, these signals have been erroneously attributed to mitral regurgitation and either a false diagnosis of regurgitation has been made or the extent of true regurgitation overstated.
FIGURE 11.45. Parasternal long-axis view recorded in a patient with color reverberation in the left atrium (*between the arrows*). This signal is a color artifact arising from the aorta and should not be confused for mitral regurgitation. Note that it is a direct extension of the turbulent flow in the proximal aorta and that it does not arise from any area of mitral valve closure. In the real-time image, note the very brief duration of this signal.
FIGURE 11.46. Apical four-chamber view recorded in a patient with turbulent flow within the left atrium. This flow represents relatively high volume and high-velocity pulmonary vein inflow. It should not be confused for mitral regurgitation. Note the complete lack of continuity of the left atrial color-flow signal with the mitral valve and the absence of the convergence zone or vena contracta. This phenomena may be exacerbated if utilizing inappropriately low Nyquist limits and in patients who are in a hyperdynamic state.
On occasion, diastolic mitral regurgitation may be noted. This is most commonly related to significant left ventricular systolic dysfunction and/or significant bradycardia. In the presence of elevated left ventricular diastolic pressure and long cardiac cycles, left ventricular diastolic pressure may equal or exceed left atrial pressure and there is backflow of blood from the left ventricle into the left atrium. Figure 11.47 was recorded in a patient with atrial flutter and high degree heart block who had intermittent diastolic mitral regurgitation. This type of regurgitation does not pose a hemodynamic burden to the left ventricle or left atrium and is simply a marker of the abnormal underlying pathophysiology.

**FIGURE 11.47.** Apical four-chamber view recorded in a patient with atrial flutter and high degree heart block. The central image was recorded after a prolonged pause and reveals low-velocity regurgitation into the left atrium. The timing of the event is better appreciated on the color Doppler M-mode at the lower right. The arrow depicts flow away from the transducer in diastole consistent with diastolic
mitral regurgitation.

Video 11-47

FIGURE 11.48. Schematic demonstrates the principal features of true mitral regurgitation. The various components of the true regurgitant signal are outlined on the schematic, including the proximal flow acceleration zone, the vena contracta, and a central high-velocity jet surrounded by lower-velocity recruited flow. The figure also schematizes a confirmatory spectral Doppler image recorded in both continuous-wave and pulsed-wave modes. Note the aliasing phenomenon with pulsed-wave Doppler imaging.

The characteristics of a true mitral regurgitation jet are: (1) evidence of proximal flow acceleration (proximal isovelocity surface area [PISA]); (2)
flow conforms to the appearance of a true “jet”; (3) the downstream (left atrial) appearance is consistent with a volume of blood being ejected through a relatively constraining orifice (vena contracta); (4) the flow signal is appropriately confined to systole; and (5) the color Doppler signals are appropriate in color for the anticipated direction and/or reveal the appropriate variance or turbulence encoding. Pulsed- and/or continuous-wave Doppler can be used to confirm the origin, timing, and direction of flow (Figs. 11.48 to 11.50).

As discussed in Chapter 8, the driving pressure of a regurgitation jet will have an impact on the registered volume of blood in motion within the downstream left atrium (Fig. 11.51). This is related to jet momentum. High-pressure jets recruit a greater volume of blood into motion within the left atrium and as such the jet area will overstate the volume of mitral regurgitation when compared to the same patient in a normotensive state. In view of this, it should be recognized that in the presence of aortic stenosis or hypertension, mitral regurgitant severity may be overestimated by jet area. Conversely mitral regurgitant severity will be underestimated in a hypotensive patient. Calculation of effective regurgitant orifice (ERO) and other quantitative measures of mitral regurgitation severity are relatively independent of jet velocity and systolic pressure.
FIGURE 11.49. Transesophageal echocardiogram in a patient with moderate mitral regurgitation demonstrates the components of true mitral regurgitation as schematized in Figure 11.48. Note the flow acceleration convergence zone (CZ), the relatively narrow vena contracta (VC), and a high-velocity turbulent downstream jet.
FIGURE 11.50. Spectral Doppler image recorded in a patient with mitral
regurgitation using continuous-wave Doppler (A) and pulsed Doppler (B) imaging. Note the aliasing phenomenon with pulsed Doppler imaging in which the signal directed away from the transducer is paradoxically recorded above the zero crossing line after exceeding the Nyquist limit (1.0 m/s in this example). In the continuous-wave signal, note the ability to record the full maximal velocity of the mitral regurgitation jet (6 m/s).

By definition, hemodynamically significant mitral regurgitation results in left-sided volume overload with subsequent left ventricular and left atrial dilation. As a consequence, there is elevation of left atrial pressure, which is transmitted to the pulmonary venous vasculature resulting in pulmonary congestion. The physiology of acute severe mitral regurgitation is substantially different from chronic mitral regurgitation. In the acute setting, there is insufficient time for chamber dilation to occur and for left atrial compliance to increase. As such, acute severe mitral regurgitation is associated with immediate elevation of left atrial pressure, which results in the instantaneous onset of symptoms. With chronic mitral regurgitation, the left atrium dilates and atrial compliance increases. Because of this, left atrial pressure is lower in the chronic than in the acute setting for any given degree of mitral regurgitation and development of symptoms may be delayed for years.

The jet of mitral regurgitation may be central, peripheral, single, or multiple and may be eccentric within the left atrium and impinge on a wall depending on mitral pathology. Figure 11.11 schematizes the mitral closure pattern and jet direction in a number of disease states. Figures 11.52 through 11.55 were recorded in patients with mitral regurgitation of varying severity and regurgitation jet morphology.
FIGURE 11.51. Illustration of the impact of systolic blood pressure on mitral regurgitation jet size. The images are recorded in the same patient during a single transesophageal echocardiographic study at different levels of systolic blood pressure. A was recorded at a systolic blood pressure of 122 mm Hg. Note the downstream mitral regurgitation jet consistent with mild mitral regurgitation. Also note the relatively narrow vena contracta and small convergence zone. At the lower right note the calculations derived from the Doppler information suggesting...
a mitral regurgitation volume of 23 mL and a mitral regurgitant orifice of 0.13 cm². 

B was recorded in the same patient during the same echocardiographic study but at a systolic blood pressure of 153 mm Hg. Note the substantially larger downstream mitral regurgitation jet suggestive of at least moderate mitral regurgitation but the relatively stable size of the vena contracta and proximal convergence zone. At the lower right note the Doppler calculations of effective regurgitant orifice and mitral regurgitation volume which are quite similar to those noted at the time of a systolic blood pressure of 122 mm Hg when the downstream jet suggested only mild mitral regurgitation.

Video 11-51

FIGURE 11.52. Transthoracic echocardiogram recorded in a patient with mild to moderate mitral regurgitation. This illustration demonstrates some of the difficulty with assessing mitral regurgitation severity based on color Doppler jet area. A is a mid systolic parasternal long-axis view in which a limited color Doppler mitral regurgitation jet is noted within the left atrium. Note however the dilation of the left atrium which suggests a more severe and long-standing hemodynamic overload. B is an apical four-chamber view recorded at mid systole. The color Doppler jet area was planimetered to be 4 cm². Left atrial area was 20 cm² and the ratio of mitral regurgitation to left atrium is 20%, slightly above the threshold for mild mitral regurgitation. Note in the apical four-chamber view the very small proximal
convergence zone and narrow vena contracta suggesting mild mitral regurgitation.

Video 11-52

FIGURE 11.53. Apical four-chamber view recorded in a patient with a nonischemic dilated cardiomyopathy and severe functional mitral regurgitation related to apical tethering of the mitral supporting apparatus. The central image was recorded in mid systole and reveals a large color Doppler jet filling two-thirds of the left atrial cavity. The image at the upper left is a short-axis image at the
level of the distal mitral valve, recorded at mid systole at the time equivalent to the main central illustration. Note the failure of coaptation of the mitral leaflets from which a regurgitant area of 0.687 cm² can be directly measured.

**Video 11-53**

**Determination of Mitral Regurgitation Severity**

Assessment of mitral regurgitation severity requires integration of multiple echocardiographic and Doppler parameters. Tables 11.5 and 11.6 outline various findings seen in mitral regurgitation and their relationship to determining severity. It should be emphasized that no single parameter is completely accurate for determining severity. Assessment of mitral regurgitation severity should be based on a combination of findings. Many observations are valid only at the extremes, that is, in accurately identifying mild and severe regurgitation but have substantial overlap in the moderate range. In many instances, one or more of these findings may not correlate with other findings. In this instance, severity should be based on the overall findings and not one isolated feature.

The initial detection and determination of the severity of mitral regurgitation relies heavily on color flow Doppler imaging. While providing a widely available and rapid screening method, there are numerous limitations to using this methodology for assessing regurgitation, which were discussed in Chapters 2 and 8. Initial validation studies were done in relatively small cohorts with left ventriculography as the standard. All these suggested a correlation between the angiographic grade of regurgitation and the color flow area within the left atrium (Fig. 11.56). When assessing
regurgitation jet size, it is imperative to adjust Doppler gains appropriately to avoid “blooming,” which will increase the apparent jet size. In addition, an inappropriately low Nyquist limit will result in low-velocity pulmonary vein flow and recruited flow being encoded as turbulence and systematically overstate the severity of regurgitation (Fig. 11.57). As noted previously and in Chapter 8, mitral regurgitation occurring at pathologically elevated systolic blood pressure, typically will be overestimated by jet area. In addition, jet size may vary over the systolic cycle. While the eye rapidly integrates this change in size over time to estimate overall jet size, any given still frame may either dramatically over- or underestimate the jet size and hence mischaracterize severity.

FIGURE 11.54. Parasternal short-axis view recorded in a patient with functional mitral regurgitation related to a nonischemic cardiomyopathy. The central image is recorded at end-systole. Note that the failure of coaptation has resulted in a visible systolic regurgitant orifice (arrows) which can be planimeted to 0.8 cm². The image at the upper left is recorded with color Doppler in the same patient revealing the systolic regurgitant flow.
FIGURE 11.55. Off-axis apical imaging of a patient with mitral valve prolapse and mitral regurgitation. In the central image note the relatively hemispherical convergence zone (small downward-pointing arrows). Also note the absence of a typical downstream jet in the body of the left atrium. In this instance mitral regurgitation takes on a highly disorganized path in the left atrium with several regurgitant jets medially and laterally (arrows). Disorganized jets such as seen here are not suitable for quantitation. The inset at the lower left is an expanded view of the mitral valve anatomy revealing myxomatous changes of the prolapsing
The assessment of mitral regurgitation severity can be enhanced by
indexing the regurgitation jet area to left atrial size. Several relatively small studies have confirmed the correlation between this type of assessment of mitral regurgitation severity and a standard such as contrast ventriculography. In addition, the width of the regurgitant jet at its origin (the vena contracta) can be measured from the color Doppler image and correlates with regurgitation severity (Fig. 11.48).

Most schemes for determining the severity of mitral regurgitation were developed in the presence of central jets in which the regurgitant jet recruits into motion left atrial blood adjacent to all its surfaces. As such, the overall Doppler encoded size of the “jet” in the left atrium overstates the true volume of flow from the left ventricle by the amount of pre-existing left atrial blood recruited into motion. If a similar regurgitant volume arises from an eccentric jet, which impinges on a wall, then recruitment of left atrial blood occurs only over the portion of the jet surface area that is not constrained by a wall. This results in a smaller amount of recruitment for an impinging jet than for a central jet and underestimation of regurgitation severity when compared with an identical regurgitant volume due to a central jet. This phenomenon is schematized in Figure 11.58. In general, color flow Doppler imaging of a highly eccentric jet impinging on the left atrial wall will understate the regurgitation volume by as much as 40% when compared with an identical regurgitant volume that is centrally located.

Table 11.6  MITRAL REGURGITATION SEVERITY

<table>
<thead>
<tr>
<th></th>
<th>I (Mild)</th>
<th>II</th>
<th>III</th>
<th>IV (Severe)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular size</td>
<td>N</td>
<td>N</td>
<td>↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>Left atrial size</td>
<td>N</td>
<td>N</td>
<td>↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>MR jet (% LA)</td>
<td>&lt;15</td>
<td>15–30</td>
<td>35–50</td>
<td>&gt;50</td>
</tr>
<tr>
<td>Spectral Doppler density</td>
<td>Faint</td>
<td>—</td>
<td>—</td>
<td>Dense</td>
</tr>
<tr>
<td>Vena contracta</td>
<td>&lt;3 mm</td>
<td>—</td>
<td>—</td>
<td>&gt;7 mm</td>
</tr>
<tr>
<td>Pulmonary vein flow</td>
<td>S &gt; D</td>
<td>—</td>
<td>—</td>
<td>Systolic reversal</td>
</tr>
<tr>
<td>RV (mL)</td>
<td>&lt;30</td>
<td>30–44</td>
<td>45–59</td>
<td>&gt;60</td>
</tr>
<tr>
<td>ERO (cm²)</td>
<td>&lt;0.2</td>
<td>0.2–0.29</td>
<td>0.3–0.39</td>
<td>&gt;0.40</td>
</tr>
<tr>
<td>PISA</td>
<td>Small</td>
<td>—</td>
<td>—</td>
<td>Large</td>
</tr>
</tbody>
</table>
For some parameters, the observation is valid at the extremes of mitral regurgitation severity and there may be marked overlap in moderate (grades 2 and 3) mitral regurgitation.

ERO, effective regurgitant orifice; MR, mitral regurgitation; N, normal; % LA, percentage of left atrial area encompassed by the mitral regurgitation jet with color flow Doppler imaging; PISA, proximal isovelocity surface area; RV, regurgitant volume; S>D, antegrade pulmonary vein flow in systole exceeds diastolic flow; ↑, increased; ↑↑, markedly increased.

FIGURE 11.56. Schematic representation demonstrates the methodology by which jet area is used to determine jet severity. A series of centrally located, not eccentric, jets are demonstrated, which encompass approximately 15%, 25%, 35%, and 60% of the left atrial area, representing grades 1 through 4 (mild to severe) mitral regurgitation.

A final qualitative parameter that relates to the severity of regurgitation is the density of the spectral Doppler signal. Spectral density is directly proportional to the number of red blood cells being interrogated by the
Doppler beam. If only a very low volume of blood is in motion, the spectral signal will be relatively faint, whereas with severe regurgitation, a greater volume is in motion and the spectral signal is substantially more robust (Fig. 11.59). The shape of the spectral signal also confers diagnostic information. While the continuous-wave spectral Doppler signal in chronic mitral regurgitation is symmetric, acute severe regurgitation results in a rapid equilibration of left atrial and ventricular pressure. In this setting, the spectral profile will be more triangular in shape (Fig. 11.60).

In addition to qualitative assessment of mitral regurgitation severity by color flow Doppler imaging, there are quantitative measurements that can be made for the determination of mitral regurgitation severity. These include determination of volumetric flow by the PISA method and determination of regurgitant volume and regurgitant fraction based on calculation of ventricular volumes and forward stroke volume. Using the volumetric analyses described in Chapter 8, one can determine the diastolic and systolic volumes of the left ventricle from which total stroke volume can then be calculated. Using principles elucidated in Chapter 8 to determine volumetric flow in the left ventricular outflow tract, one can then calculate the forward stroke volume. The difference between the total left ventricular stroke volume and forward stroke volume in the left ventricular outflow tract then equals the mitral valve regurgitant volume (Fig. 11.61). This calculation assumes the absence of aortic regurgitation. A major limitation of this technique is the number of different measurements that must be made, each of which introduces a quantitative error. Alternatively, one can use the mitral valve inflow time velocity integral and an assumed or measured mitral orifice area to determine forward-going mitral flow in diastole. This volume of flow is then equal to the regurgitant mitral volume plus the forward stroke volume and can provide a different route for determination of mitral regurgitation volume. In general, this later methodology has seen little acceptance because of the difficulty in determining the true mitral orifice or annular area.
FIGURE 11.57. Impact of Nyquist limit on apparent regurgitation jet size. All four images were recorded in the same patient with moderate mitral regurgitation. C, D: Recorded at Nyquist limits of 0.69 and 0.88 m/s, resulting in a smaller apparent jet than A, B, recorded at inappropriately low Nyquist limits of 0.3 and 0.4 m/s. Note that at the low Nyquist limit of 0.3 m/s, even normal pulmonary vein inflow has been encoded as turbulent flow, thus resulting in a substantially larger area of turbulence in the left atrium and overstating the severity of mitral regurgitation.
FIGURE 11.58. Schematic representation of the effect of an impinging wall jet versus a centrally located wall jet on overall jet size detected with color Doppler flow imaging. **Left:** A centrally located “free” jet within the body of the left atrium, which is not constrained by a solid boundary such as the atrial wall. The darker central jet represents the actual volume of regurgitant blood, which originated in the left ventricle and has been ejected into the left atrium. The fainter outer signal represents the blood that has been recruited into motion and is also detected by color flow Doppler imaging. Note that the total amount of blood in motion exceeds the actual regurgitant volume by approximately 40%. **Right:** The effect on jet size for a jet directed along a constraining surface, such as the left atrial wall. The dark area represents the blood ejected from the left ventricle into the left atrium and is the same area as the dark regurgitant jet on the left. Note that the blood is recruited into motion only along one surface of the jet, hence making the total area of the jet (regurgitant blood plus recruited blood) smaller than the free jet, even though the total true regurgitant volume is identical.

Finally, by inspection of the isovelocity curves in the proximal convergence zone (Figs. 11.62 through 11.65), the regurgitant volume and several derived indices can be calculated. The general use of PISA method for determining volumetric flow is discussed in Chapter 8. With this technique, one maximizes the dimension of the proximal velocity hemisphere
by using a relatively low Nyquist limit and by shifting the baseline toward the
direction of regurgitant flow (RF). Maximizing the distance over which the
measurement is made reduces error. One can then determine the velocity of
flow in the hemispherical flow zone at its aliasing line as well as the radius of
any given hemisphere of flow. If one assumes a hemispherical flow profile
toward the regurgitant orifice, the surface area of this hemisphere of flow can
be calculated by the formula: surface area = $2\pi r^2$. The product of the
hemisphere area and the aliasing velocity of the color flow display equals the
flow rate. Once volumetric RF has been determined, the ERO can be
calculated. This is calculated as RF divided by the peak velocity of the mitral
regurgitation jet ($MR_{max}$), obtained from the continuous-wave spectral profile
(ERO = RF/$MR_{max}$). The ERO area thus determined is related to the
regurgitant volume by the formula: $RV = ERO \times TVI_{MR}$, where $TVI_{MR}$ is the
time velocity integral of the mitral regurgitation jet.
FIGURE 11.59. Continuous-wave Doppler spectral recordings from patients with mild (A), moderate (B), and severe (C) mitral regurgitation. Note the progressive increase in signal density with increasing severity of mitral regurgitation due to
There are several limitations to the PISA method, the most important of which is that flow convergence may not conform to a true hemispherical shape. If convergence toward the regurgitant orifice occurs over a surface greater than 180 degrees, the flow volume will be overstated by the PISA method with a directionally opposite error occurring if the flow converges in a narrower angle. An additional source of error with the PISA method is a mitral regurgitation jet through a noncircular orifice or through multiple regurgitant orifices. In the first case, the surface area of the regurgitation flow volume does not conform to a hemisphere but rather to a half-cylinder. Inspection of the PISA shape in an orthogonal plane should help avoid this source of underestimation (Fig. 11.66). In many instances, the geometry of the proximal regurgitant jet may preclude accurate use of the PISA method, and severity assessment should be based on other factors. Finally if multiple regurgitation jets are present the total volume will not accurately be reflected by analysis of only one jet.

**FIGURE 11.60.** Continuous-wave Doppler recorded in a patient with severe mitral regurgitation. This image was recorded from the apex of the left ventricle and demonstrates a triangular mitral regurgitation jet with a maximum velocity of 4.6 m/s. Both the relatively low regurgitant velocity and triangular shape are consistent with severe mitral regurgitation with early equalization of left atrial and left ventricular pressures. The inset at the lower left is the color Doppler image demonstrating severe mitral regurgitation based on jet area.
FIGURE 11.61. Method for determining mitral regurgitation volume using left ventricular volume. An apical four-chamber view was recorded in diastole and in systole from which diastolic and systolic volumes are determined (left panels). The difference between the diastolic and systolic volumes is the total left ventricular stroke volume (LV$_{SV}$), which represents the sum of forward flow in the left ventricular outflow tract and the regurgitant volume. Alternatively, this view can also be used to determine the diastolic transmitral flow by determining the diameter of the annulus from which the annular area can be calculated. The product of annular area and the time velocity integral of mitral flow equals forward flow from the left atrium into the left ventricle in diastole, which in turn equals the sum of regurgitant flow and forward flow. This total LV$_{SV}$ is used to calculate regurgitant volume as the difference in total LV stroke volume and forward stroke volume from the LVOT. Parasternal long-axis view is recorded from which the diameter of the left ventricular outflow tract is measured and then the outflow tract area (LVOT$_{a}$) determined as demonstrated (upper right). The time velocity integral (TVI) in the left ventricular outflow tract is recorded from an apical view (lower right) and the product of LVOT$_{a}$ times TVI equals forward stroke volume (F). This forward stroke volume can then be subtracted from the total transmitral volume or from the total left ventricular stroke volume (LV$_{SV}$) to determine the regurgitant volume. LVV$_{d}$, left ventricular volume in diastole; LVV$_{s}$, left ventricular volume in systole.
FIGURE 11.62. Schematic demonstration of the principle involved in calculating mitral regurgitation severity from the proximal isovelocity surface area method. In this schematic, mitral regurgitation has been visualized from the apical four-chamber view. The color Doppler scale has been shifted downward so that the mitral regurgitation aliasing velocity has been reduced to 40 cm/s, thus maximizing resolution for measuring the aliasing radius. The area of hemispherical flow through the regurgitant orifice can be calculated as $2\pi r^2$. Instantaneous flow can be calculated as area $\times$ flow velocity at the aliasing boundary ($V_A$). The effective regurgitant orifice (ERO) is calculated as $\text{flow}/V_{\text{MAX}}$. The regurgitant volume (RV) can be calculated as the product of $\text{ERO} \times \text{TVI}$ (where TVI is the time velocity integral of the mitral regurgitation flow as measured by continuous-wave Doppler imaging).
FIGURE 11.63. Apical four-chamber view recorded in a patient with mild mitral regurgitation. In the central image, note the mild degree of mitral regurgitation based on the magnitude of color flow within the left atrium. In this instance, the PISA calculation has been utilized to calculate the mitral regurgitant orifice (ERO). The upper left panel is the continuous-wave Doppler profile recorded at the time of calculating quantitative measures of mitral regurgitation. 

Video 11-63
**FIGURE 11.64.** Apical four-chamber view recorded in a patient with moderate mitral regurgitation, demonstrating the method by which the proximal convergence zone can be utilized to quantify mitral regurgitation volume and effective regurgitant orifice (ERO). In this instance, the color flow Doppler suggests moderate to severe mitral regurgitation, and the quantitative calculations reveal a regurgitant volume of 40 mL and an ERO of 0.3 cm$^2$ consistent with moderate mitral regurgitation. [Video 11-64](coming-soon)
Three-dimensional color flow imaging has been used investigationaly to quantify the convergence zone in three dimensions. The calculation of ERO and regurgitant volume generally assumes a circular regurgitant orifice on a planar surface. Clearly, the regurgitant orifice may be slit-like or elliptical and convergence may be directed not toward a planar surface but toward a complex surface and as such the convergence zone could encompass either an angle greater or less than the assumed 150 degrees. Three-dimensional color flow imaging potentially provides an accurate representation of the true three-dimensional geometry of the convergence zone but at this time has not seen widespread clinical applicability.

FIGURE 11.65. Echocardiogram recorded in a patient with severe mitral regurgitation. The central image is an expanded view of the proximal convergence zone. Note that the mitral regurgitation radius is measured at 1.23 cm from which a mitral regurgitation regurgitant orifice of 0.656 cm² can be calculated. At the lower right note the expanded view of the vena contracta which measures 1.36 cm, consistent with severe mitral regurgitation.
FIGURE 11.66. “X-plane imaging” recorded in a patient with mitral regurgitation. These images were recorded from a transesophageal echocardiogram. The panel on the right is recorded at a 3-degree rotation. Note the relatively small and presumably hemispherical proximal convergence zone (arrows). The panel on the left is recorded at 85-degree rotation where the convergence zone can be seen to be elongated and ellipsoid (arrows), rather than hemispherical. Reliance on the hemispherical convergence zone on the right would clearly lead to underestimation of volumetric flow.
Using three-dimensional echocardiography with or without three-dimensional color flow Doppler, it is often possible to directly visualize and quantify the mitral regurgitation orifice. This is typically best accomplished with a transesophageal echocardiogram utilizing the “surgeon’s view.” As previously discussed for evaluation of flail valves, the actual regurgitant orifice can be identified whether it is related to a flail leaflet, perforation, or functional mitral regurgitation. It should be emphasized that, with a flail leaflet, the regurgitant orifice will be off axis with respect to a view from directly behind the left atrium, and navigating the three-dimensional imaging plane would be necessary to view it en face. It is often most advantageous to utilize three-dimensional color flow imaging to specifically identify the actual orifice in three dimensions, after which the orifice can be planimetered either as the actual color regurgitant area or, after suppression of the color signal, its anatomical boundaries (Figs. 11.67 and 11.68).

**Other Considerations in Assessing Mitral Regurgitation**

Virtually all schemes for quantifying mitral regurgitation have assumed holosystolic mitral regurgitation. In many instances, such as mitral valve prolapse, regurgitation may be confined to only a portion of systole. As such, the volume of flow either estimated from a color flow image area or calculated by the PISA technique should be corrected for the fraction of systole over which flow occurs. One occasionally encounters the situation of apparent chronic moderate or severe mitral regurgitation on color flow
Doppler imaging, but which is not associated with secondary left atrial dilation. Careful attention to the timing of mitral regurgitation in these instances often reveals that the mitral regurgitation jet, although encompassing a substantial area of the left atrium, is present only for 30% to 50% of systole. Figure 11.69 illustrates an example in which the color jet area fills approximately 40% of the left atrium but has not resulted in chamber dilation in spite of the known chronicity of the mitral regurgitation. Note in the insert in the upper left that the jet is present only in the latter half of systole. Figure 11.70 is a color M-mode image of a similar mitral regurgitation jet demonstrating that it is confined to the later 40% of systole, and hence the jet area method would overstate the actual severity of regurgitation. A valuable clue to this phenomenon is a discrepancy between the color flow area and left atrial size. It is unlikely to have chronic moderate or greater mitral regurgitation in the absence of left atrial dilation.

FIGURE 11.67. Full volume three-dimensional transesophageal echocardiogram recorded in a patient with severe mitral regurgitation. The tissue structures are coded in gray and the three-dimensional full volume color flow Doppler has been used to visualize the mitral regurgitation jet. This image has been rotated to visualize the actual regurgitant orifice en face, which is denoted by the series of
dots outlining the color-flow signal in the central part of the mitral valve. The panel at the lower left is a depiction of the mitral regurgitation jet in three dimensions. The tissue signature has been subtracted leaving only the color Doppler image. The longer horizontal echoes denote the plane of the regurgitant orifice allowing demonstration of the actual *vena contracta*. The *smaller arrows* outline the boundary of the proximal convergence zone.  

**Video 11-67**

An additional finding noted in severe mitral regurgitation is retrograde flow in the pulmonary veins during systole. This can be directly attributed to the increasing left atrial pressure and regurgitant volume in the left atrium. This finding is a marker of moderate to severe mitral regurgitation and is not seen in mild regurgitation. It occasionally can be absent in the presence of a highly eccentric jet, which is directed away from the pulmonary veins. Although its presence is a reliable marker of moderate and severe mitral regurgitation, its absence should not be used to exclude significant mitral regurgitation in the presence of echocardiographic and Doppler features suggesting severe regurgitation. **Figure 11.71** presents an example of mitral regurgitation associated with retrograde flow in a pulmonary vein.
FIGURE 11.68. Transesophageal echocardiogram with real-time three-dimensional imaging in a patient with severe functional mitral regurgitation. The central illustration is from the left atrial perspective and is oriented to show an en face view of the regurgitant orifice. The area is as outlined by the dotted lines and calculates to 1.18 cm². The side panel at the lower left is a standard two-dimensional image showing severe mitral regurgitation related to apical tethering of the mitral leaflets.

Video 11-68
FIGURE 11.69. Apical four-chamber view recorded in a patient with mitral regurgitation. This image recorded at late systole suggests the presence of at least moderate mitral regurgitation based on the size of the color flow jet which encompasses 39% of the left atrial area. On the upper left, note the continuous-wave spectral profile of the mitral regurgitation jet, confirming that it is confined to the latter 50% of ventricular systole, thereby resulting in an overestimation of regurgitant volume for any given jet area. Video 11-69
An additional limitation of echocardiographic and Doppler evaluation of mitral regurgitation is the frequent dynamic nature of mitral regurgitation. The severity of the regurgitation itself as well as its appearance on Doppler echocardiography can be highly dynamic and vary with both ventricular pre- and afterload. This may be particularly relevant in functional rather than organic mitral regurgitation and the apparent mitral regurgitation severity needs to be considered in light of the underlying hemodynamic status of the patient.
FIGURE 11.70. Color Doppler M-mode images recorded in patients with mitral regurgitation. Both tracings were recorded from the left ventricular apex. **A:** Recorded in a patient with mitral valve prolapse and regurgitation confined to the latter 40% of systole. The two vertical lines indicate the duration of mechanical systole (double-headed arrow). **B:** Recorded in a patient with holosystolic mitral regurgitation.

FIGURE 11.71. Doppler recording of the pulmonary vein flow from the apex of the left ventricle in a patient with severe mitral regurgitation. The central image is the pulsed-wave Doppler with a sample volume placed in the pulmonary vein. Note the prominent retrograde flow in systole (arrows). The inset at the upper left is an apical four-chamber view with color Doppler demonstrating a large color Doppler regurgitant jet area in the left atrium.
Because of the variability and limitations in determining the severity of mitral regurgitation, revised recommendations have been published (Fig. 11.72). The current recommendation is that multiple parameters be considered, combinations of which provide a highly specific diagnosis of either mild or severe mitral regurgitation. In the absence of a highly specific diagnosis of regurgitation at these two extremes, additional quantitative methods are required for further characterization of mitral regurgitation severity. In many instances, even after a detailed Doppler evaluation with quantitative techniques, the severity of mitral regurgitation may be indeterminate and consideration of further testing with cardiac magnetic resonance imaging or hemodynamic evaluation may be indicated.

**Flail Leaflets**

Any portion of the mitral apparatus can become anatomically disrupted and result in a portion of the mitral valve becoming flail. The underlying substrate is commonly myxomatous degeneration. The degree of resultant regurgitation is directly related to the extent of anatomic disruption. Rupture of only a few isolated chordae may not result in disruption of normal coaptation and hence can be seen in the absence of significant mitral regurgitation. Rupture of an entire papillary muscle or papillary muscle head typically results in acute severe mitral regurgitation. Between these two extremes, a wide range of anatomic disruption with varying degrees of mitral regurgitation can be noted. Anatomic disruption of a portion of the mitral apparatus usually results
in an eccentric direction of the regurgitation jet with an orientation opposite the direction of the flail leaflet (Figs. 11.73 to 11.75).

![Flow chart for evaluation of severity of chronic mitral regurgitation](image)

**FIGURE 11.72.** Flow chart for evaluation of severity of chronic mitral regurgitation by Doppler echocardiography as recommended by the new valve disease guidelines. This scheme relies on specific identification of patients as having either mild or severe mitral regurgitation by classic criteria. Patients who fall between these two extremes require further evaluation for assessment of severity with more quantitative techniques. (Redrawn with permission from O’Gara PT, Grayburn PA, Badhwar V, et al. 2017 ACC Expert Consensus Decision Pathway on the Management of Mitral Regurgitation. *J Am Coll Cardiol* 2017;70(19):2421–2449. Copyright © 2017 Elsevier.)

The recognition and complete description of flail mitral valve leaflet plays a critical role in patient management. Transesophageal and three-dimensional echocardiography play a critical role in the full evaluation of flail leaflets. The superior visualization afforded by transesophageal echocardiography allows precise localization and confirmation of a flail leaflet and is helpful for presurgical planning. Modern three-dimensional techniques provide incremental information regarding the precise location of flail leaflets. The preferred view is the “surgeon’s view” from which all six scallops of the mitral valve can simultaneously be visualized (Figs. 11.76 to 11.80). Three-
dimensional modeling of the mitral valve annulus and mitral leaflets can also be obtained and the location of prolapsing or flail tissue quantified (Fig. 11.81).
FIGURE 11.73. Schematic representation of jet direction in the presence of a flail anterior (top) or posterior (bottom) leaflet. In each instance, note that the tip of the flail leaflet is located behind the belly of the intact leaflet. This results in eccentric orientation of the regurgitant orifice, with the direction of the regurgitant jet opposite that of the flail leaflet. Note that the flail posterior leaflet results in a jet directed along the left atrial wall and the posterior wall of the aorta. This may result in a mitral regurgitation jet, which on auscultation is heard in the typical aortic area. The more laterally directed jet, attributable to the anterior flail leaflet, will result in a jet directed toward the lateral wall of the left atrium and a murmur heard best laterally rather than anteriorly.

FIGURE 11.74. Transthoracic parasternal long-axis view recorded in a patient with a flail anterior mitral valve leaflet. The central image is recorded in systole with color Doppler. Note the highly eccentric, posteriorly directed mitral regurgitation jet. The inset at the upper right is an expanded view of mitral valve coaptation. Note the failure of coaptation of the anterior leaflet (arrow) in systole which clearly is behind the posterior mitral valve leaflet.
FIGURE 11.75. Apical four-chamber recordings in a patient with a partially flail posterior mitral leaflet. A: An expanded systolic frame in which a small portion of the mitral valve and chordae can be seen within the body of the left atrium (arrow). B: A color Doppler image revealing a highly eccentric mitral regurgitation
jet which courses underneath the anterior leaflet of the mitral valve and subsequently along the atrial septum (arrows). While the anatomical imaging reveals relatively subtle evidence of a flail leaflet, the highly eccentric course of the mitral regurgitation jet is relatively specific for flail pathology.

**Video 11-75**

**FIGURE 11.76.** Transesophageal echocardiogram recorded in a patient with myxomatous mitral valve degenerative and a flail P2 leaflet (*downward-pointing arrows*). In A, note the diffuse myxomatous thickening of both mitral leaflets and the distinct prolapse of the posterior leaflet into the left atrium. There was discontinuity between the tip of the posterior leaflet and the chordal apparatus (*upward-pointing arrow*). In the panel at the upper left is the three-dimensional echocardiogram recorded from the left atrial perspective in this patent. Note the diffuse irregular myxomatous thickening of both mitral leaflets and the ruptured chord protruding into the left atrium in systole (*downward-pointing arrow*). (The *upward-pointing arrows* denote the actual regurgitant orifice.) B is the color flow image recorded in this patient revealing a highly eccentric mitral regurgitation jet. 

coming soon
FIGURE 11.77. Three-dimensional reconstruction of the mitral valve recorded in the same patient presented in Figure 11.76. In the central image, note the marked bulging of P2 into the left atrium (arrows). At the lower left is a schematic reconstruction of the mitral valve annulus, as well as the leaflets. Note the marked protrusion of P2 into the left atrium colored in red compared to the normal location of other mitral valve tissue in blue.
In the presence of a flail leaflet, the mitral regurgitation spectral Doppler signal may have an atypical appearance. The interrogation beam may intersect the jet either tangentially or only partially for part of the cycle. This may result in varying density and velocity of the signal, mimicking a less than holosystolic jet. In addition, if there are flail portions of the mitral apparatus that oscillate in the regurgitation flow stream, they result in a “tiger stripe” appearance of the spectral signal associated with a “whistling” sound on the audible signal (Fig. 11.82).

**Functional Mitral Regurgitation**

Functional mitral regurgitation is a common sequela of diseases which result in either segmental or global left ventricular systolic dysfunction, especially if associated with left ventricular remodeling. Remodeling typically results in apical and lateral displacement of the papillary muscles and the underlying wall, thus tethering the mitral apparatus toward the apex. This type of mitral regurgitation is commonly seen in patients with otherwise normal mitral leaflets. Effective therapy to reduce left ventricular size and promote reverse remodeling will result in reduction in mitral regurgitation.
FIGURE 11.78. Transthoracic echocardiogram recorded at 147 degrees in a patient with flail anterior mitral leaflet. In A, note the diffuse myxomatous thickening of the mitral valve leaflets, as well as the flail component protruding into the left atrium in systole (arrows). B is the color flow image recorded in this patient at mid systole. Notice the irregular convergence zone and the highly eccentric jet of mitral regurgitation impinging on the left atrial wall. Video 11-78
FIGURE 11.79. Three-dimensional echocardiogram with reconstruction of the mitral annular plane and leaflet tissue recorded in the same patient depicted in Figure 11.78. In the central image, notice the diffuse thickening of the mitral leaflet and the protrusion of a large portion of the anterior leaflet into the left atrium (X). In the lower right, note the computer-generated mitral annulus demonstrating substantial prolapse of a large portion of the anterior leaflet into the left atrium.

Video 11-79
Apical tethering results in leaflet coaptation not over the length of the zona coaptation but rather in a “tip-to-tip” fashion which inherently results in regurgitation. Figures 11.83 through 11.85 were recorded in a patient with dilated cardiomyopathy and severe functional mitral regurgitation. Note in the schematics the normal closing pattern and the abnormal closing pattern resulting in systolic “doming” with failure of coaptation, resulting in significant mitral regurgitation. In a dilated cardiomyopathy with equal involvement of both papillary muscles, the regurgitant jet is frequently central. A parameter that is related to the severity of functional mitral regurgitation is the mitral “tenting area.” Tenting area is quantified as the area subtended by the plane of the mitral annulus and the belly of the mitral leaflets in systole (Fig. 11.86). With normal coaptation, tenting area is minimal or negative. With progressive degrees of apical displacement of the mitral leaflets tenting area increases and is directly related to the severity of subsequent regurgitation.

In patients with ischemic heart disease, regional wall motion abnormalities may predominate and only one papillary muscle may be apically or laterally displaced. This results in restricted systolic motion of an otherwise normal mitral leaflet, so that when fully “closed” it is located more apically than the noninvolved leaflet. Figures 11.87 through 11.89 were recorded in a patient with restricted motion of the posterior mitral leaflet related to posterior myocardial infarction. As mentioned previously, in the section titled “Flail Leaflets,” jet direction can be indicative of the specific pathology involved. When dealing with a flail leaflet, the direction of the jet is opposite to that of the flail leaflet. When asymmetric restricted motion is the etiology of the mitral regurgitation, the eccentric jet will be in the direction of the restricted and not the normal leaflet as noted in Figures 11.87 and 11.88. Three-dimensional echocardiography can provide valuable insight as to the mechanism of functional mitral regurgitation as noted in Figures 11.89 to 11.91. Figure 11.91 was recorded in a patient with ischemic heart disease and apical tethering of both mitral leaflets. Note in this real-time three-dimensional transesophageal echocardiogram recorded from a left atrial view, the “double-barrel” regurgitant orifice in systole, which matches the two mitral regurgitation jets on both two- and three-dimensional color Doppler imaging.
FIGURE 11.80. Transesophageal echocardiographic imaging performed in a patient with a flail mitral valve involving multiple scallops. A: Real-time three-dimensional imaging centered on the plane of the mitral valve. The image at the right is the reconstructed three-dimensional image. Note the flail components of the mitral valve (arrows). The regurgitant orifice is noted by the “X.” B: Two-dimensional imaging recorded in the same patient noted in (A). In the central image note the flail portion of the posterior leaflet (arrow) within the body of the left atrium in systole. The inset at the upper left was recorded with color Doppler and reveals severe mitral regurgitation.
A final form of functional mitral regurgitation is that seen in obstructive hypertrophic cardiomyopathy where the anterior mitral leaflet is pulled toward the ventricular septum in systole separating it from its normal coaptation point with the posterior mitral leaflet. This is a form of dynamic functional mitral regurgitation, typically resulting in a posteriorly directed eccentric jet. This issue was further discussed in Chapter 18.
Initially mitral regurgitation represents a pure volume overload on the left ventricle. Initially, in the presence of moderate or severe mitral regurgitation, left ventricular systolic function, typically as evaluated by ejection fraction is hyperdynamic. Over time as the left ventricle dilates, systolic wall stress increases and eventually results in left ventricular systolic dysfunction. The presence of “normal” or reduced left ventricular systolic function is an indication of an adverse impact of mitral regurgitation on left ventricular mechanics. For this reason, even mild reductions of left ventricular systolic function, as measured by left ventricular ejection fraction, represent an indication for surgical correction of mitral regurgitation. More recently small studies have suggested that reduced global longitudinal strain (GLS) is a marker of subclinical left ventricular dysfunction and may confer an adverse
prognosis and a lower likelihood of clinical benefit from mitral valve repair or replacement.

**FIGURE 11.83.** Parasternal long-axis view recorded in a patient with a dilated cardiomyopathy and apical displacement of the papillary muscles, leading to functional mitral regurgitation. Note the dilation of the left ventricle and left atrium. This frame was recorded in mid systole. Because of the displacement of the papillary muscles, the mitral leaflets are tethered apically and cannot coapt along a normal zone. The mitral valve is attempting to coapt in a tip-to-tip manner. In this example, the actual regurgitant orifice (arrow) can be visualized. In the upper left, the normal mitral closure along a 2- to 3-mm distance is schematized. In the upper right, the abnormal closure pattern with incomplete coaptation is schematized.
FIGURE 11.84. Parasternal long-axis echocardiogram with color flow Doppler recorded in the same patient depicted in Figure 11.83. Note the large color flow Doppler jet filling more than 50% of the left atrial cavity, consistent with severe mitral regurgitation. Note also that the origin of the jet is in the area identified by the arrow in Figure 11.83 at the point of noncoaptation of the mitral leaflets.
FIGURE 11.85. Transesophageal echocardiogram recorded in a patient with a dilated cardiomyopathy and functional mitral regurgitation. A: Recorded in systole and reveals failure of coaptation of the anterior and posterior mitral leaflets. Notice the easily visualized regurgitant orifice (arrow), which corresponds to the color Doppler jet of mitral regurgitation in (B).
FIGURE 11.86. Apical four-chamber view recorded in a patient with a nonischemic dilated cardiomyopathy and severe functional mitral regurgitation. In this end systolic frame note the apical displacement of the mitral valve leaflets (arrows). The solid line denotes the plane of the mitral valve annulus and the area underneath the apically displaced mitral valve leaflets. The area thus defined is referred to as the “tenting area” and in this instance can be quantified as 4.5 cm$^2$. 

Video 11-85
MITRAL VALVE PROLAPSE

Mitral valve prolapse is commonly encountered in clinical practice. Early studies, which suggested a prevalence of mitral valve prolapse of 6% to 21% in otherwise healthy females, dramatically overestimated its true prevalence. Using contemporary criteria, mitral valve prolapse is found in 2% to 5% of the population. There are two forms of mitral valve prolapse that represent the two ends of a spectrum. In clinical practice, many patients fall between these two extremes. The first, which represents true organic heart disease, is mitral valve prolapse associated with myxomatous thickening of the leaflets. The second form of mitral valve prolapse represents mild buckling of an otherwise anatomically normal valve. It was inclusion of individuals with the latter type of mitral valve “prolapse” that inflated the apparent disease prevalence. From a clinical outcome standpoint, it is individuals with myxomatous thickening of the leaflets in association with prolapse who are most prone to complications such as progressive mitral regurgitation, spontaneous chordal rupture, neurologic events, and endocarditis. Individuals with prolapse but with otherwise anatomically normal leaflets and no mitral regurgitation are at substantially lower risk of complications.
FIGURE 11.87. Apical four-chamber view recorded in a patient with a prior posterolateral myocardial infarction and functional mitral regurgitation. In the central figure note the highly eccentric mitral regurgitation jet which courses along the lateral wall of the left atrium. The inset at the upper right is an expanded view of mitral closure. Note the normal position in systole of the anterior leaflet (upward-pointing arrow) and the apically tethered posterior leaflet (downward-pointing arrow). Note that the direction of the regurgitant jet is in the direction of the leaflet with restricted motion. [Video 11-87]
FIGURE 11.88. Transesophageal echocardiogram recorded in a patient with functional mitral regurgitation related to prior inferior wall infarction. Note the position of the anterior mitral leaflet (arrow), which in systole appears to coapt
behind the posterior leaflet. This is the result of restriction of the posterior leaflet toward the apex and results in an eccentric mitral regurgitation jet. The middle inset is a three-dimensional real-time image also demonstrating the restricted motion of the posterior leaflet in the apparent coaptation of the anterior leaflet behind the posterior leaflet.  

Video 11-88

FIGURE 11.89. Long-axis apical view of a patient with a nonischemic dilated cardiomyopathy and global left ventricular systolic dysfunction. In the central
image note the mitral regurgitation jet adhering to the posterior wall of the left atrium. The inset at the upper left is an expanded view of mitral closure in systole. Note that the posterior leaflet is apically tethered (arrow) and does not fully coapt with the anterior leaflet. The inset at the lower left is a continuous-wave Doppler through the mitral regurgitation jet. Note the dense triangular signal as well as the relatively low velocity consistent with reduced left ventricular systolic pressures.

Video 11-89
**FIGURE 11.90.** A: Transesophageal echocardiogram recorded in a patient with an ischemic cardiomyopathy and two separate mitral regurgitation jets. B: Recorded from a full volume, three-dimensional image with color flow Doppler and likewise reveals the two regurgitant jets.

**FIGURE 11.91.** Real-time three-dimensional echocardiogram recorded from a left atrial perspective in the same patient depicted in Figure 11.90. In this in systolic frame, note the two clearly visualized regurgitant orifices (arrows) of the mitral
valve related to an ischemic-mediated mitral regurgitation.

Video 11-91

coming soon
Multiple criteria have been proposed for the diagnosis of mitral valve prolapse. With M-mode echocardiography (Fig. 11.92), mitral valve prolapse is diagnosed in the presence of leaflet thickening with posterior buckling of the mitral valve during systole. This buckling can be either holosystolic or confined to late systole. From a technical standpoint, it is important that the M-mode beam be aligned to encompass the area just behind the mitral annulus if one is to document buckling of the mitral valve leaflet into the left atrium.

Transthoracic two-dimensional echocardiography is the standard for
screening for mitral valve prolapse. Several quantitative techniques have been recommended, including determining the angle in systole between the posterior aortic wall and the proximal anterior mitral valve leaflet. In general, quantitative techniques for separating mitral valve prolapse from normal closure patterns have not seen clinical acceptance. In the past, there has been much debate regarding the sensitivity and specificity of mitral valve bowing when seen in a parasternal versus apical view. It is more important to appreciate the presence or absence of valve thickening and the symmetry versus asymmetry with which the valve “prolapses.” Because the mitral annulus is not a planar structure, gradual bowing of both leaflets is to be anticipated in the apical four-chamber view but is less common in the apical two-chamber view (Figs. 11.2 and 11.3). Mitral valve bowing in the four-chamber view has been considered less specific for the diagnosis of mitral valve prolapse than detection of buckling in either a parasternal long-axis view or apical two-chamber view. Recognizing that the mitral annulus is a complex three-dimensional structure and that the mitral valve has multiple scallops, one should recognize that the view in which mitral valve prolapse is best appreciated will depend on which portion of the mitral valve is involved. The diagnosis of mitral valve prolapse should be made when one or both leaflets break the plane of the mitral annulus in a nonsymmetric manner, typically taking on a “buckling” appearance. The leaflet should be described as thickened or anatomically normal. Figures 11.93 through 11.98 depict echocardiograms in patients with varying degrees of mitral valve prolapse. Figure 11.94 represents classic myxomatous mitral valve disease with diffuse leaflet thickening and bileaflet prolapse. Figure 11.93 was recorded in an individual with prolapse of the posterior leaflet but near normal mitral valve thickness. Occasionally, very marked myxomatous degeneration, leaflet thickening, and redundancy result in a mass-like appearance, which could be confused with vegetation or tumor (Fig. 11.99). Similarly, a markedly redundant valve may buckle back on itself and result in the appearance of a cystic structure (Fig. 11.100).

Several sequelae and complications of mitral valve prolapse are well recognized. These include mitral regurgitation, ruptured chordae, and flail leaflets as well as endocarditis. Figure 11.98 shows an echocardiogram recorded in an individual with mitral valve prolapse and severe mitral regurgitation. Note the eccentric mitral regurgitation jet due to eccentric
coaptation. **Figure 11.101** shows an echocardiogram recorded in a patient with mitral valve prolapse and a partially flail leaflet. Note the highly disorganized regurgitation jet in the left atrium, which cannot be quantified by jet area methods.

Once the diagnosis of mitral valve prolapse has been established, it is important to further characterize other areas of the cardiovascular system that may also be involved. Mitral valve prolapse can be an integral part of Marfan syndrome, in which case aortic pathology may be encountered and should be evaluated. As such, detailed attention to the aortic valve and proximal aorta should be undertaken in patients in whom prolapse has been diagnosed.

**MISCELLANEOUS MITRAL VALVE ABNORMALITIES**

**Surgical Repair**

When considering patients for intervention for mitral valve disease, there are a number of clinical, hemodynamic, and echocardiographic findings of direct relevance. In general, for any individual with symptomatic valvular heart disease an anatomically corrective procedure will be indicated for both symptom relief and survival benefit. The data supporting this concept are probably most robust for mitral regurgitation where there are a number of echocardiographic findings for which surgical or interventional approaches are considered appropriate.
FIGURE 11.93. Parasternal long-axis view recorded in a patient with classic posterior mitral valve prolapse. This image was recorded at end-systole. Note the buckling of the posterior leaflet behind the plane of the mitral annulus (arrow), but only mild thickening of the mitral leaflets.

Video 11-93
FIGURE 11.94. Parasternal long-axis view recorded in a patient with classic myxomatous mitral valve disease and mitral valve prolapse. At this mid-systolic frame, note the open aortic valve and the diffuse thickening of the mitral leaflets (white arrows), both of which prolapse behind the plane of the mitral annulus (black upward-pointing arrow).
FIGURE 11.95. Parasternal long-axis (A) and short-axis (B) images recorded at end-systole in a patient with mitral valve prolapse, predominantly of the posterior leaflet. In the parasternal long-axis view, note the distinct prolapse and buckling of the posterior leaflet behind the plane of the mitral annulus, into the body of the left atrium (arrow). In B, note the prolapse of the posterior leaflet (arrows) which, in this instance, appears as a cystic mass behind the anterior leaflet within the body of the left atrium.
Surgical repair of a flail mitral valve involves placing an annuloplasty ring and resection of the flail portion of the leaflet. Other surgical techniques include placement of prosthetic chordae, chordal shortening procedures, and
translocation of chordae from one leaflet to another. After repair, the annuloplasty ring typically appears as an echodensity most easily seen in the posterior annulus area (Fig. 11.102). Because the most common repair is of the posterior leaflet, this leaflet often appears shortened with most valve motion attributable to the anterior leaflet. Three-dimensional echocardiography can be a valuable tool for assessing the integrity of mitral valve repair and detecting residual flail or ring dehiscence. See Chapters 23 and 14 for further discussion of mitral valve repair.

FIGURE 11.97. Split screen transesophageal echocardiogram recorded in a patient with myxomatous mitral valve disease and complex prolapse. In the left panel, note the myxomatous leaflets which are buckling dramatically into the left atrium (arrows). The right panel was recorded in a split-screen format with color Doppler. Note the multiple mitral regurgitation jets.
coming soon

Video 11-97
FIGURE 11.98. Transesophageal echocardiogram recorded in a patient with mitral valve prolapse. Both panels were recorded in systole. A: Note the marked prolapse of the posterior leaflet into the body of the left atrium (arrow). B: A color Doppler image also recorded in systole. Note the relatively large convergence zone and the highly eccentric mitral regurgitation jet directed toward the atrial septum. [Video]

Video 11-98
FIGURE 11.99. Apical four-chamber view recorded in a patient with myxomatous mitral valve disease and pronounced mitral valve prolapse. In this example, the combination of myxomatous thickening and exaggerated buckling of the leaflet results in the appearance of a mass on the left atrial side of the mitral leaflet. Transesophageal echocardiography confirmed the absence of a mass and that this effect was (arrows) due to the pronounced myxomatous thickening and prolapse alone.
Video 11-99
**FIGURE 11.100.** A: Transesophageal echocardiogram recorded in a patient with marked mitral valve prolapse of multiple scallops, resulting in the appearance of cystic masses (arrows) on the mitral valve. B: An expanded view of a different portion of the mitral valve revealing a partial flail of one scallop (small arrow) and direct visualization of the regurgitant channel (bold arrow).

**Calcification of the Mitral Annulus**

Fibrosis and calcification of the fibrous skeleton of the heart are common sequelae of aging. This is most often appreciated in the posterior mitral valve annulus and can range from limited degrees of focal calcific deposits to nearly circumferential heavy calcification. Figures 11.103 through 11.107 are echocardiographic examples of mitral annular calcification. In addition to
age, other conditions that accelerate annular calcification include hypertension and chronic renal insufficiency. In patients with chronic renal insufficiency, the degree of annular calcification can be substantial and take on a mass-like effect that has been confused for tumor. Mild degrees of mitral regurgitation are not uncommon. If the fibrotic and calcific process extends throughout the entire annulus and into the valve leaflets, secondary leaflet dysfunction can occur and result in greater mitral regurgitation. In advanced cases, invasion of the proximal leaflet portions by the fibrotic and calcific process can reduce the mitral orifice and result in functional mitral stenosis (Fig. 11.105). This type of mitral stenosis is not amenable to balloon valvuloplasty and may pose substantial challenges for surgical correction. Extensive mitral annular calcification may result in difficulty in seating a prosthetic valve. Patients with heavy mitral annular calcification are more likely to have subsequent paravalvular regurgitation than are patients without calcification.
FIGURE 11.101. Parasternal long-axis view echocardiogram with color flow Doppler imaging recorded in a patient with mitral valve prolapse and a partial flail leaflet. There is a highly eccentric and disorganized mitral regurgitation jet, with one component confined behind the anterior mitral leaflet and the second component directed immediately posteriorly (arrows).
Video 11-101
FIGURE 11.102. Parasternal long-axis (A) and short-axis (B) echocardiograms
recorded in a patient after mitral valve repair with an annular ring. The ring is noted as an echo density at the base of the posterior mitral leaflet. In both the long- and short-axis views in real-time, note that most of the mitral valve leaflet motion occurs with the anterior rather than the posterior leaflet. An, mitral annulus.
FIGURE 11.103. Parasternal long-axis view recorded in a patient with heavy mitral annular calcification (downward-pointing arrows). Note the acoustic shadowing behind the dense calcium in the annulus (upward-pointing arrow). This shadowing should not be confused for an abscess. The inset is an M-mode echocardiogram recorded from the same patient. Note the dense band of calcium posterior to the mitral valve.

Video 11-103

Patients with extensive mitral annular calcification may have a number of associated echocardiographic findings. Commonly there may be extensive
shadowing posterior to the mitral annulus (Fig. 11.104). This shadowing should not be confused for an annular abscess or vascular structure. Extensive annular calcification may also invade the posterior ventricular myocardium and posterior mitral valve leaflet. Occasionally small mobile echodensities are seen on either the atrial or ventricular aspect of the calcified annulus. The differential of this is broad and obviously includes vegetation. Not uncommonly these mobile echodensities simply represent degenerating portions of the annulus and/or mitral apparatus. Superimposed thrombus on a heavily calcified mitral annulus has been noted but is not common.

**FIGURE 11.104.** Transthoracic echocardiogram recorded in a patient with end-stage renal disease and marked mitral annular calcification. In the central image note the marked calcification of the posterior mitral annulus (*small arrows*). The calcification has created extensive shadowing posterior to the mitral annulus (*horizontal arrows*). This shadowing should not be confused for an annular abscess or other structure. In the real-time image note the multiple small mobile echodensities attached to the annulus which in this case represents degenerative components of the calcified annulus and/or posterior leaflet. The inset at the lower left is a parasternal short-axis view from the same patient. Again note the extensive calcification of the posterior annulus (*arrows*) creating shadowing. This frame was recorded at end-diastole and the open anterior mitral valve leaflet is as noted by the *small downward-pointing arrow*. (©)
FIGURE 11.105. Apical four-chamber view recorded in a patient with advanced mitral annular calcification (arrows). The inset is a transmitral Doppler confirming a mean gradient of 18 mm Hg across the functionally stenotic mitral orifice.

Video 11-105

Not uncommonly reverberation or side lobe artifacts will arise from a heavily calcified mitral annulus which appear as vague echodensities within the body of the left atrium, attached to the mitral annulus. These can
occasionally be confused for vegetation, thrombus, tumor, or other mass. This generally does not result in substantial confusion for an experienced echocardiographer, however when the study is performed for evaluation of possible endocarditis further evaluation with transesophageal echocardiography may be necessary to demonstrate the artifactual nature of these echoes (Fig. 11.108).

**Tumors of the Mitral Valve**

On occasion, a myxoma may arise from the mitral valve rather than from the atrial septum (Fig. 11.109). They present as a relatively large, bulky tissue density mass moving with the mitral valve tissue. More commonly, a typically located atrial myxoma on a relatively long stalk will move in close conjunction with the mitral valve leaflets and appears to be physically attached to the leaflet. Transesophageal echocardiography can often identify the true location of the tumor attachment and confirm the separation of the tumor from the left atrial side of the mitral leaflet.

**FIGURE 11.106.** Transesophageal echocardiogram recorded in a patient with dense, nearly circumferential annular calcium (downward-pointing arrows). The
mitral leaflet tips are noted by the *upward-pointing arrows*. In the real-time image, note the restricted orifice related to the marked circumferential calcification and the preserved mobility of the shortened mitral leaflets. The inset at the upper left is a three-dimensional echocardiogram recorded from within the left atrium, depicting the circumferential, dense annular calcium (arrows).
FIGURE 11.107. Apical two-chamber transthoracic view (A) and three-dimensional transesophageal echocardiographic image (B) recorded in a patient with marked symmetric mitral annular calcification. In panel A, note the dense masses of calcium in both the anterior and posterior mitral annulus (arrows). In panel B, note the marked irregular annular calcific deposits (arrows). The inset at the upper left is a close-up view of the area depicted by the arrows in the main illustration. In this figure, the instrument gains have been decreased to as low as possible, and as such, all normal tissue myocardium has been suppressed. The heavily calcific deposits retain a degree of ultrasound signature and therefore remain visible in the image.

Video 11-107A
Other mitral valve tumors include the papilloma or fibroelastoma. These typically present highly mobile masses 2 to 10 mm in diameter, attached to the distal mitral valve or to the chordae tendineae (Fig. 11.110). Smaller fibroelastomas may appear as a highly mobile, strand-like mass.

A final, rare mass to be noted on a mitral valve is the mitral blood cyst. This is a developmental cystic structure that is more commonly encountered in pediatric populations. Cysts can range in size from 2 mm to 1 cm and appear as smooth, usually spherical or ovoid cystic structures. Single or multiple cysts can be encountered on the mitral valve. The echocardiogram shown in Figure 11.111 was recorded in an asymptomatic woman with multiple mitral valve blood cysts. Because of the cyst bulk, they can interfere
with appropriate mitral valve coaptation and result in secondary mitral regurgitation, and periodic surveillance echocardiography is probably warranted.

**FIGURE 11.108.** Parasternal long-axis view recorded in a patient with a calcified mitral annulus (*downward-pointing arrow*). Note the fainter vague echo arising off the mitral annulus which appears in the body of the left atrium (*small arrow*). This represents a side lobe artifact off the mitral annulus which could be confused for a vegetation, clot, or other pathologic mass. Imaging with transesophageal echocardiography may occasionally be necessary to resolve the nature of this mass as either being a pathologic mass attached to the annulus versus an artifact.
FIGURE 11.109. Transesophageal echocardiogram recorded in a patient with a myxoma of the mitral valve leaflet. Note the smooth, homogeneous, nearly spherical mass attached to the mitral leaflet (arrow), which was demonstrated at the time of surgical excision to be an atypically located myxoma.
FIGURE 11.110. Transthoracic echocardiogram recorded in a young patient incidentally noted to have a mass on the mitral valve chordae, subsequently proven to be fibroelastoma. In the central figure note the mass attached to the proximal mitral chordae (arrows). The mass is also visualized in the short-axis view at the upper left inset. In the real-time image note the oscillating nature of the mass with a suggestion of a frond-like appearance.
FIGURE 11.111. Transthoracic apical four-chamber view recorded in a patient with mitral valve blood cysts. In A, note the cystic-appearing mass associated with the tips of the mitral leaflet and chordae representing a mitral valve blood cyst.
(arrow). B is the color flow Doppler image demonstrating mild to moderate mitral regurgitation, presumably as a result of either cyst impingement on normal coaptation or secondary degenerative changes of the leaflet.  

Video 11-111A

Video 11-111B
FIGURE 11.112. Transesophageal echocardiogram recorded in a patient with a history of mitral valve endocarditis 2 years prior. In this instance the vegetation itself has resolved, however there is a residual perforation at the base of the anterior mitral valve leaflet through which there is a mitral regurgitation jet of moderate severity. The panel at the upper left is a real-time three-dimensional echocardiogram recorded in the same patient from which the three-dimensional nature of the perforation and subsequent jet can be appreciated. [Video 11-112]

Video 11-112
**Aneurysms of the Mitral Valve**

On occasion, one encounters a discrete aneurysmal outpouching of the mitral leaflet, most commonly at the base of the anterior leaflet and protruding into the left atrium. In many instances, this is the sequela of endocarditis, in which case the aneurysm may be thick walled or irregular in contour and associated with perforation into the left atrium. More rarely, a similar aneurysm is seen with thin walls and without evidence of endocarditis. The etiology of these aneurysms is unknown but assumed to be congenital.

**Endocarditis and Valve Perforation**

Endocarditis of the mitral valve is usually detected by the presence of a vegetation and pathologic mitral regurgitation. On occasion, healed endocarditis results in a chronic perforation of one of the mitral leaflets (Fig. 11.112). Perforations can be very well characterized with three-dimensional echocardiography. The degree of mitral regurgitation is proportional to the size of the perforation. Abscess of the mitral valve annulus is well-described sequela of endocarditis and typically is confined to the posterior annulus. Further discussion of annular abscess and other sequelae of endocarditis can be found in Chapter 13.

**FIGURE 11.113.** Parasternal long-axis view recorded in a patient with systemic lupus erythematosus and Libman–Sacks lesions. In A, note the mild diffuse thickening of the mitral valve leaflet tips (arrows) and in B, recorded with color flow Doppler, the severe degree of resultant mitral regurgitation.
FIGURE 11.114. Parasternal long-axis view recorded in a patient with a congenitally cleft mitral valve. A was recorded at end-systole. Note the mild focal thickening at the tip of the anterior mitral leaflet (arrow). B was recorded with color
flow Doppler and reveals a highly eccentric, posteriorly directed mitral regurgitation jet (arrow).

Patients with connective tissue disease, as typified by systemic lupus, may develop noninfectious vegetative lesions on the mitral valve (Libman–Sacks lesions). They are typically located on the atrial aspect of the leaflet (Fig. 11.113) and are associated with variable degrees of regurgitation. They may stabilize with successful treatment of the underlying disease but if resulting in severe symptomatic mitral regurgitation often require surgical replacement. See Chapter 22 for further discussion.
Cleft Mitral Valve

A cleft mitral valve is a congenital lesion resulting in mitral regurgitation. It is often seen as part of an endocardial cushion defect where a primum atrial septal defect and inlet VSD will be encountered. Cleft mitral valve is also encountered as an isolated entity. From an echocardiographic perspective, one often notes mild thickening at the tip of the anterior leaflet (where the cleft is most prevalent) and with color flow imaging a posteriorly directed mitral regurgitation jet (Fig. 11.114). In the parasternal short-axis view, the actual cleft can be visualized as a break in the continuity of the anterior mitral leaflet in diastole (Fig. 11.115).

![Image of cleft mitral valve](image)

**FIGURE 11.115.** Parasternal short-axis view recorded in the same patient depicted in Figure 11.114. This image was recorded at end-diastole. Note the break in the continuity of the anterior mitral leaflet (arrows) which, in the real-time image, can be seen to open in a “drawbridge” manner rather than with continuity of the anterior mitral leaflet.
Annular Dehiscence

Annular dehiscence is a very infrequent sequela of blunt chest trauma. The presumed mechanism is a sudden dramatic increase in intracardiac pressure against a closed mitral valve resulting in tearing of the posterior leaflet from the mitral valve annulus or less commonly of a portion of the annulus from the adjoining wall. Annular dehiscence results in substantial mitral regurgitation with an eccentrically directed mitral regurgitation jet. Transesophageal echocardiography is essential to confirm the diagnosis. Anatomically, the defect is similar to that seen in an annular abscess and the diagnosis of dehiscence requires both the echocardiographic appearance and a history of chest trauma sufficient to have caused the injury.
FIGURE 11.116. Parasternal long-axis view of the left ventricle recorded in a patient with a history of mantle radiation for lymphoma 25 years prior. In the central image note the thickened aortic valve cusps in the rigid board-like appearance of the proximal two-thirds of the anterior mitral leaflet (arrows). At the upper left panel note the transmitral Doppler recorded from an apical position confirming a 5 mm Hg transmitral gradient. The inset at the lower right is an expanded view. Note the thickened aortic valve cusps consistent with radiation-induced valve disease and the thickening and immobility of the proximal two-thirds of the anterior mitral leaflet (three small arrows). Only the distal 25% of the anterior mitral leaflet has retained mobility which can be better appreciated in the real-time image (single arrow).
Radiation Damage
Because of the degree to which radiation therapy is anatomically targeted and the cardiovascular system shielded, it is increasingly uncommon to encounter radiation-induced mitral valve disease. It is most common after mediastinal radiation delivered as therapy for Hodgkin or non-Hodgkin lymphoma (mantle radiation). It may be the sequela of radiation therapy occurring 10 to 15 years before presentation. The degree and location of damage are highly variable and dependent on the direction of the radiation beam. Because most radiation portals are anterior, it is the more anterior cardiac structures that are most prone to injury, including the anterior mitral valve leaflet (Fig. 11.116). Although the nature of radiation damage can be highly variable, the most common finding is fibrosis and stiffening of the proximal portions of the anterior mitral valve leaflet, often in conjunction with radiation-induced aortic valve disease. Other issues regarding radiation-induced cardiac disease are discussed in Chapter 22.

Carcinoid and Diet–Drug Valvulopathy
There are several metabolic syndromes that affect the mitral valve. The first to be described was carcinoid heart disease, which more often involves the tricuspid and pulmonary valves. The lesions are similar to those seen in
ergotamine heart disease and consist of diffuse thickening of the valve and chordae resulting in a combination of stenosis and regurgitation. Because the biologically active, serotonin-related metabolites are metabolized in the lung, left-sided structures are typically spared. In instances of pulmonary metastases or a right-to-left shunt, the mitral or aortic valves may also be involved (Fig. 11.117).

A nearly identical abnormality, both pathologically and echocardiographically, has been noted in patients taking anorexic agents, typically a combination of phentermine and fenfluramine. Substantial controversy exists regarding the true prevalence of diet–drug valvulopathy. The majority of well-done, case-controlled trials suggest a prevalence of significant (moderate or greater) mitral insufficiency due to diet–drugs substantially less than suggested in initial reports. Subsequent studies have suggested that diet–drug valvulopathy often regresses after withdrawal of the agents. As these drugs have been largely withdrawn from the market encountering new cases of diet–drug valvulopathy would be expected to be exceptionally uncommon.

**FIGURE 11.117.** Transesophageal echocardiogram recorded in a patient with
metastatic carcinoid disease. In this mid-diastolic frame note the board-like rigidity in immobility of the mitral leaflets which has resulted in mild to moderate mitral regurgitation is noted in the panel at the lower left. At the upper left is a three-dimensional view from within the left atrium of the functionally stenotic mitral valve nicely demonstrating diffuse leaflet thickening and a restricted orifice. Typical carcinoid valve involvement is also noted on the tricuspid valve.
Suggested Readings

**General Principles/Guidelines**


**Mitral Regurgitation**


transsthoracic echocardiography in quantifying mitral regurgitation: a comparison with conventional
Kusunose K, Cremer PC, Tsutsui RS, et al. Regurgitant volume informs rate of progressive cardiac
dysfunction in asymptomatic patients with chronic aortic or mitral regurgitation. *JACC Cardiovasc
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Mentias A, Naji P, Gillinov AM, et al. Strain echocardiography and functional capacity in
asymptomatic primary mitral regurgitation with preserved ejection fraction. *J Am Coll Cardiol*
myxomatous mitral regurgitation undergoing exercise echocardiography. *Circulation*
2014;129:1310–1319.
O’Gara PT, Grayburn PA, Badhwar V, et al. 2017 ACC expert consensus decision pathway on the
management of mitral regurgitation: a report of the American College of Cardiology Task Force on
with degenerative mitral valve regurgitation predicts postoperative worsening of left ventricular
patients with heart failure. A quantitative analysis of 1256 patients with ischaemic and non-
Silbiger JJ. Novel pathogenetic mechanisms and structural adaptations in ischemic mitral regurgitation.
Thavendiranathan P, Phelan D, Collier P, Thomas JD, Flamm SD, Marwick TH. Quantitative
of mitral regurgitation severity: a prospective multicenter trial. *J Am Coll Cardiol* 2015;65:1078–
1088.
Yang LT, Liu YW, Shih JY, et al. Predictive value of left atrial deformation on prognosis in severe
regurgitation: geometric differences from three-dimensional transesophageal echocardiography. *J
Zoghbi WA, Adams D, Bonow RO, et al. Recommendations for noninvasive evaluation of native
valvular regurgitation: a report from the American Society of Echocardiography developed in
collaboration with the Society for Cardiovascular Magnetic Resonance. *J Am Soc Echocardiogr*
Zurcher F, Brugger N, Jahren SE, de Marchi SF, Seiler C. Quantification of multiple mitral regurgitant
jets: an in vitro validation study comparing two- and three-dimensional proximal isovelocity surface

**MITRAL STENOSIS**


**Mitral Valve Prolapse**

Delling FN, Vasan RS. Epidemiology and pathophysiology of mitral valve prolapse: new insights into disease progression, genetics, and molecular basis. *Circulation* 2014;129:2158–2170.


**Miscellaneous Topics**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Chapter 12
Tricuspid and Pulmonary Valves

CLINICAL OVERVIEW

Compared to the aortic and mitral valves, primary pathology of the tricuspid and pulmonary valves is relatively infrequent in adults. Clinical entities resulting in pulmonary and tricuspid valvular disease are listed in Table 12.1. The appropriate use of echocardiography for known or suspected tricuspid and pulmonary valve disease is outlined in Table 12.2.

Multimodality imaging with cardiac magnetic resonance imaging (MRI) or computed tomography (CT) has less of an established role in disease of the tricuspid and pulmonary valves than for many other forms of cardiac disease. MRI plays a significant role for evaluation of right ventricular size and function, as well as for prognostication and procedural planning in congenital heart disease.

PULMONARY VALVE

The normal pulmonary valve is a three-cusp structure, anatomically similar to the aortic valve. It inserts into the pulmonary annulus distal to the right ventricular outflow tract. Developmentally, the aorta and pulmonary arteries arise in a parallel fashion. The two arteries then rotate such that the right ventricular outflow tract, pulmonary valve, and proximal pulmonary artery effectively wrap around the aortic valve and the ascending aorta.
Table 12.1  DISEASES OF THE TRICUSPID AND PULMONARY VALVES

<table>
<thead>
<tr>
<th>Disease</th>
<th>Stenosis</th>
<th>Regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rheumatic heart disease</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Carcinoid heart disease</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Obstructive tumors</td>
<td>✓</td>
<td>–</td>
</tr>
<tr>
<td>Congenital pulmonary stenosis</td>
<td>✓</td>
<td>±</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>±</td>
<td>✓</td>
</tr>
<tr>
<td>Ebstein anomaly</td>
<td>–</td>
<td>✓</td>
</tr>
<tr>
<td>Endocardial fibroelastosis</td>
<td>±</td>
<td>✓</td>
</tr>
<tr>
<td>Tricuspid valve prolapse</td>
<td>–</td>
<td>✓</td>
</tr>
<tr>
<td>Traumatic rupture</td>
<td>–</td>
<td>✓</td>
</tr>
<tr>
<td>Right ventricular infarction</td>
<td>–</td>
<td>✓</td>
</tr>
<tr>
<td>Ischemic papillary muscle dysfunction</td>
<td>–</td>
<td>✓</td>
</tr>
<tr>
<td>Pulmonary hypertension(^a)</td>
<td>–</td>
<td>✓</td>
</tr>
<tr>
<td>Left-to-right shunt with dilation(^a)</td>
<td>–</td>
<td>✓</td>
</tr>
<tr>
<td>Right ventricular cardiomyopathy(^a)</td>
<td>–</td>
<td>✓</td>
</tr>
<tr>
<td>Pacemaker leads, right heart catheter</td>
<td>–</td>
<td>✓</td>
</tr>
</tbody>
</table>

\(^a\)Tricuspid disease is secondary to right ventricular dilation. The leaflets are anatomically normal.

When viewed with two-dimensional echocardiography, typically only one or two cusps are simultaneously visualized. Specialized imaging planes may allow visualization of the pulmonary valve in its short axis; however, the relatively thin, highly pliable leaflets are often not visualized in their entirety. Visualization of the pulmonary valve in adults typically is optimal from a parasternal short-axis transducer position at the base of the heart, at which time the aortic valve and/or proximal aorta are simultaneously visualized (Fig. 12.1). The bifurcation of the pulmonary artery is also visualized from this view (Fig. 12.2). In addition to the parasternal short-axis view, a long-axis projection of the right ventricular outflow tract and pulmonary valve can be obtained by rotation and angulation of the imaging plane toward the right shoulder (Fig. 12.3). This visualization plane is often problematic in large-stature adults. A final transthoracic imaging plane for visualization of the
pulmonary valve is the subcostal view in which, with anterior angulation, the entire sweep of the right ventricular outflow tract can often be visualized including the pulmonary valve leaflets (Fig. 12.4).

<table>
<thead>
<tr>
<th>Indication</th>
<th>Appropriateness</th>
<th>Score (1–9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15. Evaluation of suspected pulmonary hypertension including evaluation of</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>right ventricular function and estimated pulmonary artery pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>32. Severe deceleration injury or chest trauma when valve injury,</td>
<td>A (8)</td>
<td></td>
</tr>
<tr>
<td>pericardial effusion, or cardiac injury are possible or suspected</td>
<td></td>
<td></td>
</tr>
<tr>
<td>42. Routine surveillance of trace valvular regurgitation</td>
<td>rA (1)</td>
<td></td>
</tr>
<tr>
<td>43. Routine surveillance (&lt;3 yrs) of mild valvular regurgitation without a</td>
<td>rA (2)</td>
<td></td>
</tr>
<tr>
<td>change in clinical status or cardiac exam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>44. Routine surveillance (≥3 yrs) of mild valvular regurgitation without a</td>
<td>U (4)</td>
<td></td>
</tr>
<tr>
<td>change in clinical status or cardiac exam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>45. Routine surveillance (&lt;1 yr) of moderate or severe valvular</td>
<td>U (6)</td>
<td></td>
</tr>
<tr>
<td>regurgitation without a change in clinical status or cardiac exam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>46. Routine surveillance (≥1 yr) of moderate or severe valvular</td>
<td>A (8)</td>
<td></td>
</tr>
<tr>
<td>regurgitation without change in clinical status or cardiac exam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>52. Initial evaluation of suspected infective endocarditis with positive</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>blood cultures or a new murmur</td>
<td></td>
<td></td>
</tr>
<tr>
<td>92. Initial evaluation of known or suspected adult congenital heart</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>93. Known adult congenital heart disease with a change in clinical status</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>or cardiac exam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>94. Reevaluation to guide therapy in known adult congenital heart disease</td>
<td>A (9)</td>
<td></td>
</tr>
</tbody>
</table>

FIGURE 12.1. Transthoracic parasternal short-axis view at the base of the heart visualizing the pulmonary valve. Notice the central closure point in diastole (A) and the inability to visualize the normal leaflets that are fully open in systole (B).

Video 12-1

With transesophageal echocardiography, the views that maximize visualization of the pulmonary valve include imaging at the level of the aorta in a 40- to 60-degree plane and in the horizontal (0-degree) plane at relatively shallow depths (typically 25 to 30 cm from the incisors) with counterclockwise rotation of the probe. In this view, the bifurcation of the pulmonary artery is typically seen and the pulmonary valve can be likewise visualized (Fig. 12.5). An additional transesophageal echocardiographic window providing visualization of the pulmonary valve is often obtained from a deep gastric imaging plane. With clockwise rotation of the transducer, the entire sweep of the right ventricular inflow and outflow tracts can often be obtained and simultaneous visualization of the right atrium, tricuspid valve, right ventricular outflow tract, pulmonary valve, and proximal pulmonary artery often accomplished (Fig. 12.6).
FIGURE 12.2. Parasternal short-axis view at the base of the heart depicting normal anatomy. In this view, the main pulmonary artery (MPA) and bifurcation into the left and right pulmonary arteries (LPA, RPA) are clearly visualized.

Video 12-2
FIGURE 12.3. Parasternal long-axis view of the RVOT, PA, and pulmonary valve recorded in diastole. In the real-time image, note the full motion of the valve to the margins of the arterial wall. Video 12-3
FIGURE 12.4. Subcostal short-axis view of the base of the heart shows a portion of the right atrium, tricuspid valve, right ventricle and outflow tract, pulmonary valve, and pulmonary artery. Structures are as noted on the schematic in the upper left of the figure. [ ]
Using M-mode echocardiography from a parasternal approach, motion of the pulmonary valve can be recorded. Typically, only one leaflet will be intersected by the M-mode beam. Characterization of pulmonary valve motion provided one of the earlier clues to the presence of pulmonary hypertension and indirect evidence of other right heart pathologies. There are several components to normal pulmonary valve motion (Fig. 12.7). The first is presystolic A-wave motion away from the transducer, which is due to relatively low-amplitude excursion (<6 mm) of the pulmonary valve with atrial systole.

This phenomenon is dependent on mechanical atrial systole and is not present in atrial fibrillation. It is also dependent on relatively low pulmonary artery diastolic pressures so that atrial contraction creates the driving force for partial opening of the pulmonary valve. The pulmonary valve leaflet then moves posteriorly (in a patient in supine position), that is, away from the transducer during systole. It is not uncommon for visualization to be incomplete throughout the entire cardiac cycle and for only the A-wave and opening slope of the pulmonary valve to be detectable. With excellent acoustic windows, the full opening and closing pattern of the pulmonary
valve can occasionally be appreciated (Fig. 12.8).

Pulsed and continuous wave Doppler imaging can also be recorded at the level of the pulmonary valve. Typically, the pulmonary valve flow profile is recorded from a parasternal short-axis view along an interrogation line identical to that used for M-mode echocardiography. Figure 12.9 schematizes the appropriate sample volume position and provides an example of normal pulsed Doppler of pulmonary flow. It should be emphasized that many of the indirect parameters of right heart hemodynamics that can be derived from the pulmonary outflow tract spectral profile are dependent on optimal imaging planes, including the central position of the sample volume within the pulmonary artery (as opposed to recording it along the periphery) and recording at a level just distal to the tips of the pulmonary valve. The normal pulmonary outflow tract velocity ranges from 1 to 1.5 m/sec. As with other valves, the time velocity integral of this valve can be determined and in combination with the outflow tract dimension can be used to calculate volumetric flow (Fig. 12.9). Other parameter of the pulmonary outflow tract velocity includes acceleration time. Acceleration time is defined as the time in milliseconds from the onset of ejection to peak systolic velocity. In normal individuals, acceleration time exceeds 140 milliseconds and progressively shortens with increasing degrees of pulmonary hypertension (Fig. 12.10).

There is an inverse relationship between pulmonary acceleration time and pulmonary artery systolic, diastolic, and mean pressures. An acceleration time of less than 70 to 90 milliseconds is typically indicative of a pulmonary artery systolic pressures $\geq 70$ mm Hg. This assessment has been largely replaced by the more direct Doppler assessment of right ventricular systolic pressure from the tricuspid regurgitation signal. On occasion one encounters a patient without a measurable tricuspid regurgitation velocity and a short acceleration time may be the only evidence of pulmonary hypertension.
FIGURE 12.6. Transesophageal echocardiogram recorded at 91 degrees from a low esophageal position showing the body and outflow tract of the right ventricle, the pulmonary valve in a closed position (arrow). 

Video 12-6

Pulmonary Valve Stenosis

Pulmonary valve stenosis is virtually always congenital in origin and is easily
detected and quantified using two-dimensional echocardiographic and Doppler techniques. The hallmark of congenital pulmonic stenosis is thickening and systolic doming of the pulmonary valve cusps (Figs. 12.11 and 12.12). Continuous wave Doppler can be used to accurately determine the peak instantaneous and mean gradients (Fig. 12.13). Because the orientation of the right ventricular outflow tract and pulmonary artery flow is directed posteriorly, there is a natural alignment of the interrogating beam with the direction of flow, and off-angle interrogation is less of a problem than with aortic stenosis.

On M-mode echocardiography (Figs. 12.7 and 12.8), the findings of pulmonary valve stenosis are accentuated A-wave amplitude (>6 mm) with thickening of the leaflets. The accentuated A wave occurs only in patients in sinus rhythm and is probably dependent on the presence of concurrent right ventricular hypertrophy. The origin of the accentuated A wave is the relatively elevated right ventricular diastolic pressure in comparison with the pulmonary artery diastolic pressure. With atrial contraction, pressure is
transmitted by the hypertrophied noncompliant right ventricle to the pulmonary valve and pulmonary artery resulting in accentuated presystolic opening of the pulmonary valve. As noted previously, this is a qualitative descriptor implying the presence of pulmonary valve stenosis but provides no quantitative information.

**FIGURE 12.8.** M-mode echocardiograms recorded in patients with different abnormalities. A, B: Recorded in patients with pulmonary hypertension. Note the loss of the pulmonic valve A wave (*downward-pointing arrow*) and midsystolic notching (*upward-pointing arrow*) of the valve. B: Note the low-amplitude biphasic A wave. C: Image recorded in a patient with infundibular obstruction shows coarse fluttering of the valve in systole. D: Image recorded in a patient with
pulmonary valve stenosis. Note the accentuated A wave (1 cm).

**FIGURE 12.9.** Schematic representation of the methods for recording pulmonary/right ventricular outflow tract velocities. The parasternal short-axis view is used with the interrogating beam aimed posteriorly along the long axis of the proximal pulmonary artery. The spectral display is schematized at the lower right, including its various components such as time velocity integral (TVI) and acceleration time (AT). In the upper right is an example of a normal flow profile. The method for calculating stroke volume from these parameters is also displayed.

\[
\text{Area RVOT} = 2 \pi r^2 \\
\text{Stroke Volume} = TVI_{RVOT} \times \text{Area}_{RVOT}
\]
FIGURE 12.10. Spectral flow profiles recorded in a normal individual (A) with an AT of 190 milliseconds and a patient with significant pulmonary hypertension in whom the acceleration time is 80 milliseconds (B). AT, acceleration time.
FIGURE 12.11. Parasternal short-axis transthoracic echocardiogram recorded in a patient with congenital pulmonic stenosis. Note the thickening of the pulmonary valve cusps (arrows) and the continuous wave Doppler velocity of 4.5 m/sec corresponding to a peak pressure gradient across the pulmonary valve of 81 mm Hg. The color Doppler image depicts eccentric acceleration toward the stenotic orifice as well as an eccentric jet in the pulmonary artery.
Transesophageal echocardiogram recorded in an adolescent with congenital pulmonary valve stenosis. This image was recorded in midsystole. Note the thickening of the pulmonary valve leaflets and the doming motion (arrows) characteristic of valvular pulmonary stenosis. (Courtesy of Gregory Ensing, MD.)

**Pulmonary Valve Regurgitation**

Inconsequential degrees of pulmonary regurgitation are often noted with color Doppler imaging in normal disease-free subjects (Fig. 12.14) and should be considered a normal variant. These inconsequential jets of pulmonary valve insufficiency may arise centrally or more peripherally at the junction of the valve cusps with the pulmonary artery (Fig. 12.15). When they arise immediately adjacent to the aortic wall, they have been confused for a pathologic communication between the aorta and the pulmonary artery. Recognition of the exclusively diastolic flow should avoid any confusion.

There are several pathologic causes of pulmonary valve regurgitation, including its association with pulmonary valve stenosis. Dilation of the pulmonary annulus, which can be idiopathic, or due to pulmonary artery dilation as a consequence of pulmonary hypertension, also results in pulmonary valve regurgitation. Occasionally, one encounters congenital absence of one or more pulmonary valve cusps, which results in severe pulmonary valve regurgitation.
Detection of pulmonary valve regurgitation relies heavily on color flow imaging. Using color Doppler imaging, typically from a parasternal short-axis view at the base of the heart, one detects a diastolic retrograde jet. Using pulsed Doppler imaging, one can detect a retrograde spectral profile directed toward the transducer similar to that seen in aortic regurgitation. Because mild degrees of pulmonary valve regurgitation can be highly eccentric, blind scanning with spectral Doppler may miss the pulmonary valve regurgitation jet, whereas it is easily detected by color flow Doppler imaging.

FIGURE 12.13. Continuous wave Doppler imaging through the right ventricular outflow tract and pulmonary valve in a patient with PS. Note the peak pressure gradient of 61 mm Hg and the presence of concurrent PI. PI, pulmonary valve insufficiency; PS, pulmonary valve stenosis.
FIGURE 12.14. Parasternal short-axis view at the base of the heart in a normal individual reveals trivial central pulmonary valve insufficiency. A: Note the very small central regurgitant jet (arrow). B: Note the faint early diastolic retrograde Doppler spectral signal consistent with minimal pulmonary insufficiency.
Determination of the severity of pulmonary valve regurgitation is less well validated than determination of aortic regurgitation, in large part due to the lack of reliable standards for comparison. In general, similar guidelines are clinically used for determining the severity of pulmonary valve regurgitation, including determination of overall jet size, depth of penetration into the right ventricle, vena contracta width, and its overall width in relation to the right ventricular outflow tract (Fig. 12.16). One should also rely on indirect evidence of a hemodynamic effect from the pulmonary valve regurgitation such as right ventricular dilation and a right ventricular volume overload pattern. The latter, in the absence of other causes of right ventricular overload, is evidence of at least moderate pulmonary valve regurgitation.
FIGURE 12.15. Parasternal short-axis view recorded at the base of the heart in a patient with minimal pulmonary valve insufficiency originating at the lateral aspect of the cusp commissure. Because this jet originates immediately adjacent to the aorta, it could be confused for an aorta-pulmonary fistula. Note, however, the exclusively diastolic flow, which would not be expected in the presence of the true shunt.
Video 12-15

Color flow Doppler imaging can be misleading in the presence of wide-open or “free” pulmonic regurgitation. This phenomenon is seen in patients with congenital absence of one or more pulmonary valve cusps or who have had resection of one or more cusps for repair of severe congenital stenosis in infancy. Because the pulmonary artery is a low-pressure system and there is no constraining regurgitant orifice, a classic convergence zone, vena contracta and downstream jet may not be easily visualized, but rather one notes a continuous color flow signal in the right ventricular outflow tract and proximal pulmonary artery without the classic findings of a true regurgitant “jet.” The spectral Doppler profile will help confirm the nearly continuous “to and fro” flow through the outflow tract (Fig. 12.17). Additionally, the regurgitant profile is triangular with truncated late diastolic flow or cessation of flow prior to the end of mechanical diastole.
FIGURE 12.16. Parasternal short-axis view color Doppler flow images recorded in patients with mild (A), moderate (B), and severe (C) pulmonary valve insufficiency.
Video 12-16c
FIGURE 12.17. Parasternal short-axis view recorded in an individual following a pulmonary valvectomy for treatment of severe congenital pulmonic stenosis as an infant. Note the dilated right ventricular outflow tract and proximal PA. The anticipated plane of the pulmonary valve is as noted by the arrow in A. B: Recorded in early diastole notice the “free” pulmonic insufficiency without any evidence of an organized regurgitant “jet” or convergent zone. The small inset is a continuous wave Doppler through the right ventricular outflow tract revealing a peak systolic velocity of approximately 1.1 m/sec and a dense, brief, triangular-shaped pulmonic insufficiency signal terminating well before ventricular systole.

As with other valvular lesions, inspection of the retrograde spectral signal also provides indirect clues to the severity of pulmonary valve regurgitation, with relatively dense signals suggesting a higher volume of regurgitant blood
flow than faint signals, and short deceleration times having the same implication as for aortic regurgitation (Fig. 12.18).

The flow velocity of pulmonary valve regurgitation can be used to calculate pulmonary artery diastolic pressure using the modified Bernoulli equation. In this setting, one calculates the end-diastolic gradient between the pulmonary artery and the right ventricular outflow tract from the velocity of the pulmonary regurgitation jet (Fig. 12.19). If one then adds an assumed right ventricular diastolic pressure (in turn assumed to equal right atrial pressure), the equation $P_{ADP} = RA + \Delta P_{pv}$ can be applied, where $\Delta P_{pv}$ equals the pressure gradient between the pulmonary artery and the right ventricular outflow tract. When combined with the determination of right ventricular systolic pressure from the tricuspid regurgitation jet, it allows calculation of both systolic and diastolic pulmonary artery pressures. Using the combination of pulmonary artery diastolic and systolic pressures, one can then calculate mean pulmonary artery pressure as $P_{A_{mean}} = (P_{A_{systolic}} + 2P_{A_{diastolic}})/3$. 
FIGURE 12.18. Continuous wave spectral recordings from patients with pulmonary valve insufficiency. A: Note the relatively faint signal and slow diastolic decay consistent with relatively mild insufficiency. Compare this with the denser signal with a steeper decay slope (B), which was recorded in a patient with severe pulmonary valve insufficiency. C: Image recorded in a patient with moderate pulmonary valve insufficiency and a hypertrophied noncompliant right ventricle. Note the late systolic interruption of regurgitant flow coincidental with atrial systole (arrow). This phenomenon occurs when the atrium contracts, ejecting blood into a noncompliant right ventricle. This results in presystolic flow into the right ventricular outflow tract, which interrupts the diastolic insufficiency flow. PI, pulmonary valve insufficiency.

On occasion, one notes a distinctive late diastolic notching of the pulmonary insufficiency Doppler flow profile. This notching occurs in patients in sinus rhythm and is a marker of a hypertrophied and/or otherwise noncompliant right ventricle. The origin of this notching is elevation of right atrial pressure with atrial systole which is then transmitted through the body of a noncompliant right ventricle, therefore elevating right ventricular end-diastolic pressure resulting in interruption of the regurgitant pulmonary flow (Fig. 12.18C).

FIGURE 12.19. Continuous wave Doppler image recorded in a patient with
pulmonary hypertension illustrates the manner in which pulmonary artery diastolic pressure can be calculated to be 40 mm Hg from the diastolic flow velocity and an assumed right atrial pressure of 15 mm Hg.

**Miscellaneous Abnormalities of the Pulmonary Valve**

There are rare tumors and masses that can be seen on the pulmonary valve. As with any of the four cardiac valves, infectious endocarditis can involve the pulmonary valve, although it is substantially less frequent than involvement of any of the other cardiac valves. When present, vegetations take on a similar oscillating appearance to that noted in other valve involvement. Occasionally, a fibroma or papilloma can be seen on the pulmonary valve, in which case, it takes on the typical appearance of a small spherical mass, usually attached to the leaflet by a thin stalk.

There is a clinically described phenomenon of idiopathic dilation of the pulmonary artery typically seen in elderly female patients (Fig. 12.20). This can result in marked dilation of the proximal pulmonary artery, occasionally involving both major branches and frequently results in secondary pulmonary valve regurgitation. An additional finding noted in idiopathic dilation of the pulmonary artery is high-frequency oscillation of the pulmonary valve leaflets noted best with M-mode echocardiography. Additionally, occasional isolated aneurysms of the pulmonary artery are encountered (Fig. 12.21).
FIGURE 12.20. Parasternal short-axis view recorded at the base of the heart in an elderly female with idiopathic dilation of the pulmonary artery. Note the 3.68-cm dimension of the main pulmonary artery (MPA) and the dilated right and left pulmonary arteries (RPA, LPA). There was no evidence of pulmonary hypertension or significant pulmonic valve regurgitation in this instance.

Video 12-20

Evaluation of the Right Ventricular Outflow Tract
The right ventricular outflow tract is defined as the portion of the right ventricle extending from the *crista supraventricularis* to the pulmonary artery annulus. It is a relatively trabeculated area of the right ventricle. Because of its muscular nature, diseases that elevate right ventricular pressure, such as pulmonary hypertension or pulmonary valve stenosis, result in hypertrophy in the right ventricular outflow tract. Because of its proximity to the anterior chest wall, the right ventricular outflow tract is usually easily evaluated from a parasternal short-axis view, where its dimension and degree of trabeculation and hypertrophy can frequently be easily ascertained (Fig. 12.22). Obstruction can occur in the right ventricular outflow tract as a primary abnormality such as discrete outflow tract obstruction or more commonly due to physiologic hypertrophy. Obstruction related to physiologic hypertrophy often has a dynamic component. Obstruction in the right ventricular outflow tract can result in characteristic abnormalities of pulmonary valve motion, which are often best appreciated on M-mode echocardiography. In a manner similar to the abnormalities seen on the aortic valve in discrete subvalvular stenosis, coarse fluttering of the pulmonary valve can be seen on M-mode echocardiography (Figs. 12.7 and 12.8). Other instances in which abnormalities of the right ventricular outflow tract are noted include patients in whom corrective surgery has been undertaken, in which case, either a patch or aneurysmal dilation of the outflow tract may be visualized.

**FIGURE 12.21.** Parasternal short-axis view recorded in a patient with a pulmonary artery aneurysm. In A, note the location of the right ventricular outflow tract and relatively normal dimension of the very proximal pulmonary artery, after which there is marked dilation. B is recorded with color Doppler and allow somewhat better delineation of the margins of the aneurysm which measures approximately 5.5 cm.
FIGURE 12.22. Parasternal short-axis view recorded at the papillary muscle level in a patient with pulmonary hypertension and secondary infundibular hypertrophy. Note the dilated right ventricle, the flattened ventricular septum with a small left ventricular cavity, and hypertrophy of the right ventricular infundibulum (arrow).
In adults, the most common form of tricuspid valvular pathology is secondary or functional tricuspid regurgitation due to either annular or right ventricular dilation, with subsequent malcoaptation of the leaflets. This is a common secondary finding in pulmonary hypertension or any other disease resulting in right ventricular dilation. In general, detection of tricuspid regurgitation with a dilated annulus should lead to a search for underlying causes such as pulmonary hypertension, or disease of the right ventricular myocardium such as infarction or cardiomyopathy.

Anatomically, the tricuspid valve is the most complex of the four cardiac valves. The three tricuspid valve leaflets are attached around the tricuspid annulus, which has a more variable geometry than does the relatively circular mitral valve annulus. The three leaflets are not equally sized, with the anterior (or lateral) leaflet typically being substantially larger than the septal and posterior leaflets. Typically, the septal leaflet is smaller than the other two and inserts in a more apical position compared with the anterior leaflet of the mitral valve. This relatively apical position is one of the key discriminators between the tricuspid and the mitral valves and is a reliable means of identifying the anatomic right ventricle in congenital heart disease such as “corrected” or L-transposition. Coaptation of the tricuspid valve involves interaction of all three leaflets with a variable degree of overlap of leaflet tissue at the coaptation line. Chordal attachments are to three papillary muscles arising from the ventricular septum and free wall of the right ventricle. Because of the variable size of each of the three tricuspid leaflets, it is often difficult to ascertain the independent location, size, and motion of any given tricuspid leaflet.

The tricuspid valve can be visualized from multiple transthoracic and transesophageal imaging planes (Fig. 12.23). From the parasternal transducer position, the tricuspid valve is well visualized from the right ventricular inflow tract view, obtained by medial angulation of the transducer such that the ultrasound beam is directed beneath the sternum. In this view, the right atrium and right ventricle as well as the coronary sinus and occasionally the inferior vena cava with an associated eustachian valve are clearly visualized (Fig. 12.24A). From this view, the posterior and anterior leaflets of the tricuspid valve can be clearly seen. In a parasternal short-axis view at the base of the heart, the tricuspid valve can be seen at the 9 o’clock position in relation to the aorta. In this view, the septal and anterior leaflets are
visualized (Fig. 12.24B). From an apical four-chamber view, the tricuspid valve can be visualized and its position relative to the mitral valve ascertained (Fig. 12.25). As discussed in Chapter 19, the tricuspid annulus is more apically positioned than is the mitral annulus. From an apical four-chamber view, the septal and anterior leaflets of the tricuspid valve are clearly visualized. Because the tricuspid valve is complex both in its anatomy and motion, M-mode echocardiography plays little role in identification of tricuspid valve pathology. When employed, it can demonstrate a two-phase opening pattern of the tricuspid valve, similar to the mitral valve (Fig. 12.26).

FIGURE 12.23. Schematic representation of transthoracic and transesophageal echocardiographic views illustrates the position of the tricuspid valve leaflets in each. Visualization of the anterior (A), posterior (P), and septal (S) leaflets are as noted in each figure. RAA, right atrial appendage.
FIGURE 12.24. Parasternal right ventricular inflow tract view (A) and short-axis view (B) recorded in a patient with a normal heart. In A, note the coaptation of the tricuspid leaflets. In the inset, note the very small, inconsequential, physiologic jet of tricuspid regurgitation which is commonly seen in a normal tricuspid valve. B is a short-axis view from the same patient, again depicting the closed tricuspid valve (upward-pointing arrows) and the partially open pulmonic valve (downward-pointing arrow). [Video 12-24A]

coming soon
Using transesophageal echocardiography, the tricuspid valve can be imaged from multiple imaging planes. The incremental yield of transesophageal echocardiography compared to transthoracic imaging is often less for the tricuspid valve than for the mitral valve. The tricuspid valve can be visualized in the four-chamber equivalent view from behind the left atrium (Fig. 12.27), in which case its appearance is similar to that noted for a transthoracic apical four-chamber view. It is also well visualized at the base of the heart in an 80- to 110-degree view (Fig. 12.28). From a midesophageal
transducer position, a short-axis view of the tricuspid valve can also be obtained (Fig. 12.29). The deeper gastric views in a longitudinal plane often provide superb visualization of the tricuspid valve as well (Fig. 12.30).

Visualization of the tricuspid valve with three-dimensional imaging is often technically problematic and of less clinical benefit than for the mitral valve. This is related to the less than ideal angle of interrogation, variable leaflet structure, and more variable leaflet motion. Figure 12.31 depicts a transesophageal three-dimensional image of a normal tricuspid valve in which the coaptation of three leaflets can be visualized. Figure 12.32 was recorded in a patient with severe functional tricuspid regurgitation in which the regurgitant orifice can be visualized with three-dimensional imaging.

**Doppler Evaluation of the Tricuspid Valve**

Both tricuspid valve inflow and tricuspid regurgitation can be evaluated from multiple echocardiographic windows. Because the effective orifice area of the tricuspid valve is greater than that of the mitral valve, the inflow velocities are lower than for the mitral valve at any given flow volume. As for the mitral valve, the normal pattern consists of relatively higher early inflow (E wave) and a lower velocity flow concordant with atrial systole (A wave). In the absence of significant pathology, the tricuspid valve E/A ratio typically exceeds 1.0 (Fig. 12.33). Color flow imaging can be used to document the presence of tricuspid regurgitation. In the normal disease-free state, the tricuspid valve, because of its complex closure pattern, often exhibits mild degrees of regurgitation, which may be confined to early systole (Figs. 12.24A and 12.34). This degree of functional tricuspid regurgitation is usually of no clinical relevance. The prevalence of regurgitation increases with age. When noted, the normal physiologic degrees of regurgitation typically are associated with relatively low-tricuspid regurgitation velocities, implying right ventricular systolic pressures in the normal range and with normal size of the right atrium and ventricle.
FIGURE 12.26. M-mode echocardiograms recorded in the parasternal short axis (A) and right ventricular inflow tract (B) views demonstrate normal tricuspid valve
FIGURE 12.27. Transesophageal echocardiogram recorded in a horizontal (0-degree) view from behind the LA. Note the slight apical positioning of the tricuspid valve septal leaflet (S) compared with the anterior leaflet (A) of the mitral valve. See Figure 12.22 for leaflet anatomy.

Video 12-27
FIGURE 12.28. Transesophageal echocardiogram recorded in a 110-degree view at the base of the heart. See Figure 12.23 for leaflet anatomy. A, anterior leaflet; P, posterior leaflet.

Video 12-28
FIGURE 12.29. Transesophageal echocardiogram recorded in a patient depicting a normal tricuspid valve. This image was recorded in diastole, and the anterior (A), posterior (P), and septal (S) leaflets are clearly depicted.
FIGURE 12.30. Transesophageal echocardiogram recorded at 120 degrees from the gastric probe position. The apex of the right ventricle, tricuspid valve, and a portion of the right atrium are clearly visualized. This view gives excellent visualization of the tricuspid valve chordae and papillary muscles. A, anterior leaflet; P, posterior leaflet. Video 12-30
FIGURE 12.31. Real-time transesophageal three-dimensional echocardiogram recorded at a 45-degree angle in a patient with a normal tricuspid valve. The anterior, posterior, and septal (A, P, S) leaflets are clearly visualized. In the real-time image, the complex closure pattern of the tricuspid leaflets can be better appreciated. (Video 12-31)

Video 12-31

Tricuspid Stenosis
Tricuspid stenosis is infrequently encountered. The etiologies of tricuspid stenosis include exceptionally rare cases of congenital stenosis, tricuspid stenosis due to rheumatic heart disease, in which case mitral stenosis will invariably be present, and milder degrees of stenosis in the carcinoid syndrome. The stenotic tricuspid valve has thickened leaflets with restricted motion at the level of the tips and chordae (Fig. 12.35); the transvalvular gradient can be determined from any of the available imaging planes.

**Tricuspid Regurgitation**

Unlike tricuspid stenosis, tricuspid regurgitation is common and can be due to the primary disease of the tricuspid valve or more often secondary to annular or right ventricular dilation. As noted previously, mild physiologic degrees of tricuspid regurgitation are commonly encountered in the normal disease-free individual. Etiologies of tricuspid regurgitation are listed in Table 12.1. The most common cause of tricuspid regurgitation is functional valvular regurgitation secondary to annular or right ventricular dilation, which in turn may be the result of pulmonary hypertension of any cause. Additionally, functional tricuspid regurgitation may occur in any disease causing right ventricular dilation including volume overload related to shunt or primary disease of the right ventricular myocardium. The severity of functional tricuspid regurgitation is related to the degree of apical tethering of the tricuspid leaflets. This can be quantified as tethering area or height (Fig. 12.36), which, in addition to providing mechanistic insight, may predict success of tricuspid valve repair. The severity of functional tricuspid regurgitation can range from mild to severe (Figs. 12.37 to 12.39). Over time, tricuspid regurgitation results in progressive right ventricular dilation which subsequently results in progressively more severe functional tricuspid regurgitation. Three-dimensional echocardiography can be used to directly visualize the regurgitant orifice although the success of direct visualization of the tricuspid regurgitant orifice is substantially less than for the mitral regurgitant orifice (Figs. 12.32 and 12.40).
FIGURE 12.32. Real-time transesophageal three-dimensional echocardiogram recorded in a patient with severe functional tricuspid regurgitation. The central illustration is a real-time three-dimensional image from a perspective within the right atrium, recorded at mid systole. Note the large functional tricuspid regurgitation orifice outlined by the dotted line. At the upper right is a detailed view of the right atrium, right ventricle, and tricuspid valve in a persistently open position. The lower right is the transthoracic color flow Doppler image confirming the presence of severe tricuspid regurgitation due to coaptation failure of the tricuspid leaflets.
FIGURE 12.33. Pulsed spectral Doppler recording of tricuspid inflow (upper panel) and mitral inflow for comparison (lower panel). Note the relatively lower absolute velocity of the tricuspid inflow due to the larger effective orifice area of the tricuspid valve compared with the mitral valve.
FIGURE 12.34. Apical four-chamber view recorded in a patient with mild tricuspid regurgitation. In the central illustration note the mildly dilated RA and normal RV. There is a color flow jet recorded in systole consistent with mild tricuspid regurgitation. The panel at the upper right is the continuous wave spectral Doppler of the tricuspid regurgitation jet confirming a normal velocity, suggesting absence of pulmonary hypertension. [Video]
FIGURE 12.35. Apical four-chamber view recorded in a patient with rheumatic involvement of both the mitral and tricuspid valves. In this mid-diastolic view note the doming pattern of opening of the tricuspid valve (arrows). At the upper left is a detailed view of color Doppler recorded in mid diastole through the tricuspid valve showing the relatively constrained diastolic orifice. The upper right is the spectral Doppler demonstrating a mean pressure gradient of 5 mm Hg through the stenotic tricuspid valve. [Video 12-35 CFD]
Several echocardiographic parameters play a role in surgical decision making with respect to tricuspid valve annuloplasty in functional tricuspid regurgitation. Parameters that have been evaluated as a marker of patients likely to benefit from tricuspid annuloplasty have included an annular dimension in the apical four-chamber view, as well as the tenting area of the tricuspid valve. Current recommendations suggest that an annular dimension >40 mm, measured in the apical four-chamber view, represents an indication for concurrent tricuspid annuloplasty at the time of other cardiac surgery. A major issue in determining the potential success of tricuspid annuloplasty is assessment of right ventricular function. This assessment remains problematic. Echocardiographic techniques which have been proposed for assessment of right ventricular function as it relates to successful tricuspid annuloplasty have included fractional area change, right ventricular ejection fraction, and more recently longitudinal strain of the right ventricular free wall. At this time, none of these findings have attained a level of reliability or accuracy to be considered a definitive method for decision making. MRI has also been utilized to assess right ventricular size and function in this setting with similar success and results.
FIGURE 12.36. Apical four-chamber view emphasizing the tricuspid valve anatomy in secondary, functional tricuspid regurgitation. Note the apical displacement of both the belly and the coaptation of the tricuspid valve leaflets. The “tenting area” can be calculated as the area defined by the plane of the tricuspid annulus (dashed line) and the apically displaced leaflets (shaded area). In the color flow image note the severe tricuspid regurgitation.
Video 12-36a

Video 12-36b
As with the mitral valve, tricuspid regurgitation may be a result of tricuspid valve prolapse seen in myxomatous valve syndrome (Fig. 12.41). In
most instances, this will be seen in conjunction with mitral valve prolapse. Because of the variable anatomy of the tricuspid valve leaflets, its motion both in diastole and in systole is far less predictable than that of the mitral valve, and in one or more views, the normal tricuspid valve may appear to prolapse behind the plane of its annulus in the absence of any clinically relevant disease state.

**FIGURE 12.38.** Apical four-chamber view oriented to emphasize the right heart. This image was recorded in a patient with mild pulmonary hypertension and moderate secondary tricuspid regurgitation. In the central figure notice the tricuspid regurgitation jet filling approximately one-third of the area of the dilated right atrium. At the upper left note the continuous wave Doppler spectral profile confirming a gradient of 36 mm Hg between the RV and RA, which when combined with estimated right atrial pressure suggests a right ventricular systolic pressure of 46 mm Hg.
coming soon

Video 12-38
A: Recorded from an apical transducer position is an expanded view of the RV and RA demonstrating a dilated tricuspid annulus with tethering of the tricuspid leaflets due to right ventricular dilation and dysfunction. A: Recorded in early systole, note that the tricuspid leaflets fail to coapt. B: Note the somewhat eccentric tricuspid regurgitation jet filling approximately 30% of the right atrium. Because this jet impinges on a wall, the jet size will understate the true severity of tricuspid regurgitation.
Additional causes of tricuspid regurgitation include ruptured chordae, which can occur spontaneously or on occasion as a result of blunt chest trauma. Figure 12.42 was recorded in a patient after a motor vehicle accident. There is a partial flail of the tricuspid valve secondary to chordal rupture. As with any of the other cardiac valves, involvement by endocarditis leading to perforation and/or chordal rupture can lead to tricuspid regurgitation (Fig. 12.43).
FIGURE 12.40. Real-time transesophageal three-dimensional echocardiogram of a patient with severe functional tricuspid regurgitation. This image is from the perspective of within the RA and was recorded in systole. Note the large, directly visualized regurgitant orifice (“x”).

Video 12-40
FIGURE 12.41. Transthoracic echocardiogram recorded in a patient with Marfan syndrome. Note the myxomatous changes in the tricuspid valve with pronounced bileaflet prolapse (small arrows). Incidental note is made of a prominent eustachian valve (EV) as well.
Video 12-41
FIGURE 12.42. A: A transthoracic echocardiogram recorded in an apical four-chamber view revealing highly eccentric tricuspid regurgitation. B:
Transesophageal echocardiogram recorded in a horizontal imaging plane from behind the left atrium in the same patient. Note the disruption of the septal tricuspid leaflet (arrow), which protrudes behind the plane of the annulus in systole. These images were recorded in an individual with a traumatic rupture of the tricuspid valve due to a motor vehicle accident.
FIGURE 12.43. Transthoracic echocardiogram recorded in a right ventricular inflow view in a patient with a large destructive vegetation on the tricuspid valve (arrows in upper panel). Note the severe tricuspid regurgitation in the color flow image.
Tricuspid regurgitation can occur as a result of interference by a permanent pacemaker or defibrillator lead or other catheters. It is estimated that new tricuspid regurgitation occurs in up to 20% of individuals after implantation of a permanent pacing lead. The mechanism can be either direct trauma to the tricuspid valve with leaflet perforation, partial chordal rupture, or more commonly impingement of free motion of the tricuspid leaflet by the lead as it transverses the tricuspid valve. Three-dimensional echocardiography has demonstrated that this form of tricuspid regurgitation is most common if the lead traverses the mid portion of a tricuspid leaflet and less common if the lead is positioned between two leaflets nearer leaflet insertion into the tricuspid annulus (Fig. 12.44). More chronically, by
inducing inflammation and fibrosis on the tricuspid leaflets, tissue retraction occurs and may lead to regurgitation. Generally, the degree of tricuspid regurgitation is mild but, on occasion, can be more severe and result in clinically relevant right ventricular dysfunction and symptoms of right ventricular failure. Figures 12.45 to 12.48 were recorded in individuals with tricuspid regurgitation developing after insertion of a permanent pacemaker/defibrillator lead. Often the tricuspid regurgitation is eccentric and the convergence zone of the tricuspid regurgitation jet is displaced apically at a point where leaflet mobility is constrained by the pacemaker lead.

A second pacemaker-related form of tricuspid regurgitation is that seen after extraction and/or replacement of defective or infected leads. In this instance, there may have been chronic fibrosis and attachment of tricuspid leaflet tissue to the lead. Extraction of the lead results in direct trauma to the leaflets, which may become partially flail and result in tricuspid regurgitation (Fig. 12.49).

In patients who have undergone cardiac transplantation, repeated right ventricular myocardial biopsy often results in significant tricuspid regurgitation. This is presumably due to trauma to the tricuspid valve and/or chordae tendineae. Typically, other features of a posttransplantation heart are noted including biatrial enlargement and prominent suture lines. The appearance of the disrupted tricuspid valve is similar to that noted in Figures 12.42 and 12.49.
FIGURE 12.44. Transesophageal, three-dimensional imaging of the tricuspid valve plane illustrating the potential locations of a transvenous pacemaker lead as it traverses the tricuspid plane. The lead positions at the periphery within the interleaflet commissures and those located exclusively centrally (dots) are less likely to interfere with tricuspid valve function and therefore less likely to result in significant tricuspid regurgitation. Conversely, lead positions at the middle of a leaflet (x) are more likely to result in interference with tricuspid valve function.

Ischemic Heart Disease

The right ventricle is less often involved with myocardial infarction than is the left ventricle. When right ventricular infarction occurs, it is seen almost invariably in association with an inferior infarction and is related to proximal right coronary artery occlusion. Subtle degrees of right ventricular dysfunction are not uncommon in otherwise uncomplicated inferior myocardial infarction and may be transient. Acute right ventricular ischemia may result in outward dilation of the lateral wall, thus displacing papillary muscles and resulting in tricuspid regurgitation. Similar to the relationship between myocardial infarction and mitral regurgitation, papillary muscle rupture can rarely occur as a result of an ischemic process resulting in acute, severe tricuspid regurgitation. More commonly, right ventricular infarction
results in remodeling of the right ventricular wall with apical displacement and/or scarring and fibrosis of a papillary muscle. This remodeling results in functional tricuspid regurgitation. Figure 12.50 was recorded in a patient with a right coronary artery occlusion and a limited inferior wall infarct. Note the dilation of the right ventricular lateral wall and apex, which has resulted in functional tricuspid regurgitation of mild to moderate severity. The natural history of right ventricular ischemia and inferior myocardial infarction is highly variable, and many individuals will recovery of right ventricular function with diminution of tricuspid regurgitation severity over time.
FIGURE 12.45. Apical four-chamber view recorded in a patient with a dilated cardiomyopathy and a recently implanted defibrillator. The pacing wires can be noted in A (arrow). In B, note the centrally originating, but subsequently eccentric moderate tricuspid regurgitation jet. The initial convergence zone (downward-pointing arrow) corresponds to a location of leaflet tethering by the pacemaker wire.
FIGURE 12.46. Right ventricular inflow tract views recorded in a patient with a permanent transvenous pacemaker. In A, note the dilated RA and RV and the redundant curve of the pacemaker lead (leftward-pointing arrows) which tethers
the tricuspid valve leaflets into the right ventricle. In B, note the resultant severe tricuspid regurgitation due to direct interference of the malpositioned pacemaker lead with the tricuspid apparatus.

Video 12-46A

Video 12-46B
FIGURE 12.47. Transesophageal echocardiogram of the right ventricular inflow
tract recorded at 134 degrees in a patient with pacemaker-mediated tricuspid regurgitation. In A, recorded in mid systole, note the failure of coaptation of the tricuspid leaflets (upward-pointing arrows). The pacemaker lead (rightward-pointing arrow) can be seen impeding the motion of the tricuspid leaflet. B is the accompanying color Doppler revealing severe tricuspid regurgitation through the non-coapting leaflet tips. [Video 12-47A]

Video 12-47A

coming soon

Video 12-47B

coming soon
FIGURE 12.48. Three-dimensional echocardiogram recorded in the same patient depicted in Figure 12.47. The anatomy is better appreciated in the real-time image. In the still frame, note the pacemaker lead protruding through the tricuspid orifice in systole (arrow). This has resulted in interference with normal closing motion of the tricuspid leaflet, causing a visible regurgitant orifice (x). [Video 12-48]
FIGURE 12.49. These images were recorded from transesophageal echocardiogram in a patient who developed significant tricuspid regurgitation following removal of an infected pacemaker lead. This has resulted in a flail leaflet. In A, note the protrusion of the leaflet tip behind the septal leaflet (arrow) and in B the color flow Doppler signals consistent with an eccentric tricuspid regurgitation jet of at least moderate severity (arrows). Video 12-49B
Quantitation of Tricuspid Regurgitation

Color flow Doppler imaging is used to quantify tricuspid regurgitation in a manner analogous to that for the mitral valve. Because standards for determining the severity of tricuspid regurgitation are less robust than for mitral regurgitation, the algorithms for relating jet area to severity of tricuspid regurgitation are less well developed. In clinical practice, most echocardiography laboratories rely on a qualitative assessment of tricuspid regurgitation as being minimal (within normal limits), mild, moderate, or severe. Generally, the same thresholds of jet area, indexed to the right atrial area as used for mitral regurgitation, are used for tricuspid regurgitation. Figures 12.36 to 12.39 and 12.51 are examples of color flow Doppler imaging in tricuspid regurgitation demonstrating varying degrees of severity. The same limitations and cautions that were discussed with respect to color Doppler evaluation of mitral regurgitation also apply to the evaluation of tricuspid regurgitation.

There are several anatomic findings commonly noted in the presence of significant tricuspid regurgitation, including right atrial and right ventricular dilation and detection of a right ventricular volume overload pattern. Figure 12.52 was recorded in a patient with moderate tricuspid regurgitation in whom there is right heart enlargement, and a right ventricular volume overload is apparent. Evidence of right heart dilation with a right ventricular volume overload is not specific for tricuspid regurgitation but can be noted in left-to-right atrial level shunts, pulmonary valve regurgitation, and anomalous pulmonary venous return as well. When due to tricuspid regurgitation, it implies at least moderate tricuspid regurgitation. Conversely, in the absence of evidence for right ventricular volume overload, a color Doppler signal suggesting hemodynamically significant tricuspid regurgitation is less likely to represent chronic moderate or greater regurgitation.

With persistent elevation of right heart pressure, the inferior vena cava dilates and loses its normal respiratory variation in size (Fig. 12.53). With severe tricuspid regurgitation, systolic pulsations may be noted. Additionally, retrograde systolic flow can also be detected with color flow or pulsed
Doppler imaging in patients with significant tricuspid regurgitation (Fig. 12.54).

A final marker of tricuspid regurgitation that is infrequently used and has been largely replaced by color flow Doppler imaging is the use of contrast echocardiography (Fig. 12.54). When agitated saline is injected intravenously, the contrast typically remains confined to the right atrium. Because non–contrast-enhanced blood is flowing into the atrium from the inferior vena cava, contrast is rarely present in the more inferior portion of the atrium and is not present in the inferior vena cava. Phasic (systolic) appearance of contrast in the inferior vena cava is another indirect marker of tricuspid regurgitation. As for other valve regurgitation, assessment of severity requires integrating multiple observations. Table 12.3 presents one recommended matrix for determining the severity of tricuspid regurgitation.

**FIGURE 12.50.** Four-chamber view, recorded to emphasize the right heart, in a patient with a limited inferior myocardial infarction with concurrent right ventricular infarction. In A, note the relatively normal size of the RV. This image was recorded at end systole and demonstrates dyskinetic motion of the apical right ventricular wall (upper arrow) but normal motion in the more proximal portions of the right ventricular free wall (better appreciated in the real-time image). In B recorded with color Doppler, note the moderate, functional tricuspid regurgitation. The upper left image is a spectral continuous wave Doppler demonstrating a normal right ventricle to RA pressure gradient, suggesting the absence of pulmonary hypertension.  

![Image of four-chamber view](image-url)
FIGURE 12.51. Apical four-chamber view recorded in a patient with mild to moderate tricuspid regurgitation and an otherwise normal right heart. Note the jet filling approximately 20% of the right atrial area and in the accompanying Doppler the relatively low gradient of approximately 18 mm Hg between the RV and the RA, consistent with normal pulmonary artery pressure.
As discussed in Chapter 8, the tricuspid regurgitation jet can be used to determine right ventricular systolic pressure. This is done by calculating the pressure gradient between the right ventricle and the right atrium using the modified Bernoulli equation and then adding an assumed right atrial pressure. Figure 12.55 schematizes this approach, and Figure 12.56 is an example of this application. The relationship between the gradient determined by Doppler and the gradient determined invasively in the catheterization laboratory has been demonstrated to be quite good. A major variable in determining the right ventricular systolic pressure is the method by which right atrial pressure is determined. Multiple algorithms have been proposed, each of which has provided a relatively good correlation over a broad range of pulmonary artery pressures. Some of the potential methods for determining right atrial pressure are listed in Table 12.4. Many laboratories use a floating constant of 5, 10, or 15 mm Hg, based on size of the right atrium and the severity of tricuspid regurgitation. Using this qualitative approach, when tricuspid regurgitation is mild and the right atrial size is normal, an assumed right atrial pressure of 5 mm Hg is used. For moderate degrees of tricuspid regurgitation with mild or no right atrial enlargement, an assumed constant of 10 mm Hg can be used. If tricuspid regurgitation is severe and noted in the presence of a dilated right atrium, an assumed constant of 15 mm Hg can be used. An alternate approach is to use an assumed fixed constant in all patients. Typically, either 10 or 14 mm Hg has been used. Although this approach provides excellent correlation over a broad range of right ventricular systolic pressures, it will systematically overestimate the right ventricular systolic pressure in the low ranges and potentially underestimate it in the high ranges in which the right atrial pressure could exceed 20 mm Hg. Table 12.5 outlines one scheme used to determine right atrial pressure based on a combination of echocardiographic features. It should be emphasized that this is one of many proposed schemes, and multiple different algorithms can be used with similar success.
FIGURE 12.52. Parasternal short-axis view recorded in a patient with moderate tricuspid regurgitation. Note the secondary effects on the heart from the right-side volume overload, which include a dilated right ventricle and diastolic flattening of the ventricular septum consistent with a right ventricular volume overload. Video 12-52
FIGURE 12.53. Composite illustrations of the IVC in patients with variable degrees of RA pressure elevation. A was recorded in a normal individual without evidence of significant tricuspid regurgitation or right atrial pressure elevation. The images are recorded from a subcostal imaging plane with the M-mode cursor directed through the inferior vena cava. Note the normal dimension of the inferior vena cava and the respiratory dependent narrowing of the inferior vena cava diameter with inspiration (arrows). B was recorded in a patient with a mildly dilated right atrium and mild tricuspid regurgitation. Note the mildly dilated inferior vena cava. The arrows denote intermittent sniff by the patient. Note the abrupt upward motion of the inferior vena cava and narrowing of the vena caval diameter suggesting normal vena caval and right atrial pressures. C was recorded in a patient with pulmonary hypertension, a dilated right atrium, and evidence of right ventricular volume and pressure overload. Note the dilated inferior vena cava with no apparent respiratory narrowing, suggesting elevated vena caval and right atrial pressures. D was recorded in a patient similar to that in C. In this instance, the vena cava is dilated and does not show evidence of spontaneous respiratory collapse. During a forceful sniff (arrow) notice the overall anterior motion of the entire vena cava, but no appreciable collapse, suggesting significant elevation in vena caval and right atrial pressures.
FIGURE 12.54. Subcostal echocardiograms recorded in two patients with severe tricuspid regurgitation. A: Recording with color flow Doppler imaging reveals
significant regurgitant flow into the IVC and hepatic veins. In the real-time image, note the systolic pulsation of the venous system secondary to severe tricuspid regurgitation. The small inset is a pulsed Doppler recorded from the hepatic vein in the same patient revealing systolic flow into the hepatic vein. B: Image recorded after an upper extremity intravenous contrast injection in a patient with severe tricuspid regurgitation. Notice the free reflux of the contrast into the markedly dilated inferior vena cava and hepatic veins. HV, hepatic vein.
FIGURE 12.55. Schematic representation of the method by which right ventricular systolic pressure (RVSP) can be calculated from the tricuspid regurgitation jet velocity. Using the Bernoulli equation, the pressure gradient ($\Delta P$) between the right ventricle and the right atrium is calculated as noted. Solving the equation for RVSP requires adding an assumed right atrial pressure, which can be calculated using a variety of methods (see text for details). TR, tricuspid regurgitation.

Table 12.3  ECHOCARDIOGRAPHIC AND DOPPLER PARAMETERS USED IN GRADING TRICUSPID REGURGITATION SEVERITY

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tricuspid valve</td>
<td>Usually normal</td>
<td>Normal or abnormal</td>
<td>Abnormal/flare leaflet/poor coaptation</td>
</tr>
<tr>
<td>RV/RA/IVC size</td>
<td>Normal$^a$</td>
<td>Normal or dilated</td>
<td>Usually dilated$^b$</td>
</tr>
<tr>
<td>Jet area-central jets (cm$^2$)$^c$</td>
<td>$&lt;$5</td>
<td>5–10</td>
<td>10</td>
</tr>
<tr>
<td>VC width (cm)$^d$</td>
<td>Not defined</td>
<td>Not defined, but $&lt;$0.7</td>
<td>0.7</td>
</tr>
<tr>
<td>PISA radius (cm)$^e$</td>
<td>$&lt;$0.5</td>
<td>0.6–0.9</td>
<td>0.9</td>
</tr>
<tr>
<td>Jet density and contour CW</td>
<td>Soft and parabolic</td>
<td>Dense, variable contour</td>
<td>Dense, triangular with early peaking</td>
</tr>
<tr>
<td>Hepatic vein flow$^f$</td>
<td>Systolic dominance</td>
<td>Systolic blunting</td>
<td>Systolic reversal</td>
</tr>
</tbody>
</table>
Unless there are other reasons for right atrial or right ventricular dilation. Normal two-dimensional measurements from the apical four-chamber view: right ventricular mediolateral end-diastolic dimension ≤4.3 cm; right ventricular end-diastolic area ≤35.5 cm²; normal right atrial mediolateral and superoinferior dimensions ≤4.6 cm and 4.9 cm, respectively; maximal RA volume ≤33 mL/m².

The exception is acute tricuspid regurgitation.

At a Nyquist limit of 50 to 60 cm/sec. Not valid in eccentric jets. Jet area is not recommended as the sole parameter of tricuspid regurgitation severity due to its dependence on hemodynamics and technical factors.

At a Nyquist limit of 50 to 60 cm/sec.

Baseline shift with Nyquist limit of 28 cm/sec.

Other conditions may cause systolic blunting (e.g., atrial fibrillation, elevated right atrial pressure). CW, continuous wave Doppler imaging; IVC, inferior vena cava; PISA, proximal isovelocity surface area; RA, right atrium; RV, right ventricle; VC, vena contracta.


FIGURE 12.56. Calculation of right ventricular to right atrial tricuspid regurgitation jet velocity is illustrated in a patient with moderate, functional tricuspid regurgitation related to pulmonary hypertension. The Doppler profile at the upper left was recorded from a four-chamber view and reveals a peak velocity of 4.8 m/sec corresponding to a pressure gradient of 92 mm Hg. The recording in the upper right was recorded from the RVIT and reveals a peak pressure gradient of approximately 3 m/sec and in the lower right from a slightly different angle from a four-chamber view revealing a peak velocity of just under 4 m/sec. These flow profiles illustrate the need to interrogate the tricuspid regurgitation jet from multiple windows and angles to ensure that the maximum velocity has been recorded.
MISCELLANEOUS CONDITIONS OF THE TRICUSPID VALVE

Carcinoid Heart Disease

Carcinoid heart disease occurs when a neuroendocrine-secreting tumor releases high levels of serotonin and its metabolites such as 5-hydroxytryptophan into the bloodstream. This results in an inflammatory reaction on the endothelial surface of valves. The active metabolite is deactivated in the lung parenchyma. As such, pulmonary vein blood does not contain the active metabolites, thus sparing the left-side heart structures. There are two exceptions in which the left-side valves can be involved as well. If a right-to-left shunt exists so that the active compound is not deactivated in the pulmonary bed, then left-sided involvement can occur. Similarly, if pulmonary metastases have occurred, then there will be direct release of metabolically active metabolites into the pulmonary veins and the mitral and aortic valves may be involved as well.

<table>
<thead>
<tr>
<th>Table 12.4 METHODS FOR DETERMINING RIGHT ATRIAL PRESSURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jugular vein height (clinical estimation)</td>
</tr>
<tr>
<td>Inferior vena caval appearance</td>
</tr>
</tbody>
</table>
Dilated vs. normal
Sniff plethysmography vs. visual behavior
Respiratory variation in size

Empiric constant (i.e., 10 or 14 mm Hg)
Floating constant (5, 10, 15, 20 mm Hg)
Percentage constant (10% of $\Delta P$)

$\Delta P$, right ventricular-right atrial pressure gradient from the tricuspid regurgitation jet.

**FIGURE 12.57.** A: RA inflow tract view recorded in a patient with carcinoid heart disease and involvement of the tricuspid valve. Note the diffusely thickened, immobile tricuspid leaflets (arrows) in this end-systolic frame demonstrating complete coaptation failure. B: Corresponding color flow imaging recorded in the right ventricular inflow tract view revealing severe tricuspid regurgitation due to coaptation failure of the tricuspid leaflets in carcinoid disease.
Figures 12.57 to 12.59 are examples of advanced carcinoid heart disease in which the tricuspid and the pulmonic valves have been involved. Note that the entire length of the leaflets is involved and has a rigid stiffened appearance. There is some retraction of overall leaflet length resulting in failure of coaptation and tricuspid regurgitation. Typically, pulmonary hypertension is not present, and thus the tricuspid regurgitation jet occurs at a relatively low velocity.
RAP (mm Hg) | RA Size | TR | TR $V_{\text{max}}$ | IVC
---|---|---|---|---
5 | Normal | ≤Mild | <2.5 m/sec | Normal
10 | ↑ | Moderate | 2.6–4 m/sec | Dilated
15 | ↑↑ | Severe | >4 m/sec | Dilated, no respiratory variation

RAP, estimated right atrial pressure; TR, severity of tricuspid regurgitation; TR $V_{\text{max}}$, Doppler determined peak velocity of the tricuspid regurgitation jet; ↑, mildly increased; ↑↑, moderately or severely increased.

**FIGURE 12.58.** A: Apical four-chamber view recorded in a patient with carcinoid disease and involvement of the tricuspid valve. Notice the thickened “board-like” appearance of the tricuspid leaflets (arrows). In the real-time motion, note that the leaflets are fixed in space with little motion in either diastole or systole leading to total coaptation failure. B: Corresponding color flow Doppler revealing severe tricuspid regurgitation due to coaptation failure of the thickened tricuspid leaflets.

Video 12-58A
As with unrestrained or “free” pulmonic insufficiency (previously discussed), tricuspid regurgitation occurring at a low pressure through a nonconstrained orifice can sometimes result in diagnostic confusion as a classic convergence zone, vena contracta and “jet-like” appearance of the downstream flow may not be present. Recognizing the situation of an anatomically large regurgitant orifice with essentially low-velocity free flow both in diastole and systole should allow the echocardiographer to identify this as being severe tricuspid regurgitation. An additional sign of severe, “free” tricuspid regurgitation is an abbreviated triangular-shaped Doppler tricuspid regurgitation profile (Fig. 12.60). The origin of this phenomenon is the rapid equalization of right atrial and ventricular pressures due to the severe regurgitation.
FIGURE 12.59. Transthoracic echocardiogram through the RVOT and proximal PA in a patient with carcinoid heart disease involving the pulmonic valve. The plane of the pulmonary valve leaflets are as noted by the black arrow. Notice the thickening of the cusps and the combined pulmonary stenosis and insufficiency confirmed by the spectral Doppler display in the inset.
Endocardial Fibroelastosis

Endocardial fibrosis can be seen in a variety of diseases including hypereosinophilia syndrome and tropical forms of endocardial fibroelastosis. The underlying pathology is a marked inflammatory response in the endocardium that extends to the chordae and subsequently interferes with normal valve coaptation. Typically, the leaflets will appear to be restricted and bound down toward the ventricular wall. This is often seen in association with obliteration of the right ventricular apex due to inflammatory tissue and secondary thrombosis (Fig. 12.61). The left ventricle and mitral valve are often involved in a similar fashion.

Ebstein Anomaly

Ebstein anomaly is a congenital abnormality of the tricuspid valve in which there is apical displacement of the septal leaflet as well as tethering of the lateral leaflet to the ventricular wall. This results in coaptation of the tricuspid leaflets in a position displaced toward the right ventricular apex and creates an atrialized portion of the right ventricle. The degree of displacement can be highly variable and can range from as little as 12 mm to several centimeters. Ebstein anomaly is discussed in more detail in Chapter 19. When initially scanning in a parasternal long-axis view, the first clue to the presence of Ebstein anomaly may be the dilated right ventricle with visualization of tricuspid valve tissue in what would have anticipated to be the right ventricular outflow tract (Fig. 12.62). The apical four-chamber view is typically diagnostic and will demonstrate apical displacement of the septal leaflet as well as tethering of the lateral leaflet resulting in a coaptation point of the tricuspid leaflets well into the body of the right ventricle. On occasion, if the actual leaflet coaptation cannot be identified, color Doppler demonstrating tricuspid regurgitation with an apically displaced convergence zone provides another clue as to the presence of Ebstein anomaly (Fig. 12.63).
FIGURE 12.60. Continuous wave Doppler of tricuspid flow in an apical four-chamber view. Note the triangular-shaped tricuspid regurgitation jet without evidence of significant pulmonary hypertension. The severe tricuspid regurgitation results in diminution of regurgitant flow in later systole resulting in the triangular shape.
FIGURE 12.61. Subcostal short-axis image recorded in a patient with eosinophilic heart disease with involvement of the right ventricle and base of the tricuspid valve. In the central image note the marked thickening (linear measurement 2.77 cm) just distal to the tricuspid annulus which has immobilized the base of the lateral tricuspid valve leaflet. The inset figure is a cardiac MRI from the same patient demonstrating apical obliteration (arrows) by eosinophilic infiltration/thrombus. [Video 12-61]
FIGURE 12.62. Parasternal long-axis view recorded in a patient with Ebstein anomaly of the tricuspid valve. Note that in this view the tricuspid valve (arrows) can be clearly visualized within the right ventricular outflow tract. This is related to the apical displacement of the tricuspid valve within the right ventricle.

Video 12-62
FIGURE 12.63. Apical four-chamber view recorded in the same patient presented in Figure 12.62. In this apical view note the apical displacement of the septal tricuspid leaflet and the tethering of the lateral leaflet to the RV wall. The displacement of the septal leaflet from the annulus is denoted by the double-headed arrow and has resulted in a large atrialized portion of the right ventricle (AtRV). The side panel at the upper right was recorded with color flow Doppler and reveals moderate tricuspid regurgitation. Note that the origin of the tricuspid regurgitation is apically displaced (arrow).
Tricuspid Valve Resection

As a treatment for bacterial endocarditis, a number of patients in the late 1970s and early 1980s underwent removal of the tricuspid valve leaflets. This resulted in free tricuspid regurgitation. Many of these patients have presented 15 to 20 years later with evidence of significant right heart failure. Figures 12.64 to 12.66 were recorded in a patient who had undergone resection of a tricuspid valve leaflet for bacterial endocarditis approximately 25 years before this echocardiogram. Note the absence of any tricuspid valve tissue. By definition, wide-open tricuspid regurgitation is present. Because of the complete absence of tricuspid valve tissue, there is no organized flow from the right ventricle to the right atrium and no increase in velocity or organized jet. This results in the absence of a convergence zone, which is usually seen with organized regurgitant flow, and in low velocities of the regurgitant jet. On occasion, this entity is encountered, and because of the absence of typical findings of regurgitation, the severity of tricuspid regurgitation is not appreciated. Recognition of the marked dilation of the right heart and absence of tricuspid valve tissue should alert the sonographer and clinician to the presence of this situation.
FIGURE 12.64. Parasternal short-axis echocardiogram recorded in a patient who previously had undergone resection of a tricuspid leaflet for bacterial endocarditis. A: Note the marked dilation of the RA and RV and the absence of visible tricuspid valve tissue. B: In the color Doppler image, note the free tricuspid regurgitation with the absence of any organized jet. On occasion, the absence of a true jet with a vena contracta and convergence zone may result in the true severity of the tricuspid regurgitation not being appreciated. The spectral Doppler (upper left) reveals absence of any organized systolic or diastolic flow profiles. [Video 12-64A]
coming soon

Video 12-64B
FIGURE 12.65. M-mode echocardiogram recorded through the inferior vena cava from a subcostal transducer position in the patient presented in Figure 12.64. Note the markedly dilated IVC. Also note the phasic pulsatility and expansion of the vena cava with ventricular systole related to the severe tricuspid regurgitation. The lower panel is the pulsed wave Doppler recorded from within the hepatic vein demonstrating systolic reversal of flow in the hepatic vein, consistent with severe tricuspid regurgitation.
FIGURE 12.66. Subcostal two-dimensional image recorded in the same patient presented in Figures 12.64 and 12.65. In the central illustration, recorded with color flow Doppler at mid systole note the robust flow into the IVC and hepatic vein (HV) related to severe tricuspid regurgitation.

Video 12-66

Tumors and Other Masses

Rarely, a primary tumor can arise on the tricuspid valve. Tumors that have
been reported on the tricuspid valve have included very infrequent myxomas and occasional fibroelastoma. When present, they have the same appearance as these atypical tumors do on the mitral valve.

**Suggested Readings**

**General**


**Tricuspid Regurgitation/Hemodynamics**


**Specific Clinical Diseases**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Chapter 13
Infective Endocarditis

Infective endocarditis remains a challenging and often fatal condition. One reason for this is the difficulty of establishing an accurate diagnosis, particularly early in the course of the disease when proper management can be lifesaving. As therapeutic approaches have become more successful, the importance of early and accurate diagnosis is self-evident. Unfortunately, no single test or finding establishes the diagnosis in all cases. Instead, a constellation of findings that constitutes the diagnostic criteria continues to evolve.

The central role that echocardiography plays in the diagnosis of endocarditis began in the early 1970s with the echocardiographic demonstration of a valvular vegetation by the M-mode technique. With the advent of two-dimensional and Doppler modalities, echocardiography has become virtually indispensable in the diagnosis and management of these patients. Today, echocardiographic findings are a central part of the diagnostic criteria for infective endocarditis.

CLINICAL PERSPECTIVE

Despite improvements in therapy, infective endocarditis remains a potentially lethal disease with an incidence of 4 to 8/100,000 patient-years. Although the overall incidence has not increased appreciably over time, several factors have contributed to substantial recent changes in the epidemiology of the disease. For example, *Staphylococcus aureus* is now the most common cause of endocarditis in most series, in part due to an aging population and the increasing prevalence of intracardiac devices, including prosthetic valves, conduits, pacing wires, and indwelling catheters. This has led to the concept
of “healthcare contact” as a recognized risk factor for the development of infective endocarditis. Currently, approximately 25% of infective endocarditis cases in this country are attributable to a previous medical event or procedure, such as implantation of a prosthetic valve or pacemaker. More recently, the opioid epidemic that has plagued the United States has been associated with a striking increase in the incidence of drug-dependence–associated endocarditis. Such patients appear to have a particularly poor prognosis, in part due to the likelihood of recurrent infection following valve replacement therapy.

Infective endocarditis is defined as a localized infection anywhere on the endocardium, including the chamber walls, vessels, and within congenital defects. The vast majority of vegetations, however, occur on valve leaflets. Infection may also develop on any implanted or prosthetic material such as prosthetic valves, conduits, pacing electrodes, and catheters. The process of developing endocarditis occurs in the setting of bacteremia or fungemia. The initiating event usually requires the presence of a high-velocity jet, which may be due to a congenital anomaly such as a ventricular septal defect, a regurgitant valve, or a prosthetic valve. It is thought that the jet interferes with the protective endothelial surface, allowing the blood-borne pathogens to adhere and coalesce. As the nidus of infection organizes, masses of microorganisms attract platelets, fibrin, and other materials and become adherent to the endothelial surface to form a vegetation. The vegetation will grow in size, either as a sessile clump or as a highly mobile and even pedunculated mass with the potential for embolization. As the hallmark of endocarditis, the ability to detect the vegetation is the focal point of diagnosis. This sequence of events offers a mechanism for development of endocarditis in patients with underlying heart disease. However, since as many as 50% of patients who get endocarditis do not have lesions associated with a high-velocity jet, some other set of conditions must be operational in these patients to explain the link between bacteremia and cardiac involvement.

ECHOCARDIOGRAPHIC CHARACTERISTICS OF VEGETATION
The versatility of echocardiography in the evaluation of endocarditis is illustrated in Table 13.1. Among its important functions is the identification of underlying heart disease known to increase a patient’s risk of infection. Although the absence of underlying disease does not confer protection against endocarditis, particular conditions, such as congenital heart disease, bicuspid aortic valve, and a myxomatous mitral valve, are known risk factors. At the same time, these conditions often confound the diagnosis of endocarditis by creating abnormalities that mimic or conceal echocardiographic evidence of infection.

An essential first step in the echocardiographic evaluation is to search for evidence of acute ongoing infection. Although there are several manifestations of endocarditis, including abscesses and fistulae, the most common and direct evidence of infective endocarditis is the vegetation. Because a vegetation begins as a microscopic focus of infection and gradually grows into a conspicuous mass, its presence may or may not be evident on an imaging study. Thus, echocardiography must be sensitive enough to detect the vegetation and specific enough to distinguish it from other echocardiographic abnormalities or artifacts. As can be seen in Table 13.2, certain echocardiographic features can be used to either increase or decrease the probability that a visualized mass is due to endocarditis. A vegetation is typically irregularly shaped, highly mobile, and attached to the free edge of a valve leaflet. They tend to develop on the upstream side of the valve, that is, the ventricular side of the aortic valve and the atrial side of the mitral valve (Fig. 13.1). Vegetations may be sessile or pedunculated but usually have motion that is independent of the valve itself. Figure 13.2 is an example of endocarditis involving the tricuspid valve in a patient with a history of intravenous drug use. The infectious process can be seen encasing the valve leaflets and chordae. Severe tricuspid regurgitation was present. Because vegetations often occur in the path of a high-velocity jet, their motion is frequently described as oscillating or fluttering. The presence of significant mobility, or oscillating motion, is a classic feature of most vegetations. In fact, the absence of mobility argues against the diagnosis and should suggest the possibility of an alternative diagnosis, including a healed vegetation. The shape and size of vegetations are quite variable and may either increase (due to progression of disease) or decrease (due to healing or embolization) over time (Fig. 13.3). Fungal vegetations tend to be larger than
those caused by bacterial infections, and those involving the tricuspid valve tend to be larger compared with vegetations that affect the aortic or mitral valve (Fig. 13.4).

Table 13.1  COMPREHENSIVE ROLE OF ECHOCARDIOGRAPHY IN PATIENTS WITH ENDOCARDITIS

<table>
<thead>
<tr>
<th>Initial/early role:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Identifies predisposing heart disease</td>
</tr>
<tr>
<td>Early assessment in all cases of suspected IE</td>
</tr>
<tr>
<td>Detects complications</td>
</tr>
<tr>
<td>Assesses hemodynamic consequences</td>
</tr>
<tr>
<td>Serial evaluation (assesses efficacy of therapy)</td>
</tr>
<tr>
<td>Intraoperative assessment of extent of disease</td>
</tr>
<tr>
<td>Prognosis (risk of complications)</td>
</tr>
<tr>
<td>Establishes new baseline after therapy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Repeat/follow-up role:</th>
</tr>
</thead>
<tbody>
<tr>
<td>TEE (after positive TTE) in patients at high risk for complications</td>
</tr>
<tr>
<td>Repeat TEE (after negative initial TEE) if clinical suspicion persists</td>
</tr>
<tr>
<td>Repeat TEE if suboptimal course during therapy (e.g., clinical deterioration, persistently positive blood cultures, worsening physical examination)</td>
</tr>
</tbody>
</table>

FIGURE 13.1. An example of a large, mobile vegetation on the aortic valve that fills the left ventricular outflow tract, seen from the parasternal long-axis (A) and the apical views (B).
<table>
<thead>
<tr>
<th>Positive Feature</th>
<th>Negative Feature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low reflectance</td>
<td>High echogenicity</td>
</tr>
<tr>
<td>Attached to valve, upstream side</td>
<td>Nonvalvular location</td>
</tr>
<tr>
<td>Irregular shape, amorphous</td>
<td>Smooth surface or fibrillar</td>
</tr>
<tr>
<td>Mobile, oscillating</td>
<td>Nonmobile</td>
</tr>
<tr>
<td>Associated tissue changes, valvular regurgitation</td>
<td>Absence of regurgitation</td>
</tr>
</tbody>
</table>
Although typically attached to a valve, vegetations may also attach to chordae, chamber walls, or any foreign body, such as a pacemaker lead, indwelling catheter, or prosthetic valve sewing ring. Figure 13.5 is an example of endocarditis involving a porcine tricuspid valve as well as the pacing wire that extends through it. The mass itself is typically homogeneous with echogenicity similar to that of the myocardium. However, vegetations can occasionally be cystic or appear more dense and calcified. The infectious process often alters valve structure and function. As a result, some degree of regurgitation is associated with most cases of acute endocarditis. In Figure 13.6, a patient with an aortic valve vegetation is shown. The involvement is extensive, and the valve is partially flail. There is severe aortic regurgitation. A patient with significant mitral regurgitation associated with an extensive aortic valve vegetation is shown in Figure 13.7. Despite the presence of severe mitral regurgitation, neither a vegetation nor leaflet perforation were demonstrated. Figure 13.8 shows a patient with intravenous drug use who presented early in the course of their endocarditis. Small vegetations were noted on the aortic, mitral, and tricuspid valves. These were not fully appreciated on transthoracic imaging.

**FIGURE 13.2.** Extensive infection involving the right heart from a patient with a history of intravenous drug use. The vegetation involves the tricuspid valve.
(arrow) and chordae (arrowhead).}

**Video 13-2**

**FIGURE 13.3.** A: An example of disease progression from a patient with positive blood cultures, mild aortic regurgitation, and a sclerotic aortic valve but no definite vegetation (day 1). B: Two weeks later (day 14), a small vegetation is seen on the valve (arrow). C: By day 22, the vegetation has increased in size (arrow) and there is severe aortic regurgitation, despite antibiotic therapy.
FIGURE 13.4. A very large fungal vegetation in an immunocompromised patient is shown. Extensive involvement of the mitral valve is demonstrated in the apical long-axis (A) and four-chamber (C) views. In B, the dimensions of the mass are recorded.
FIGURE 13.5. Transesophageal echocardiography shows a large mass (large arrow) attached to a pacemaker lead, most likely an infected thrombus. There are also multiple small vegetations (small arrows) attached to the pacemaker wire in the right atrium.
If the process results in destruction of underlying tissue leading to a flail or perforated valve structure, the degree of regurgitation will be severe. For example, if the infection leads to disruption of the aortic valve, severe aortic regurgitation will ensue. This is demonstrated in Figure 13.9, taken from a patient with a severely disrupted aortic valve in the setting of staphylococcal endocarditis. Figure 13.10 is an example of a small perforation of the noncoronary cusp of the aortic valve due to infection. Mild aortic regurgitation was present, but no definite vegetation was identified. Another example of a perforated valve due to *S. aureus* infection is shown in Figure 13.11. This patient had a large, highly mobile aortic valve vegetation. A perforation at the base of the anterior mitral leaflet is also demonstrated, along with severe mitral regurgitation. Much less often, a large vegetation will obstruct the valve orifice, leading to a functional form of valve stenosis (Fig. 13.12).

Although most vegetations involve the valves, in some cases the infection may extend to other structures, such as the chamber wall. Figure 13.13 shows an unusual vegetation attached to the posterior wall of the aortic root. Multiple vegetations involving the aortic valve are also present.

It should be emphasized that there is no single characteristic on the echocardiogram that will conclusively identify a mass as a vegetation. The ability to detect a vegetation depends on vegetation size, location, the presence of underlying heart disease, image quality, and instrument settings. All available echocardiographic windows should be used, and Doppler flow
mapping should be performed to identify any associated valvular regurgitation. Often nonstandard imaging planes are needed for detection, especially for smaller vegetations. Although masses as small as 2 mm have been reported, in most cases, a vegetation must be at least 3 to 6 mm in size to be reliably seen. Image quality will also influence our ability to visualize small structures. As is discussed later, these are areas in which the advantages of transesophageal echocardiography have been demonstrated.

To avoid false-positive results, vegetations must be differentiated from other echo-producing abnormalities, such as myxomatous processes, degenerative changes (including Lambl excrescences and calcification), tumors, thrombi, and imaging artifacts. Figure 13.14 is taken from a patient who was asymptomatic. The large mitral valve mass could easily be mistaken for a vegetation. However, the absence of clinical signs of infection suggests an alternative diagnosis. In this case, the mass was a blood cyst. Underlying heart disease both obscures the presence of a vegetation and increases the likelihood of false-positive findings through misinterpretation. Figure 13.15 is from a patient with mitral valve prolapse who had fever due to a viral syndrome. The prolapsing scallop was incorrectly interpreted as a vegetation. The correct diagnosis was established based on absence of signs of infection and direct comparison of the current echocardiogram to a prior study. Nonbacterial thrombotic endocarditis (Libman–Sacks) is also easily confused with infective endocarditis. In Figure 13.16, two examples of nonbacterial thrombotic endocarditis are shown. In both cases, nodular masses on present on the free edge of the mitral leaflets that could easily be misinterpreted as bacterial vegetations. Distinguishing between these two conditions can be very difficult and must rely on clinical correlation.

Thus, the accuracy of echocardiography is greater in patients without underlying valve disease. Furthermore, active vegetations must be differentiated from old or healed vegetations. Some studies have suggested that vegetations tend to become smaller and more circumscribed and echogenic over time as part of the healing process (Fig. 13.17). Although this is generally true, a reduction in vegetation size might also suggest embolization. Thus, distinguishing active from healed vegetations can never rely on echocardiography alone but must take into account clinical factors.
FIGURE 13.6. Transesophageal echocardiography demonstrates a vegetation (arrow) on a partially disrupted aortic valve (A). In B, severe aortic regurgitation is present (arrow). Video 13-6a

coming soon

Video 13-6b
FIGURE 13.7. Panels A and B demonstrate extensive and diffuse involvement of the aortic valve (arrows). Although a mitral valve vegetation was not seen, Doppler shows severe mitral regurgitation (arrows, C). [Video 13-7a]
coming soon

Video 13-7b

coming soon

Video 13-7c
FIGURE 13.8. Multi-valve involvement of *Staphylococcus aureus* endocarditis is shown. Transesophageal echocardiography demonstrated small vegetations (arrows) on the aortic (A), mitral (B), and tricuspid valves (C). Video 13-8a coming soon
FIGURE 13.9. Another example of *Staphylococcus aureus* endocarditis is provided. A shows a prolapsing or disrupted aortic cusp (arrow). In B, a vegetation on the aortic valve is apparent (arrows). Color (C) and continuous-wave Doppler (D) demonstrate severe aortic regurgitation. Note the steep slope of the spectral Doppler signal (arrows).
FIGURE 13.10. A transesophageal echocardiogram demonstrates a small perforation of the noncoronary cusp of the aortic valve. **A:** Focal thickening is seen but no definite vegetation. **B:** Color Doppler imaging demonstrates the jet extending through the cusp (arrows). **C:** A short-axis view confirms the location of the perforation (arrow).
FIGURE 13.11. A large aortic valve vegetation (A, arrow) and a perforated anterior mitral valve leaflet (B, arrow) from a patient with intravenous drug use. In C, severe mitral regurgitation is demonstrated with color Doppler (arrows). [Video 13-11a]
FIGURE 13.12. A: A large vegetation involving the anterior mitral leaflet (arrows). B: Spectral Doppler imaging recorded a 10 mm Hg mean gradient across the mitral valve.
Numerous clinical studies have tested the accuracy of echocardiography to
detect vegetations and other manifestations of acute endocarditis. A limitation of all these studies is the difficulty in defining the standard by which the diagnosis is established. In most series, a clinical standard for diagnosis was used that incorporated clinical findings, blood culture results, response to therapy, and outcome measures. Although practical, this approach has obvious limitations and very likely permitted the inclusion of some patients who had bacteremia but never had endocarditis. More rigorous diagnostic standards that required pathologic and/or surgical confirmation must, by definition, exclude patients who have endocarditis but never come to either surgery nor autopsy. As a result, only the “sickest of the sick” would be included in such series. Finally, the recognition over time of the fundamental involvement of echocardiography in establishing a diagnosis made it increasingly difficult to “test the test.” That is, it becomes impossible to establish the accuracy of a test (in this case, echocardiography) that is fundamentally involved in the definition of disease. For all these reasons, the exact sensitivity and specificity of the various echocardiographic techniques must be interpreted in context. Despite these limitations, the overall utility of echocardiography as an integral part of the diagnostic algorithm is well established.

FIGURE 13.14. An example of a blood cyst (arrow) is demonstrated within the mitral valve. Diastolic (A) and systolic (B) frames are shown. Such an appearance could easily be confused with vegetation.
FIGURE 13.15. This echocardiogram was recorded from a patient with mitral valve prolapse and significant mitral regurgitation. The mitral valve was myxomatous and partially flail. A: The prolapsing valve is indicated by the arrows. B: Severe mitral regurgitation is demonstrated (arrow). C: A transesophageal echocardiogram demonstrates the prolapsing scallop (arrows). This could easily be mistaken for a vegetation. 📣
FIGURE 13.16. Two examples of nonbacterial thrombotic endocarditis are shown. In A, from a patient with metastatic cancer, masses on the tips of the mitral leaflets are noted (arrows). B is from a patient with systemic lupus, and demonstrates a nodular mass on the free edge of the anterior mitral leaflet (arrow).
Numerous studies have examined the accuracy of echocardiography for the detection of manifestations of endocarditis. The overall sensitivity of the transthoracic technique to detect vegetations has generally ranged from 60% to 70%. However, most of these studies were performed over 20 years ago, using equipment that would be considered substandard by today’s criteria. Among the important determinants of sensitivity are vegetation size and image quality. Transthoracic echocardiography is also limited in its ability to detect some of the other manifestations of endocarditis, such as abscess formation. It should be recognized that some patients with endocarditis may not have vegetations, thereby accounting for some false-negative results.

Establishing the specificity of the technique is more difficult. Although the reported false-positive rate is quite low in most series, specificity will vary widely depending on the population being studied and the criteria used to define disease. As previously discussed, in the absence of clinical data, distinguishing active vegetations from healed vegetations, myxomatous change, or tumors is nearly impossible. As a result, echocardiography is interpreted in context, thereby avoiding most false-positive results.

Beginning in the mid-1980s, the potential advantages of transesophageal echocardiography in assessing patients with suspected endocarditis were first recognized. In virtually every published series, the sensitivity of transesophageal echocardiography is consistently higher than that of the transthoracic technique. The improved image quality and the closer proximity between transducer and valves account for much of this difference. Smaller vegetations, those associated with prosthetic valves, and those in locations that would be shadowed or obscured during transthoracic scanning are some of the areas in which the transesophageal approach is superior.

When the two echocardiographic techniques are compared in the same patient population, the superior sensitivity of transesophageal imaging has been a consistent finding (Fig. 13.18). At the same time, many of these contemporary series have reported a sensitivity of transthoracic echocardiography that is lower than would be otherwise expected. This may be partly explained by the availability of transesophageal imaging. If the transthoracic examination is approached with less determination and rigor, small lesions may be missed, thereby contributing to the wide difference in
sensitivity between the two tests. Although the superiority of the transesophageal approach is beyond question, the magnitude of the difference (i.e., the surprisingly low sensitivity of transthoracic echocardiography) is noteworthy. Some of this may be explained on the basis of patient selection that included a greater percentage of individuals with a relatively low pretest likelihood of disease. Alternatively, the availability of transesophageal echocardiography may have indirectly contributed to the performance of a more cursory and less rigorous transthoracic study, followed by a thorough and complete transesophageal examination. An additional advantage of transesophageal echocardiography is its ability to identify other manifestations of endocarditis, such as annular abscesses and fistulae (Fig. 13.19). Despite the relatively modest sensitivity of transthoracic echocardiography, a normal study in the presence of acceptable image quality has high negative predictive value and is strong evidence against endocarditis, even in the setting of S. aureus bacteremia.

FIGURE 13.17. An unusual vegetation involving the mitral valve. Transesophageal two-chamber (A) and and four-chamber (B) views are shown. The patient had developed endocarditis 1 year ago which was effectively treated with antibiotics. The infection recurred with fever and positive blood cultures. A large, partially calcified mass (arrows) is seen on the mitral valve.
The value of three-dimensional echocardiography in this area continues to grow. Figure 13.20 is an example of a large vegetation on a bioprosthetic tricuspid valve recorded with three-dimensional imaging. In real-time, the mass can be seen prolapsing into the right atrium during systole. Figure 13.21 is an example of a perforated aortic valve, with severe aortic regurgitation, clearly visualized with en face three-dimensional echocardiography. In theory, the ability of three-dimensional echocardiography to visualize an entire valve (rather than individual slices of the valve) should improve sensitivity by reducing false-negative results. Unfortunately, most missed echocardiographic diagnoses are related to image quality, which would also negatively affect three-dimensional images. That is, if a vegetation is missed...
on two-dimensional imaging because of poor image quality, it may not be seen on three-dimensional imaging for the same reason.

FIGURE 13.18. Transthoracic and transesophageal echocardiograms from a patient with clinical signs of endocarditis. The transthoracic study (A) revealed moderate aortic regurgitation but no evidence of a vegetation. With transesophageal echocardiography, a perforated aortic cusp is demonstrated (B, arrow), but no vegetation. Moderate aortic regurgitation was present on color Doppler (C, arrows).
Video 13-18

Image quality is generally not a problem with transesophageal echocardiography, but the high accuracy of two-dimensional transesophageal echocardiography will make it difficult for three-dimensional transesophageal echocardiography to demonstrate incremental value. One potential advantage of three-dimensional imaging is the opportunity to obtain a complete visualization of complex cases and provide true spatial assessment of the extent of disease. Much more experience in this area can be expected over the next several years.

MULTIMODALITY IMAGING

Both MRI and CT have shown promise for the evaluation of patients with infective endocarditis. The spatial resolution and inherent three-dimensional nature of these techniques account for their ability to visualize vegetations and other manifestations of the disease (Fig. 13.22). However, for the foreseeable future, echocardiography will remain the primary imaging modality for most situations. Situations where CT and MRI may play a role include cases where there is uncertainty about the diagnosis (“possible” infective endocarditis by the Duke criteria). For example, when there is discrepancy between the echocardiographic and blood culture results, the newer imaging modalities may help confirm or reject the diagnosis. They may also be useful in the assessment of complex and/or advanced cases, involving multiple valves, or to detect cerebral embolic events, which are often clinically silent.

There is also considerable interest in the use of $^{18}$F-fluorodeoxyglucose PET/CT for the assessment of patients with endocarditis, especially involving prosthetic valves and implanted devices such as pacemaker wires. The technique creates a whole body scan and provides a sensitive means to localize active areas of inflammation. Like MRI, it may be useful to detect and localize peripheral embolization which can occur anywhere in the body.
FIGURE 13.19. Transesophageal echocardiography from a patient with a mechanical aortic valve and positive blood cultures. In A, a periaortic abscess is indicated by the arrows. In B, color Doppler demonstrates both tricuspid regurgitation (small arrowheads) and a fistula between the aortic root and right atrium (large arrows).
Although there is considerable interest in these newer modalities for the assessment of patients with endocarditis, they should not be regarded as an alternative to echocardiography and there are to date no clear guidelines on their appropriate use in this setting.

EVOLUTION OF THE DIAGNOSTIC CRITERIA

The clinical diagnosis of infective endocarditis has always been challenging. Before the routine use of echocardiography, establishing the diagnosis of endocarditis focused on evidence of ongoing infection within the blood coupled with clinical evidence of cardiac involvement. In 1994, the Duke Endocarditis Service published new criteria for the diagnosis of endocarditis that relied heavily on echocardiographic findings. In this original study, 405 cases were retrospectively reviewed and classified as definite, possible, or rejected on the basis of the presence or absence of major and minor criteria. When compared with previously used criteria, the newly proposed Duke criteria classified significantly more cases as definite endocarditis. Among pathologically proven cases, the Duke criteria were significantly more sensitive (80%) compared with the von Reyn criteria (51%).
FIGURE 13.20. A three-dimensional transesophageal echocardiogram showing a very large vegetation (arrows) on a bioprosthetic tricuspid valve, from the perspective of the right atrium.
Video 13-20
**FIGURE 13.21.** A perforation in a native aortic valve is demonstrated with three-dimensional (A) and two-dimensional (B) echocardiography (see arrows). Moderate aortic regurgitation is present (B, right).

**Video 13-21b**

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**Table 13.3**  
**DEFINITION OF TERMS USED IN THE DUKE CRITERIA**

<table>
<thead>
<tr>
<th>Major criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Blood culture positive for IE</td>
</tr>
<tr>
<td>Typical microorganisms consistent with IE from two separate blood cultures: Viridans streptococci, Streptococcus bovis, HACEK group, Staphylococcus aureus; or Community-acquired enterococci, in the absence of a primary focus; or Microorganisms consistent with IE from persistently positive blood cultures, defined as follows: At least two positive cultures of blood samples drawn &gt;12 hr apart; or All three or a majority of ≥4 separate cultures of blood (with first and last sample drawn at least 1 hr apart) Single positive blood culture for Coxiella burnetii or antiphase I IgG antibody titer &gt;1:800</td>
</tr>
<tr>
<td>(2) Evidence of endocardial involvement</td>
</tr>
<tr>
<td>(3) Echocardiogram positive for IE, defined as follows: Oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomic explanation; or Abscess; or New partial dehiscence of prosthetic valve</td>
</tr>
<tr>
<td>(4) New valvular regurgitation (worsening or changing of pre-existing murmur not sufficient)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Minor criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Predisposition, predisposing heart condition, or injection drug use</td>
</tr>
<tr>
<td>(2) Fever, temperature &gt;38°C</td>
</tr>
<tr>
<td>(3) Vascular phenomena, major arterial emboli, septic pulmonary infarcts, mycotic</td>
</tr>
</tbody>
</table>
ANEURYSM, INTRACRANIAL HEMORRHAGE, CONJUNCTIVAL HEMORRHAGES, AND JANeway LESIONS

4) IMMUNOLOGIC PHENOMENA: glomerulonephritis, Osler nodes, Roth spots, and rheumatoid factor

5) MICROBIOLOGIC EVIDENCE: positive blood culture but does not meet a major criterion as noted above\(^a\), or serologic evidence of active infection with organism consistent with IE

6) ECHOCARDIOGRAPHIC MINOR CRITERIA ELIMINATED


**FIGURE 13.22.** A cardiac CT image from the basal short-axis view from a patient with a bioprosthetic aortic valve is shown. A large perivalvular abscess is indicated by the arrows.

Although the original criteria were generally accepted as an important advance in the diagnosis of endocarditis, there were limitations that were addressed in a subsequent publication (Li et al., 2000). **Table 13.3** contains
the detailed description of terms used to define major and minor criteria, according to the updated modifications. Using these terms, the diagnosis of endocarditis can be confirmed or rejected as described in Table 13.4. On the basis of the four major and five minor criteria, patients can be classified as having definite evidence of endocarditis, possible endocarditis, or the diagnosis can be rejected. This approach has subsequently been validated in numerous studies involving a range of patient populations and endorsed by the American College of Cardiology/American Heart Association practice guidelines for the management of patients with valvular heart disease (Bonow et al., 2006).

The subsequent revision of the Duke criteria in 2000 included several updates. Most importantly, S. aureus bacteremia was defined as a major criterion, whether nosocomial or community acquired. In recognition of the challenges in identifying culture-negative endocarditis, Q-fever serology was also added as a major criterion. However, the widespread use of antibiotics in the community remains a source of false-negative blood cultures and will therefore continue to be a challenge in the diagnosis of endocarditis. Finally, the minor criterion of “echo consistent with IE but not meeting major criterion,” which had been invoked in cases of nonspecific valve thickening, was eliminated. The enhancement in sensitivity provided by the revised Duke criteria is achieved without a significant loss in specificity. A key result was that many fewer cases of “possible” endocarditis occurred and more definitive diagnoses were achieved. Although more difficult to test, most series have concluded that specificity is maintained and has been reported to be as high as 99%. This is primarily attributable to the inclusion of specific echocardiographic findings.

The value of this approach is now well established. In addition to providing a more sensitive means to establish the diagnosis of endocarditis, the Duke criteria have emphasized the essential relationship between clinical and echocardiographic findings. Despite the well-recognized importance of echocardiography in the evaluation of these patients, both false-positive and false-negative results may occur, underscoring the need to incorporate other (i.e., clinical) criteria. Furthermore, the inclusion of echocardiographic criteria has provided an impetus to standardize the various criteria used to define the essential pathologic processes, including vegetations and abscesses. These will be discussed subsequently.
### Table 13.4  
Clinical Definition of Infective Endocarditis According to the Duke Criteria

<table>
<thead>
<tr>
<th>Definite infective endocarditis (clinical criteria)</th>
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</tr>
</thead>
<tbody>
<tr>
<td>(1) Two major criteria, or</td>
<td></td>
</tr>
<tr>
<td>(2) One major criterion and three minor criteria, or</td>
<td></td>
</tr>
<tr>
<td>(3) Five minor criteria</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Possible infective endocarditis</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) One major criterion and one minor criterion, or</td>
<td></td>
</tr>
<tr>
<td>(2) Three minor criteria</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Rejected</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Firm alternate diagnosis explaining evidence of infective endocarditis, or</td>
<td></td>
</tr>
<tr>
<td>(2) Resolution of infective endocarditis syndrome with antibiotic therapy for ≤4 days, or</td>
<td></td>
</tr>
<tr>
<td>(3) No pathologic evidence of infective endocarditis at surgery or autopsy, with antibiotic therapy for ≤4 days, or</td>
<td></td>
</tr>
<tr>
<td>(4) Does not meet criteria for possible infective endocarditis, as above</td>
<td></td>
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</table>

*See Table 13.3 for definitions of major and minor criteria.


### COMPLICATIONS OF ENDOCARDITIS

A variety of complications may occur in the setting of active endocarditis that may affect outcome and alter management (*Table 13.5*). The vegetation itself is an important source of possible complications in the setting of endocarditis. Infection of the valve can lead to tissue destruction and/or perforation that result in acute, severe regurgitation (*Fig. 13.23*). This may lead to hemodynamic instability and heart failure. Two-dimensional echocardiography is useful to detect such structural changes in the valve, to confirm the hemodynamic sequelae using Doppler imaging, and to measure the overall impact on cardiac function. It is important to recognize that, in the setting of active endocarditis, such changes in valve function often occur suddenly and lead to abrupt changes in clinical status. The availability and appropriate use of echocardiography in such situations may be lifesaving.
Figure 13.24 includes two examples of anterior mitral leaflet perforation due to the destructive effects of the infectious process. Note the difference in the severity of regurgitation between the two cases. An example of perforation of the aortic valve is provided in Figure 13.25. In this case, a mechanical mitral prosthesis is present and appears to function normally. There is severe aortic regurgitation through a perforation in the aortic valve cusp, just above the mitral sewing ring. In Figure 13.26, severe tricuspid regurgitation resulted from damage to the valve despite successful antibiotic treatment of the infection. The very large regurgitant orifice that occurred as a result can be seen on the three-dimensional image.

<table>
<thead>
<tr>
<th>Structural</th>
<th>Hemodynamic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leaflet rupture</td>
<td>Acute valvular regurgitation</td>
</tr>
<tr>
<td>Flail leaflet</td>
<td>Valve obstruction</td>
</tr>
<tr>
<td>Leaflet perforation</td>
<td>Heart failure</td>
</tr>
<tr>
<td>Abscess</td>
<td>Intracardiac shunt</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>Tamponade</td>
</tr>
<tr>
<td>Fistula</td>
<td>Perivalvular regurgitation</td>
</tr>
<tr>
<td>Prosthetic valve dehiscence</td>
<td></td>
</tr>
<tr>
<td>Embolization</td>
<td></td>
</tr>
<tr>
<td>Pericardial effusion</td>
<td></td>
</tr>
</tbody>
</table>

An abscess is a localized pocket of infection (most often caused by staphylococci or enterococci bacteria) that appears on ultrasound as either an echodense or an echolucent mass within the tissue. The most common location for an abscess is near the annulus of the aortic or mitral valve where it can affect valve function and/or the conducting system of the heart. An example of an aortic annulus (or ring) abscess is shown in Figure 13.27. This form of infection will sometimes develop in the absence of an associated vegetation. Although this example is clearly visualized on the transthoracic echocardiogram, most ring abscesses require transesophageal echocardiography for detection.
Abscesses may extend locally to affect adjacent structures. Figure 13.28 is a large aortic annular abscess extending into the roof of the left atrium. An abscess involving the mitral valve is shown in Figure 13.29. In this case, multiple vegetations on the aortic valve extended to involve the mitral valve. Although an annular abscess was not demonstrated, the anterior leaflet was extensively involved, with an aneurysmal pocket and abscess formation, as well as a vegetation.

An abscess may rupture to allow communication with one of the cardiac chambers. Echocardiographically, this can be detected as a fistulous connection between two chambers of the heart (such as the right and left ventricles) or between the aortic root and a chamber (i.e., between a sinus of Valsalva and the left or right atrium; see Figs. 13.30 and 13.31). When rupture does occur, color flow imaging may demonstrate flow within the abscess cavity. Doppler imaging is essential to document flow within the fistula and to demonstrate its connection to another chamber or space. Depending on the coronary sinus involved, the location of the fistula varies widely and may communicate with any of the cardiac chambers.

Detecting abscess formation is difficult on clinical grounds. Aside from the development of atrioventricular conduction abnormalities, there are few clinical clues that suggest abscess development. Echocardiography therefore plays an important role in diagnosis. Although it is well established that transthoracic imaging has low sensitivity to detect abscesses, transesophageal echocardiography is an excellent technique for this purpose. Areas, particularly in the region of the aortic annulus, with abnormal thickening (Fig. 13.32), either echodense or echolucent, should raise the suspicion of abscess formation in the appropriate clinical setting. Notice in Figure 13.33 the presence of an echo-free space at the base of the aortic valve posteriorly. The extent of this is well defined using transesophageal echocardiography. Blood flow in this space, as well as severe aortic regurgitation, is demonstrated by color Doppler imaging. Although the large vegetation was seen on transthoracic imaging, the extent of the aortic root involvement required transesophageal echocardiography. Detecting an annular abscess in the postoperative setting can be difficult. Early after aortic valve replacement, the presence of the sewing ring and nonspecific thickening and edema, attributable to the procedure can make it difficult to distinguish a ring abscess from normal healing. Figure 13.34 is from a patient who had undergone
bioprosthetic aortic valve replacement 2 months previously and then presented with recurrent infection and evidence of multiple vegetations on the prosthetic leaflets. The short-axis transesophageal echocardiographic view suggests heterogeneous thickening in the area of the sewing ring. However, at the time of surgery, no evidence of an annular abscess was found.

FIGURE 13.23. These images were recorded from a patient undergoing treatment for staphylococcal bacteremia. A: Masses (arrows) can be seen at the base of the aortic valve, extending through the annulus into the LA. B: A diastolic frame confirms the extent of tissue involvement (arrow). C: Severe mitral regurgitation is evident (arrow). D: Severe aortic regurgitation (arrow) is demonstrated. Note the presence of a pericardial effusion.
Mycotic aneurysms result from embolization of septic material from the vegetation to endocardial surface of an artery, with subsequent extension of the infection into the vessel wall. They often affect intracranial arteries, but may involve the aortic root and are similar in many ways to abscesses. A mycotic aneurysm is defined as an echolucent outpouching of the vessel wall or, in the case of the aortic root, the coronary sinuses. It is usually connected through a single channel with the vessel from which it arises. As such, it can be either filled with infectious material or contain free-flowing blood. Such aneurysms may rupture to produce an intracardiac shunt or may undermine the function of the aortic valve. Figure 13.35 is taken from a patient who underwent aortic valve replacement and then presented 1 month later with fever. The aneurysm was evident just below the sewing ring and had partially ruptured into the right ventricle.

Complications such as abscess or aneurysm formation may result in spread of infection into the pericardial space producing purulent pericarditis. Clinical evidence of pericarditis in an acutely ill patient, coupled with echocardiographic evidence of a pericardial effusion, should suggest the possibility of purulent pericarditis, usually a surgical emergency. Such effusions are rarely large in volume. Effusions due to purulent pericarditis may be difficult to differentiate from other causes of effusion, and the diagnosis is generally established on clinical grounds.

Among the most devastating of complications of endocarditis is an embolic event. Surveillance studies using CT or MRI have shown an
unexpectedly high prevalence of unrecognized embolic events in patients with active endocarditis. Vegetations in the left side of the heart can embolize to cause stroke, distal infection, renal failure, or ischemia. Figure 13.36 is taken from a patient who presented with signs of an embolic stroke. In this case, the vegetation was moderate in size, but its high mobility was a clue to the embolic risk. Figure 13.37 demonstrates a very large and mobile vegetation involving the mitral valve. Right-sided endocarditis can lead to pulmonary emboli and pneumonia. In some cases, an embolic event is the first manifestation of endocarditis. More often, patients undergoing antibiotic therapy are suddenly affected, usually without warning. After such an event, echocardiography will sometimes show a reduction in size or a change in appearance of the vegetation (Fig. 13.38). The most important role of echocardiography in this setting is to predict patients at risk of these devastating events, a topic that is covered in the next section.
FIGURE 13.24. Two examples of mitral valve perforation as a complication of endocarditis. A, B: Images are from a patient with a small perforation studied with transesophageal echocardiography. A: Thickening at the base of the anterior leaflet, involving the aortic annulus is evident. B: Color Doppler imaging during systole demonstrates a jet into the left atrium (arrow). A larger perforation involving the anterior mitral leaflet is demonstrated (C, D). C: The defect within the midportion of the leaflet is apparent. D: Color Doppler imaging reveals a severe degree of regurgitation through the perforation (arrow).
FIGURE 13.25. From a patient with a mechanical mitral prosthesis, transesophageal echocardiography shows a large perforation of the aortic valve (A, arrow). In B, the vena contracta of the regurgitant jet is seen (arrow), but its extent is obscured by shadowing from the prosthesis.
FIGURE 13.26. In a patient with a history of intravenous drug abuse and prior tricuspid valve endocarditis, this transesophageal echocardiogram was recorded following completion of antibiotic therapy. Severe tricuspid regurgitation (arrows) is demonstrated from the four-chamber (A) view. In B, a defect in the tricuspid valve is apparent. In C, using three-dimensional imaging, this view was obtained from the perspective of the right atrium, looking down at the tricuspid valve. A very large regurgitant orifice is outlined by the arrows, consistent with severe regurgitation.
FIGURE 13.27. An aortic ring abscess (arrow) is seen from a transthoracic echocardiogram. Extensive and diffuse thickening around the circumference of the annulus can be seen in both the long-axis (A) and short-axis (B) views.

Video 13-27b
FIGURE 13.28. A very large annular abscess is seen using transesophageal echocardiography. In A, a small vegetation on the aortic valve is present (small arrow) and a large abscess (arrows) is seen extending into the LA. In B, the short-axis view demonstrates the size and extent of the abscess cavity (arrows).

Video 13-28a

coming soon
FIGURE 13.29. Endocarditis involving the aortic and mitral valves is demonstrated. In A, an aortic valve vegetation (*arrow*) is seen from the apical long-axis view. In B, an abscess within the anterior mitral valve leaflet (*arrow*) is evident.
FIGURE 13.30. A fistula complicating aortic valve endocarditis is demonstrated. Color Doppler shows the connection between the aortic sinus and the right atrium (arrows). Tricuspid regurgitation is also present.
PROGNOSIS AND PREDICTING RISK

Complications develop in approximately 40% of patients being treated for active endocarditis and are a major determinant of outcome. Since complications are invariably associated with a worsening prognosis, identifying patients at risk for their development is an important goal. Several investigations have attempted to stratify patients into low- and high-risk subsets and to identify those at risk of complications on the basis of clinical and echocardiographic findings. Most of the parameters that determine high- and low-risk status are clinical, including age, type of organism, and development of heart failure. A history of intravenous drug use is also a poor prognostic indicator, in part because of the high rate of recurrent infection (Fig. 13.39). In addition, stroke occurrence consistently has been a strong negative determinant of outcome in patients with endocarditis. Echocardiography, if it could predict the likelihood of embolization, would be very useful to predict high-risk status before complications developed.
FIGURE 13.31. As a complication of aortic valve endocarditis, this patient demonstrated a ruptured sinus of Valsalva aneurysm (asterisk). In this case, the noncoronary sinus was involved (A). When rupture occurred, a shunt developed between the aortic root and the right atrium (arrows) (B).

Video 13-31

The only echocardiographic parameter that has been consistently associated with an increased risk of complications is vegetation size. In one study (Sanfilippo et al., 1991), there was a strong and nearly linear relationship between vegetation size and the risk of complications. For example, vegetations less than 7 mm in size accounted for less than 10% of all complications, whereas those that were greater than 11 mm in size
accounted for more than half of the complications. In a meta-analysis by Tischler and Vaitkus (1997) involving 10 studies and 738 cases, the risk of an embolic event in patients with a vegetation greater than 10 mm in size was threefold higher than in patients with smaller vegetations (Fig. 13.40). It is clear that a direct relationship exists between vegetation size and risk. The larger the vegetation is, the greater the likelihood of complications, particularly embolic events. Furthermore, an increase in vegetation size after 4 weeks of antibiotic therapy is additional evidence of high-risk status and should prompt consideration for surgical intervention (Fig. 13.41). Other parameters that have been implicated as increasing the risk of complications include high mobility of the vegetation, multiple sites of involvement, and extension to extravalvular structures.

In addition to size, vegetation location has also been associated with risk. In one study by Cabell et al. (2001), patients with mitral valve involvement were three times more likely to develop embolic complications compared with patients with aortic valve vegetations. It is more likely that embolic risk depends on multiple factors, both clinical and echocardiographic. In one large multicenter study (Hubert et al., 2013), an algorithm that incorporated clinical data (age, diabetes, atrial fibrillation, and prior embolic event), echocardiographic findings (vegetation length), and microbiology (S. aureus bacteremia) were strongly predictive of the risk of an embolic event over a 6-month follow-up period.

**PROSTHETIC VALVE ENDOCARDITIS**

Endocarditis involving a prosthetic valve is a diagnostic and management challenge. The highly reflective nature of prosthetic material, the shadowing created by the prosthesis, and the effect of the device being implanted on the underlying tissue combine to reduce the accuracy of echocardiographic imaging. Vegetations on prosthetic valves most often occur on the base or sewing ring of the structure (Fig. 13.42). Distinguishing small vegetations from the prosthetic material (and especially from the sutures used to secure the valve in place) can be extremely difficult. Therefore, diagnosis of endocarditis in this setting requires a thorough echocardiographic assessment from all available windows. Transthoracic echocardiography is limited in its
ability to secure the diagnosis of prosthetic valve endocarditis. When extensive infection is present, as is shown in Figure 13.43, chest wall imaging may be adequate.

**FIGURE 13.32.** An abscess should be suspected whenever abnormal thickening of the aortic root is seen. In A, a moderate degree of thickening is seen in the long-axis posteriorly (arrows). In B, the short-axis view more convincingly reveals abscess formation along the posterior wall of the aortic root (arrow).
FIGURE 13.33. This patient presented with a stroke and evidence of aortic valve endocarditis. A shows a large aortic valve vegetation (arrows). In B, from the long-axis view, the vegetation is indicated by the white arrow. In addition, the echo-free space posterior to the aortic valve annulus (arrowhead) is consistent with abscess formation. Diastolic flow within this space (as well as severe aortic regurgitation) is demonstrated with color Doppler imaging (C).
FIGURE 13.34. Short-axis transesophageal echocardiographic image of a patient who had undergone recent bioprosthetic aortic valve replacement. He had multiple vegetations on this prosthetic valve and the echocardiogram showed diffuse thickening of his aortic annulus. No evidence of abscess formation was found at surgery.

Video 13-34

However, transthoracic echocardiography is rarely sufficient to exclude the diagnosis of endocarditis in patients with prosthetic valves in whom there
is a high index of suspicion. For example, in a patient with a mitral valve prosthesis, visualizing that portion of the left atrium immediately behind the prosthesis may be impossible from any transthoracic window. In such cases, the perspective available from transesophageal imaging is often essential (Fig. 13.44). Conversely, the ventricular aspect of a tricuspid valve prosthesis may be more readily imaged from the chest wall as opposed to the esophagus. Thus, a combination of the two techniques may be necessary for a complete interrogation. **Figure 13.45** illustrates two cases, both of which involve very large vegetations on bioprosthetic tricuspid valves in the setting of chronic intravenous drug use. The valve itself is completely obscured by the vegetation, and a minor degree of obstruction was present. Another example of obstruction due to a prosthetic valve vegetation is shown in **Figure 13.46**. In this case, a porcine mitral prosthesis is involved and the large vegetation results in a significant diastolic pressure gradient, which was recorded on both transthoracic and transesophageal imaging.

**FIGURE 13.35.** This patient underwent aortic valve replacement 1 month previously. **A:** The valve became infected, which led to the development of a mycotic aneurysm (arrows). **B:** Color Doppler imaging demonstrates a fistulous connection through the aneurysm and into the right ventricle.
Transesophageal echocardiography has increased the accuracy of detecting endocarditis in patients with prosthetic valves and investigators have consistently demonstrated a much higher sensitivity for transesophageal echocardiography in this setting. The improvement in accuracy is so great that many echocardiographers view transesophageal echocardiography as the initial procedure of choice when prosthetic valve endocarditis is suspected. In addition, complications associated with prosthetic valve endocarditis (especially annular abscesses) are more consistently visualized from the transesophageal approach (Figs. 13.47 and 13.48). Figure 13.49 is an example of dehiscence of a porcine aortic valve. On the video loop, the independent (rocking) motion of the prosthesis is apparent.
The advantages of transesophageal echocardiography for assessing patients with suspected prosthetic valve endocarditis have been well demonstrated. However, even with transesophageal echocardiography, it can be challenging to confirm a diagnosis. Figure 13.50 is from a patient who
presented with fever 9 months after transcatheter aortic valve replacement (TAVR). Shadowing from the prosthetic material made detection of the vegetation difficult. A careful and thorough examination was required to clearly demonstrate the presence of the infection.

**FIGURE 13.37.** Long-axis (A) and four-chamber (B) views of the mitral valve are provided. A large, highly mobile mitral valve vegetation is considered one of the greatest risk factors for embolization in infective endocarditis.  

**Video 13-37**
FIGURE 13.38. The appearance of a vegetation may change as a result of embolization. A: A large and mobile vegetation (arrows) can be seen attached to the left atrial side of the posterior mitral leaflet. B: An echocardiogram recorded 1 week later, after a stroke. Note that the vegetation (arrow) is much smaller, most likely the result of embolization.

Video 13-38

INFECTED INTRACARDIAC DEVICES

In addition to prosthetic valves, other types of prosthetic material within the heart or vasculature can become infected. As with prosthetic valves, infection
can occur early or later after implantation. When infection occurs early after implantation, it is usually due to the presence of pre-existing infection or as a complication of the procedure itself. Late infection is most often the result of seeding of the prosthetic material by blood-borne organisms. In either case, infected prosthetic devices are very difficult to treat without removal and are associated with a poor prognosis. An example of an infected pacemaker lead is shown in Figure 13.51. In most cases, detection of evidence of infection requires transesophageal echocardiography. The presence of a mobile mass attached to either an indwelling catheter or chamber wall suggests the possibility of endocarditis. However, distinguishing vegetations from thrombus is virtually impossible on echocardiographic grounds alone and invariably requires clinical correlation. In the absence of clinical signs of infection, such a mass most likely represents thrombus and should be treated accordingly. However, the same echocardiographic appearance, occurring in the setting of fever and/or positive blood cultures, strongly suggests endocarditis. With the growing use of these devices, the incidence of this type of endocarditis will certainly increase.

FIGURE 13.39. A shows a vegetation on the aortic valve of a young patient with a history of intravenous drug abuse. Aortic root thickening is also present and transesophageal echocardiography confirmed the presence of a ring abscess. The patient underwent bioprosthetic aortic valve replacement and then presented 8 months later with signs of recurrent infection (B). A large, extensive vegetation is seen on the prosthetic leaflets.
Video 13-39a

Video 13-39b
FIGURE 13.40. A meta-analysis of studies that examine whether vegetation size could predict the risk of systemic emboli. The pooled odds ratio for increased risk associated with large vegetation was 2.80 (95% confidence interval 1.95 to 4.02, p < 0.01). (Reprinted with permission from Tischler MD, Vaitkus PT. The ability of vegetation size on echocardiography to predict clinical complications: a meta-analysis. J Am Soc Echocardiogr. 1997;10(5):562–568. Copyright © 1997 American Society of Echocardiography.)

RIGHT-SIDED ENDOCARDITIS

Endocarditis involving the tricuspid valve is most commonly seen in the setting of intravenous drug use (see Fig. 13.2) or in association with an indwelling catheter in the right ventricle (usually a pacing lead). In one series
by Hecht and Berger (1992) involving 121 intravenous drug users, a tricuspid valve vegetation was seen in all cases, whereas the pulmonary valve was involved in only four. However, intravenous drug use is not the only cause of tricuspid valve endocarditis. Figure 13.52 is from a patient with a small perimembranous ventricular septal defect who developed a tricuspid valve vegetation, in part due to the left-to-right jet impinging on the anterior tricuspid valve leaflet. Vegetation size tends to be greater in right-sided endocarditis, and some degree of tricuspid regurgitation is generally present (see Figs. 13.26 and 13.45). Endocarditis involving the tricuspid valve also tends to be better tolerated than left-sided disease and is less likely to require valve surgery. In such patients, echocardiography is important to follow the course of the disease and to help ensure the effectiveness of medical therapy. Figure 13.53 is from a patient with a history of intravenous drug use and tricuspid valve endocarditis. On the initial study, the tricuspid valve is intact, there is a moderate-sized vegetation and moderate tricuspid regurgitation. The right ventricular size and function are normal. After completing a course of antibiotics, the patient returned 6 months later. Although the vegetation is less apparent, the right ventricle is now enlarged, the tricuspid valve is disrupted, and the regurgitation is more severe.

**FIGURE 13.52.** Vegetation size is a poor prognostic sign. In this patient, a moderate-sized aortic valve vegetation (arrow) was seen on the initial echocardiogram (A). Three weeks later, a significant increase in vegetation size is apparent (B).

**FIGURE 13.53.** Vegetation size is a poor prognostic sign. In this patient, a moderate-sized aortic valve vegetation (arrow) was seen on the initial echocardiogram (A). Three weeks later, a significant increase in vegetation size is apparent (B).
FIGURE 13.42. From a patient with a St. Jude mitral prosthesis, a large vegetation (*small arrows*) can be seen in the left atrium attached to the sewing ring (*large arrows*). [Video 13-42] coming soon
Pulmonary valve vegetations are less common and can be difficult to visualize. They may rarely develop in patients as a complication of pulmonary artery catheterization. Figure 13.54 is an example of multiple large vegetations affecting the pulmonary valve in a patient with a history of intravenous drug use. In this case, a tricuspid valve vegetation was also present.

The superiority of transesophageal echocardiography is less well established in right-sided endocarditis. Because the tricuspid valve is well seen from the transthoracic windows and because right-sided vegetations are typically large, transthoracic echocardiography is often adequate for diagnosis and both techniques have demonstrated high sensitivity. Even after successful antibiotic therapy, when infection is no longer clinically active, masses on the tricuspid valve often remain. Thus, differentiating active from healed endocarditis in this situation is often difficult.

**CLINICAL APPROACH TO THE PATIENT WITH ENDOCARDITIS**

Although it is clear that echocardiography is indispensable in the evaluation of patients with suspected endocarditis, the decisions about when and how often the test should be performed remain somewhat controversial. Guidelines for the use of echocardiography in patients with known or suspected endocarditis are provided in Table 13.1. In addition to underscoring the versatility of echocardiography in this setting, these guidelines also provide some advice about the relative value of transthoracic versus transesophageal imaging. In most patients in whom there is a clinical suspicion of endocarditis, echocardiography is helpful whether the results are positive or negative. The results help establish or exclude the diagnosis and also provide prognostic information, establish a baseline for comparison, and may even identify patients in whom prompt surgical intervention is recommended. In must be emphasized, however, that a negative echocardiogram alone does not exclude the possibility of endocarditis and must be interpreted in clinical context.

An inevitable consequence of the utility of echocardiography in endocarditis is the potential for overuse. This is particularly true among
patients in whom the pretest likelihood of endocarditis is extremely low and no additional testing, including echocardiography, is likely to yield important new information and alter clinical management. Unfortunately, there is relatively little guidance to inform clinicians when not to order an echocardiogram. Although appropriate use criteria have been published, only a few scenarios specifically address the issue of endocarditis (Table 13.6). For example, echocardiography is considered rarely appropriate in the setting of transient fever but in the absence of a new murmur or bacteremia. Thus, the rationale to perform echocardiography must depend on clinical findings that increase the pretest likelihood of disease, such as fever, an abnormal physical examination, or blood culture results for an appropriate organism.

**FIGURE 13.43.** Although transesophageal echocardiography is more sensitive, an annular abscess can sometimes be detected on transthoracic imaging. In the long-axis view (A), a large aortic valve vegetation is seen (arrow). The short-axis view (B) shows thickening and heterogeneity of the annulus (arrows), consistent with abscess formation.
Once the decision is made to perform an echocardiogram, the choice between transthoracic and transesophageal echocardiography must be made. Given the well documented superior sensitivity of transesophageal imaging, it is tempting to conclude that this should be the test of choice. However, several studies have demonstrated the reasonably high negative predictive value of a normal transthoracic echocardiogram. Thus, because of its greater cost and invasiveness, the higher sensitivity of transesophageal echocardiography must be weighed against these factors. As a result, a transthoracic echocardiogram is the initial test of choice for many situations. The negative predictive value of the test is high, and, if image quality is acceptable, the absence of positive findings is often sufficient to avoid the need for further testing. Recent studies have emphasized the importance of using rigorous criteria to define a negative transthoracic study, and to distinguish negative from indeterminate. In the absence of a prosthetic valve and when image quality is high, factors such as no valve stenosis or sclerosis, no more than trivial regurgitation, and absence of implanted hardware are important. When all these criteria are met on transthoracic echocardiography, the negative predictive value is increased substantially, permitting transesophageal echocardiography to be avoided in many cases.
FIGURE 13.44. A stentless aortic valve is recorded with transesophageal echocardiography. A: The aortic root is thickened and echogenic. B: A short-axis view demonstrates abscess formation posteriorly (arrows). C: Color Doppler imaging reveals flow within the abscess cavity (arrows).
FIGURE 13.45. Examples of large bioprosthetic tricuspid valve vegetations due to Staphylococcus aureus endocarditis are shown from two different patients (A, B). Right-sided vegetations can be large and cause partial obstruction to right ventricular inflow. [Video 2]
FIGURE 13.46. An infected porcine mitral prosthesis. The valve leaflets are thickened and relatively immobile (arrows) (A). B: On the transesophageal echocardiogram, thickening and decreased motion (arrow) were apparent. C: Doppler imaging demonstrates a mean gradient of 22 mm Hg across the prosthesis.
FIGURE 13.47. Transesophageal echocardiography in a patient with a dehisced, infected porcine aortic valve is shown. In A, the prosthesis has separated from the annulus, resulting in a large anterior clear space (asterisk) within the aortic root. Vegetations are present on the sewing ring (arrows). Color Doppler (B) reveals perivalvular regurgitation, from the aorta into the left ventricular outflow tract.
FIGURE 13.48. A transesophageal echocardiogram from a patient with porcine aortic valve endocarditis. Diffuse thickening of the bioprosthetic leaflets is seen in both the long-axis (A) and short-axis views (B). In addition, partial dehiscence of the sewing ring is demonstrated in (B) (arrow).
FIGURE 13.49. Dehiscence of a bioprosthetic aortic valve results in an unstable, rocking motion of the sewing ring. Systolic (A) and diastolic (B) frames illustrate the exaggerated motion of the prosthesis (arrow) throughout the cardiac cycle.
FIGURE 13.50. An infected transcatheter aortic valve is demonstrated using transesophageal echocardiography. In the long-axis plane (A), the arrow indicates the vegetation protruding into the outflow tract. In B, the short-axis view again shows the mass within the device (arrow).
However, if a high clinical index of suspicion remains after a negative or nondiagnostic transthoracic study, transesophageal echocardiography should be performed (see Table 13.1). Situations in which transesophageal echocardiography should be used as the initial test of choice include: (1) those patients in whom image quality on chest wall imaging is unacceptable; (2) those with prosthetic valves; and (3) those in whom complications such as abscess formation are suspected on clinical grounds. In one study (Fowler et al., 1997), the yields of transthoracic and that of transesophageal echocardiography were compared in a series of 103 patients with staphylococcal bacteremia who were evaluated relatively early in the course of their illness. Both forms of echocardiography were performed, and the results were interpreted independently (Fig. 13.55). In this clinical setting, the advantages of transesophageal imaging were clearly demonstrated, perhaps because the imaging was performed so early in the course of the disease, when vegetations are likely to be relatively small (see Fig. 13.8).

The choice between transthoracic and transesophageal echocardiography can also be addressed from the perspective of cost-effectiveness. Heidenreich et al. (1999) used a decision-analysis technique to compare the two tests in patients with a high pretest probability (4% to 60%) of having endocarditis. These investigators assessed the health and economic outcomes of various
groups using six different strategies: (1) empiric treatment of bacteremia (short-course therapy); (2) empiric treatment of endocarditis (long-course therapy); (3) treatment based on transthoracic echocardiographic results; (4) treatment based on transesophageal echocardiographic results; (5) treatment based on transesophageal echocardiography after negative transthoracic study; and (6) treatment based on transthoracic echocardiographic results (unless the study was negative and image quality was poor, in which case, a transesophageal echocardiogram was obtained). Their results confirmed that the pretest probability of endocarditis, based on the history and physical and laboratory data, was essential in deciding which strategy was most effective. Their model suggested that transesophageal echocardiography alone increased the quality-adjusted life days and reduced the cost of diagnosis compared with transthoracic echocardiography for a wide range of relative costs for the two tests. Although the limitations of this approach are evident, the results do support a prominent role for transesophageal echocardiography in many patients with suspected endocarditis.

**FIGURE 13.52.** In A, a small perimembranous ventricular septal defect is shown from the long-axis view (arrow). On the right, color Doppler demonstrates left-to-right shunting into the right ventricle. In B, a tricuspid valve vegetation is indicated by the arrow.
### Table 13.6  EXAMPLES OF APPROPRIATE USE CRITERIA IN PATIENTS WITH KNOWN OR SUSPECTED ENDOCARDITIS

<table>
<thead>
<tr>
<th>Indication</th>
<th>Appropriate Use Criteria Score (1–9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Symptoms or conditions potentially related to suspected cardiac etiology including but not limited to chest pain, shortness of breath, palpitations, TIA, stroke or peripheral embolic event</td>
<td>A (9)</td>
</tr>
<tr>
<td><strong>Infective endocarditis (native or prosthetic valves) with TTE</strong></td>
<td></td>
</tr>
</tbody>
</table>
52. Initial evaluation of suspected infective endocarditis (native and/or prosthetic valve) with positive blood cultures or a new murmur A (9)

53. Transient fever without evidence of bacteremia or a new murmur I (2)

54. Transient bacteremia with a pathogen not typically associated with infective endocarditis and/or a nonendovascular source of infection I (3)

55. Reevaluation of infective endocarditis at high risk for progression or complications or with a change in clinical status or cardiac examination A (9)

56. Routine surveillance of uncomplicated infective endocarditis when no change in management is contemplated I (2)

**Transthoracic echocardiography for evaluation of intracardiac structures and chambers**

57. Suspected cardiac mass A (9)

58. Suspected cardiac source of embolus A (9)

**Transesophageal echocardiography as initial or supplemental test**

107. To diagnose infective endocarditis with a low pretest probability (e.g., transient fever, known alternative source of infection, or negative blood cultures/atypical pathogen) I (3)

108. To diagnose infective endocarditis with a moderate or high pretest probability A (9)

ASD, atrial septal defect; PFO, patent foramen ovale.


**FIGURE 13.53.** Progression of valvular disease due to endocarditis in a young patient with a history of drug abuse is shown. The initial study (A) was recorded at the time of hospitalization and a small tricuspid valve vegetation is indicated by the arrow. Six months later (B), the right heart is enlarged and the tricuspid valve is disrupted (arrow). See text for details.
FIGURE 13.54. Pulmonic valve involvement in endocarditis is uncommon. In this example from a patient with a history of drug use, multiple large pulmonic valve vegetations are recorded from the basal short-axis view (A). There was also significant tricuspid valve involvement. As a consequence, the right ventricle was dilated and hypokinetic (B).

Video 13-54a

coming soon
A related question is whether echocardiography can be used to guide duration of antibiotic therapy. This issue has been addressed in the subset of patients with catheter-associated S. aureus bacteremia (Rosen et al., 1999). A model was constructed to test the value (and cost-effectiveness) of transesophageal echocardiography in deciding the optimal duration of therapy. The model compared empiric short-course therapy (2 weeks), long-course therapy (4 weeks), and echocardiography-guided therapy (long course if there was evidence of endocarditis and short course otherwise). The study tested whether the incremental cost of transesophageal echocardiography could be justified on the basis of superior outcomes and/or reduced duration of therapy. The results suggested that the echocardiography-guided strategy offered improved life expectancy compared with empiric short-course therapy and was more cost-effective compared with long-course therapy.
Over a wide range of costs and levels of accuracy, echocardiography was found to be cost-effective in this clinical setting.

The decision to proceed with surgery is a complex one that must rely on clinical criteria as well as echocardiographic findings. Guidelines have recently been published to address the issue of intervening in patients with endocarditis (Table 13.7). The development of heart failure, an embolic event, stroke, or extension of the infection (e.g., abscess formation) are some indications for surgical intervention. Surgery is also usually appropriate treatment for endocarditis caused by fungi or other resistant organisms. Some echocardiographic findings are also considered in this decision-making process. For example, an aortic ring abscess and valve tissue destruction leading to severe regurgitation are often considered surgical indications. Other less dramatic signs should also be sought. Evidence of disease progression might include increase in vegetation size, worsening regurgitation, chamber enlargement, ventricular dysfunction, evidence of elevated filling pressure, or extension of infection to other sites. These changes may occur during therapy in the absence of clinical deterioration and often affect management plans.

**FIGURE 13.56.** Serial changes can be detected echocardiographically. This patient had undergone mitral valve repair and a mitral annular ring was present. **A:** No evidence of endocarditis was detected, despite clinical signs suggesting infection. **B:** Seven months later, marked progression of disease is apparent, despite antibiotic therapy. A large vegetation involving the anterior mitral leaflet (arrows) is present.
Class I

1. Decisions about timing of surgical intervention should be made by a multispecialty Heart Valve Team of cardiology, cardiothoracic surgery, and infectious disease specialists. *(Level of Evidence: B.)*

2. Early surgery (during initial hospitalization before completion of a full therapeutic course of antibiotics) is indicated in patients with IE who present with valve dysfunction resulting in symptoms of heart failure. *(Level of Evidence: B.)*

3. Early surgery (during initial hospitalization before completion of a full therapeutic course of antibiotics) is indicated in patients with left-sided IE caused by *Staphylococcus aureus*, fungal, or other highly resistant organisms. *(Level of Evidence: B.)*

4. Early surgery (during initial hospitalization before completion of a full therapeutic course of antibiotics) is indicated in patients with IE complicated by heart block, annular or aortic abscess, or destructive penetrating lesions. *(Level of Evidence: B.)*

5. Early surgery (during initial hospitalization before completion of a full therapeutic course of antibiotics) for IE is indicated in patients with evidence of persistent infection as manifested by persistent bacteremia or fevers lasting longer than 5 to 7 days after onset of appropriate antimicrobial therapy. *(Level of Evidence: B.)*

6. Surgery is recommended for patients with prosthetic valve endocarditis and relapsing infection (defined as recurrence of bacteremia after a complete course of appropriate antibiotics and subsequently negative blood cultures) without other identifiable sources for portal of infection. *(Level of Evidence: C.)*

7. Complete removal of pacemaker or defibrillator systems, including all leads and the generator, is indicated as part of the early management plan in patients with IE with documented infection of the device or leads. *(Level of Evidence: B.)*

### Table 13.7

<table>
<thead>
<tr>
<th>INDICATIONS FOR INTERVENTION IN PATIENTS WITH ENDOCARDITIS</th>
</tr>
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<tbody>
<tr>
<td><strong>Class I</strong></td>
</tr>
<tr>
<td>1. Decisions about timing of surgical intervention should be made by a multispecialty Heart Valve Team of cardiology, cardiothoracic surgery, and infectious disease specialists. <em>(Level of Evidence: B.)</em></td>
</tr>
<tr>
<td>2. Early surgery (during initial hospitalization before completion of a full therapeutic course of antibiotics) is indicated in patients with IE who present with valve dysfunction resulting in symptoms of heart failure. <em>(Level of Evidence: B.)</em></td>
</tr>
<tr>
<td>3. Early surgery (during initial hospitalization before completion of a full therapeutic course of antibiotics) is indicated in patients with left-sided IE caused by <em>Staphylococcus aureus</em>, fungal, or other highly resistant organisms. <em>(Level of Evidence: B.)</em></td>
</tr>
<tr>
<td>4. Early surgery (during initial hospitalization before completion of a full therapeutic course of antibiotics) is indicated in patients with IE complicated by heart block, annular or aortic abscess, or destructive penetrating lesions. <em>(Level of Evidence: B.)</em></td>
</tr>
<tr>
<td>5. Early surgery (during initial hospitalization before completion of a full therapeutic course of antibiotics) for IE is indicated in patients with evidence of persistent infection as manifested by persistent bacteremia or fevers lasting longer than 5 to 7 days after onset of appropriate antimicrobial therapy. <em>(Level of Evidence: B.)</em></td>
</tr>
<tr>
<td>6. Surgery is recommended for patients with prosthetic valve endocarditis and relapsing infection (defined as recurrence of bacteremia after a complete course of appropriate antibiotics and subsequently negative blood cultures) without other identifiable sources for portal of infection. <em>(Level of Evidence: C.)</em></td>
</tr>
<tr>
<td>7. Complete removal of pacemaker or defibrillator systems, including all leads and the generator, is indicated as part of the early management plan in patients with IE with documented infection of the device or leads. <em>(Level of Evidence: B.)</em></td>
</tr>
</tbody>
</table>

**Class Ila**
1. Complete removal of pacemaker or defibrillator systems, including all leads and the generator, is reasonable in patients with valvular IE caused by *Staphylococcal aureus* or fungi, even without evidence of device or lead infection. (*Level of Evidence: B.*)

2. Complete removal of pacemaker or defibrillator systems, including all leads and the generator, is reasonable in patients undergoing valve surgery for valvular IE. (*Level of Evidence: C.*)

3. Early surgery (during initial hospitalization before completion of a full therapeutic course of antibiotics) is reasonable in patients with IE who present with recurrent emboli and persistent vegetations despite appropriate antibiotic therapy. (*Level of Evidence: B.*)

**Class IIb**

1. Early surgery (during initial hospitalization before completion of a full therapeutic course of antibiotics) may be considered in patients with native valve endocarditis who exhibit mobile vegetations greater than 10 mm in length (with or without clinical evidence of embolic phenomenon). (*Level of Evidence: B.*)


The final decision involves the need for repeat echocardiographic analysis in a patient with an established diagnosis. There are no firm data to support the use of serial echocardiograms in this setting. In most cases, the decision to perform subsequent echocardiograms depends on the clinical course. **Figure 13.56** shows echocardiograms from a patient with a repaired mitral valve. In the first study, the patient had clinical evidence of endocarditis, but no vegetations were apparent on the study and the patient was clinically stable. The prosthetic ring is seen and the leaflets appear normal. Seven months later, the second study shows marked progression of disease, despite a prolonged course of antibiotics. In patients who demonstrate clinical deterioration, repeat testing can be valuable in establishing a cause and guiding subsequent decision making. Alternatively, patients who demonstrate a good response to antibiotic therapy based on subsequent blood culture results as well as history and physical examination are unlikely to benefit from any form of additional testing. Some high-risk subsets of patients, such as those with staphylococcal endocarditis involving the aortic valve, may benefit from a second echocardiogram 7 to 10 days after initiation of therapy to exclude complications such as abscess formation.

**Suggested Readings**
**GENERAL CONCEPTS**


**TRANSESOPHAGEAL ECHOCARDIOGRAPHY**


**DUKE CRITERIA**


**COMPLICATIONS**


Lauridsen TK, Park L, Tong SY, et al. Echocardiographic findings predict in-hospital and one-year


**MULTIMODALITY IMAGING**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Chapter 14
Prosthetic Valves and Structural Heart Disease Interventions

The era of valve surgery preceded the development of echocardiography by only a few years. It is therefore not surprising that one of the earliest applications of echocardiography was the study of prosthetic valve function. With the tremendous advances in surgical techniques over the past four decades, the role of echocardiography has evolved and broadened in this important field. Because neither the perfect valve repair nor the perfect prosthesis yet exists, ongoing assessment of valve function is a key aspect of the management of patients after valve surgery. Echocardiography, with its noninvasive ability to evaluate both anatomy and function, has become the diagnostic modality of choice for this purpose.

More recently, newer less-invasive options have been developed, greatly expanding the role of echocardiography and establishing the echocardiographer as a key member of the heart valve team. While these transcatheter technologies will continue to evolve over the next several years, they have already fundamentally altered the management of many patients with valvular and congenital heart diseases.

The echocardiographic assessment of prosthetic valves is complex. Flow dynamics are different through prosthetic valves compared with native valves. Both the size and type of the prosthesis influence the range of expected flow velocities and thus the definition of normal versus abnormal function. The echocardiographer must determine the specific type of prosthetic valve and whether the structural and functional parameters exceed the limits of normal for a given size and type. Despite these challenges, the
combination of echocardiography and Doppler imaging techniques is ideally suited to assessing prosthetic valves. Whether monitoring valve function over time or detecting the specific cause of prosthesis dysfunction, echocardiographic techniques have become indispensable in this important clinical area.

**TYPES OF PROSTHETIC VALVES**

The two major categories of prosthetic valves are mechanical valves and tissue valves or bioprostheses (Table 14.1). The mechanical prosthetic valves can be further divided into caged-ball and tilting-disk designs. The caged-ball prosthesis was the first type of artificial heart valve, and the Starr–Edwards valve, although no longer implanted, is still encountered in clinical practice (Fig. 14.1). It consists of a circular sewing ring on which is mounted a U-shaped cage that contains a silastic ball occluder. To open, the ball moves forward into the cage, allowing blood flow around the entire circumference. To occlude, the ball is driven back into the sewing ring to prevent backflow.

Several tilting-disk prostheses are currently in use (Fig. 14.2). The single-disk prosthesis consists of a circular sewing ring and a circular disk fixed eccentrically to the ring via a hinge. The disk moves through an arc of less than 90 degrees (usually 60 to 80 degrees), thereby allowing antegrade flow in the open position and seating within the sewing ring to prevent backflow in the closed position. The Björk–Shiley, Omnicarbon, and Medtronic-Hall are examples of single tilting-disk prostheses. Because the hinge is eccentrically positioned within the sewing ring and the disk opens less than 90 degrees, major and minor orifices are created and some stagnation of flow occurs behind the disk. Bileaflet tilting-disk valves consist of two semicircular disks that open and close on a hinge mechanism within the sewing ring. The opening angle is generally more vertical (between 70 and 90 degrees) than the single-disk prosthesis and results in three distinct orifices: two larger ones on either side and a smaller central rectangular-shaped orifice. Examples of bileaflet tilting disks include the St. Jude, On-X, and Carbomedics valves.

Unlike mechanical valves, bioprostheses are constructed from either human or animal tissue (Fig. 14.3). Among the most commonly used are the porcine bioprostheses, including the Hancock and Carpentier-Edwards
valves. These are porcine aortic valves that have been preserved and fixed within a polypropylene mount attached to a Dacron sewing ring. Pericardial prostheses are also in use today. Because the tissue has been preserved, it is less pliable than native valve tissue. The leaflets themselves are supported by stents, which vary in number and design and arise vertically from the sewing ring. “Stentless” bioprostheses have also been developed for use in the aortic position. They consist of porcine aortic valves that include the annulus, valve, and root preserved intact. Stentless aortic valves have neither a prosthetic sewing ring nor supporting stents. Instead, the porcine leaflets are supported via a flexible cuff. They are often customized by the surgeon in the operating room at the time of implantation.

<table>
<thead>
<tr>
<th>Table 14.1 TYPES OF PROSTHETIC VALVES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mechanical</strong></td>
</tr>
<tr>
<td>Caged ball</td>
</tr>
<tr>
<td>Bileaflet tilting disc</td>
</tr>
<tr>
<td>Single disk</td>
</tr>
<tr>
<td><strong>Tissue</strong></td>
</tr>
<tr>
<td>Stented</td>
</tr>
<tr>
<td>Porcine</td>
</tr>
<tr>
<td>Pericardial</td>
</tr>
<tr>
<td><strong>Stentless</strong></td>
</tr>
<tr>
<td>Homograft</td>
</tr>
<tr>
<td>Autograft*</td>
</tr>
<tr>
<td>Porcine</td>
</tr>
<tr>
<td>Pericardial</td>
</tr>
<tr>
<td>Transcatheter</td>
</tr>
<tr>
<td>Aortic</td>
</tr>
<tr>
<td>Mitral</td>
</tr>
<tr>
<td>Pulmonary</td>
</tr>
<tr>
<td>Sutureless</td>
</tr>
</tbody>
</table>

\*Ross procedure, aortic position.
Homograft valves are derived from human aortic or pulmonary valve tissue that has undergone cryopreservation and may be either stented or unstented. They are most often used in the aortic position. Here, they are either implanted in the subcoronary position (called a “free-hand” valve), as a miniroot procedure (implanted within the native aortic root), or as part of a full root and valve replacement procedure. Another example of their use is the Ross procedure, which involves autotransplantation of the pulmonary valve into the aortic position and placement of homograft in the pulmonary position. Homografts are also used in valved conduits but are rarely used to replace a mitral or tricuspid valve.
Percutaneous approaches to valve replacement are a more recent development and are currently available for the pulmonary, aortic, and mitral positions. Transcatheter aortic valve replacement (TAVR) was first performed in 2002 and the technology has been commercially available in Europe since 2007 and in the United States since 2011. Current devices consist of a pericardial tissue valve suspended in an alloy frame and are either self-expanding or balloon expandable (Fig. 14.4). More recent models are both repositionable and retrievable.

Transcatheter approaches to mitral valve repair or replacement are a very active area of development. At present, the only methodology that is commercially available in the United States is the MitraClip device, which is approved for treatment of moderately severe or severe primary mitral regurgitation. Based on the Alfieri surgical procedure, this approach involves simultaneous grasping of the anterior and posterior leaflets and then securing
them with a clip-like device, creating a double-orifice mitral valve. Transcutaneous mitral valve replacement, or TMVR, remains investigational in the United States, but several technologies and approaches are undergoing clinical study. Transcatheter pulmonary valve replacement, the first of the percutaneous valve replacement technologies to be developed, is covered in Chapter 19 on congenital heart diseases.

![A porcine bioprosthetic valve.](image)

**FIGURE 14.3.** A porcine bioprosthetic valve.

Surgical valve repair, although not involving a prosthetic valve, usually requires the use of prosthetic material. Aortic valve repair has been performed successfully in a limited number of centers. It may be useful in the treatment of regurgitant bicuspid valves or in the setting of regurgitation due to aortic root pathology. Mitral valve repair is performed more widely and with more consistently successful results. It is generally undertaken in the setting of a myxomatous valve or when mitral regurgitation is due to left ventricular dilation or dysfunction. Both surgical and percutaneous approaches are available. In most cases, mitral repair involves use of a ring to reduce the effective size of the valve annulus (Fig. 14.5).
FIGURE 14.4. Two models of currently available transcatheter aortic valves are shown. Edwards SAPIEN valve on the left and a Medtronic CoreValve on the right.
NORMAL PROSTHETIC VALVE FUNCTION

The indications for echocardiography in patients with prosthetic valves are summarized in Table 14.2. Visualization of prosthetic valves often requires a combination of transthoracic and transesophageal imaging. Although the role of three-dimensional imaging continues to evolve, the improved spatial orientation provided by modern equipment provides a unique and potentially valuable perspective. Two-dimensional echocardiography is used to determine the type of valve and to evaluate its structure and function. Using this modality, the stability of the sewing ring is assessed. Rocking or independent motion of the prosthesis is often an indication of dehiscence. The presence of abnormal masses, such as thrombi or vegetations, should be determined. Shadowing from the prosthesis may obscure such pathology and multiple imaging windows may be required for a complete evaluation.
Motion of the leaflets, disks, or occluder mechanism should also be assessed from the two-dimensional study. An important early step in the echocardiographic assessment of prosthetic valves is recognizing the range of normal findings. Figure 14.6 is an example of a normally functioning porcine aortic prosthesis. Leaflet opening during systole resembles that of a normal native valve. The overall appearance is so similar, in fact, that when examined with transthoracic echocardiography (Fig. 14.6A,B), normally functioning aortic bioprostheses are occasionally mistaken for “normal” native valves. When examined carefully, however, the sewing ring and struts are more echogenic than normal and tend to shadow the leaflets, a clue to the presence of prosthetic material. Transesophageal echocardiography, however, permits clear visualization of sewing ring and supporting struts, as well as the cusps (see Fig. 14.6C,D). A normal porcine mitral prosthesis, assessed using transesophageal echocardiography, is shown in Figure 14.7. The relationship between the leaflets and the supporting sewing ring is seen clearly.

Figure 14.8 shows a Starr–Edwards valve in the mitral position. The protruding, high-profile cage in the left ventricle is diagnostic. When examined in real time, the poppet can be seen moving forward and backward in the cage. These valves are highly echogenic, and small thrombi or vegetations can be easily hidden or overlooked. A normally functioning St. Jude mitral prosthesis is presented in Figures 14.9 and 14.10. In Figure 14.9, the two hemidisks open and close in synchrony, although it is often difficult to distinguish both on transthoracic imaging. Significant shadowing occurs, and the left atrium is not well seen in most cases. In Figure 14.10, three-dimensional echocardiography is used to more completely visualize the hemidisks. This approach also provides a thorough circumferential recording of the sewing ring. Figure 14.11 shows a stable aortic St. Jude valve. In this example, the disks are obscured by the walls of the aorta. A distinct shadow from the sewing ring is apparent, extending into the left atrium. In Figure 14.12, a recently implanted On-X aortic prosthesis is shown. Stentless aortic valves are the most recent option in prostheses and are being implanted with increasing frequency. An example of a normal Medtronic Freestyle valve is provided in Figure 14.13. Distinguishing a normally functioning stentless valve from a native aortic valve can be impossible. Prosthetic valves in the tricuspid position are usually biologic. An example of a normal porcine tricuspid valve is shown in Figure 14.14. The leaflets are barely visible.
within the supporting structure.

### Table 14.2 RECOMMENDATIONS FOR EVALUATION OF PROSTHETIC VALVES

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
<th>Level of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. An initial TTE study is recommended in patients after prosthetic valve implantation for evaluation of valve hemodynamics</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>2. Repeat TTE is recommended in patients with prosthetic heart valves if there is a change in clinical symptoms or signs suggesting valve dysfunction</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>3. TEE is recommended when clinical symptoms or signs suggest prosthetic valve dysfunction</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>4. Annual TTE is reasonable in patients with a bioprosthetic valve after the first 10 years, even in the absence of a change in clinical status</td>
<td>Iiia</td>
<td>C</td>
</tr>
<tr>
<td>5. TTE is indicated in patients with suspected prosthetic valve thrombosis to assess hemodynamic severity and follow resolution of valve dysfunction</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>6. TEE is indicated in patients with suspected prosthetic valve thrombosis to assess thrombus size and valve motion</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>7. TTE is recommended in patients with suspected IE to identify vegetations, characterize the hemodynamic severity of valvular lesions, assess ventricular function and pulmonary pressures, and detect complications</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>8. Intraoperative TEE is recommended for patients undergoing valve surgery for IE</td>
<td>I</td>
<td>B</td>
</tr>
</tbody>
</table>

FIGURE 14.6. An example of normally functioning bioprosthetic aortic valve is shown. Transthoracic long-axis (A, B) and transesophageal short-axis (C, D) images of the valve are provided.

**Video 14-6**

Transcatheter-placed aortic prostheses, due to their larger frame, create a greater degree of shadowing compared to other bioprosthetic valves. In most
other aspects, however, their appearance on echocardiography is similar. The normally functioning leaflets are thin mobile structures partially obscured by the aortic wall, the native (often calcified) aortic valve material, and the alloy frame. **Figure 14.15** is an example of a recently deployed TAVR device. The stent is seen within the aortic root, extending just below the annulus. The leaflets are barely discernable within the lower portion of the scaffold.

**FIGURE 14.7.** A transesophageal echocardiogram from a patient with a normally functioning porcine mitral prosthesis. The valve is seen from the long-axis view in systole (A) and diastole (B). In C, Doppler reveals a normal laminar flow pattern.
FIGURE 14.9. A normally functioning St. Jude mitral prosthesis. **A:** During systole, the hemidisks are shown in the closed position (arrows). **B:** During diastole, the two disks are recorded in the open position (arrows).
FIGURE 14.10. A three-dimensional echocardiogram of a normal St. Jude mitral prosthesis is shown from the perspective of the left atrium. In real time, the hemidisks (arrows) are seen to open and close from above.

Blood flow through normally functioning prosthetic valves differs from flow through native valves in several important ways. First, artificial heart valves are inherently stenotic. There is a variety of explanations for this consistent observation. The sewing ring of the valve may be too small relative to the flow. In young patients, what passes for an adequately sized valve in childhood may become functionally stenotic as the patient grows. More importantly, the effective orifice area (EOA) is significantly smaller
than the area of the sewing ring because the valve assembly (i.e., the occluder mechanism) occupies some of the central space. Leaflets of bioprostheses, by virtue of the preservation process, are stiffer, and therefore, these valves have a higher resistance to forward flow compared with equivalently sized native valves. Thus, flow velocity through a normally functioning artificial valve is generally higher than would occur through a normal native valve. However, the range of velocities through a normally functioning bioprosthesis is considerable. Both valve size and type determine the pressure gradient that one can expect in the absence of dysfunction. For example, stented bioprosthetic valves may have slightly higher gradients than mechanical valves of similar size, which tend to have higher gradients than stentless valves. For all these reasons, the range of velocities that must be considered normal varies widely among prosthetic valves. This is illustrated in Figure 14.16. In Figure 14.16A, a newly implanted St. Jude aortic prosthesis is shown. Although functioning normally by clinical criteria, the Doppler study demonstrates a maximal velocity of 290 cm/sec and a mean gradient of 20 mm Hg. Also note the distinctive “clicks” that correspond to the opening and closing of the disks. In contrast, Figure 14.16B illustrates flow through a normally functioning bioprosthetic aortic valve. In this case, no significant increase in velocity is present. Prosthetic valve clicks are not typically seen in normally functioning bioprostheses.
FIGURE 14.11. A normally functioning St. Jude aortic prosthesis. The sewing ring is indicated by the arrows. The walls of the aortic root often obscure the motion of the disks.
FIGURE 14.12. A recently implanted On-X aortic prosthesis is shown. This is a bileaflet tilting-disc valve. The arrows indicate the position of the discs and the shadowing that occurs behind the sewing ring.

Video 14-12

Blood flow within transcatheter aortic valves is similar to other aortic
bioprostheses. Mean gradients of 10 to 15 mm Hg are considered normal. An important aspect in the evaluation of TAVR valves is the detection and quantitation of valvular and paravalvular regurgitation. In this regard, valvular, or central, regurgitation is assessed similarly to surgically placed bioprosthetic valves. Paravalvular regurgitation, a more common complication of transcatheter compared to surgical valves, must be analyzed from multiple windows (Fig. 14.17). Multiple parameters, including the circumferential extent of the paravalvular flow, should be assessed and reported.

Another important difference between native and prosthetic valves is the shape and number of orifices through which forward flow occurs. As noted previously, a bileaflet tilting-disk valve has three separate orifices, a rectangular-shaped central orifice surrounded by two larger semicircular orifices (Fig. 14.18). Flow velocity is highest through the central orifice, and if this flow is sampled with continuous wave Doppler imaging, an overestimation of the true gradient can occur. This is because flow through all three orifices contributes to net gradient. By only sampling the highest velocity through the central orifice and ignoring lower-velocity flow through the other two, an overestimation of true gradient occurs. Flow through a caged-ball valve does not go through a well-defined orifice but rather goes around the periphery of the spherical occluder (Fig. 14.19). The variability and orientation of the flow complicate the Doppler interrogation of these valves. Flow through bioprostheses is often triangular in shape and may occur through an area that is significantly smaller than the sewing ring itself. Note in Figure 14.20 the position of the three struts and how they effectively form a triangular orifice, the area of which is considerably smaller than the surrounding sewing ring. All these factors contribute to the challenges inherent to assessing prosthetic valve function by any technique.
FIGURE 14.13. A normally functioning Medtronic Freestyle valve is shown in the aortic position. **A:** During systole, the valve is shown in the open position. **B:** During diastole, the cusps are barely visible. Normally functioning stentless valves appear very similar to normal native valves.

FIGURE 14.14. A normal porcine tricuspid valve prosthesis is shown from the basal short-axis (**A**) and the four-chamber (**B**) views. The arrows indicate the supporting struts.
FIGURE 14.15. A recently inserted TAVR prosthesis is provided. The surrounding stent shadows the bioprosthetic valve, making it difficult to visualize on transthoracic echocardiography.

Video 14-15

A potentially important phenomenon affecting flow through prosthetic valves involves pressure recovery. This occurs when a portion of the kinetic
energy released as blood crosses the valve is recovered in the form of pressure downstream. The amount of energy that is recovered depends on how smoothly the transition of flow occurs between the valve and the downstream conduit. For this reason, pressure recovery is most clinically relevant for a St. Jude prosthesis in the aortic position, particularly in the presence of a normal-sized aortic root. In this setting, the deceleration (and relaminarization) of blood downstream from the prosthesis is associated with a rise in pressure (i.e., pressure recovery). The net effect is the development of a high, but very localized, gradient through the central orifice of the prosthesis immediately distal to the disks (Fig. 14.21). Then, as pressure recovers (or increases) downstream, the net pressure gradient diminishes. This means that Doppler imaging, by recording the maximal velocity within the vena contracta, will demonstrate a higher gradient compared to catheter-based methods, which will be lower due to pressure recovery. Although pressure recovery is one potential explanation for a discrepancy in which Doppler imaging reports a higher gradient than catheterization, it does not imply that either method is “right” or “wrong,” rather that local changes in pressure will naturally result in differences in methodology. It should be emphasized that this higher-gradient value obtained by Doppler imaging is a real phenomenon, although less physiologically relevant than the net gradient between the left ventricle and the aorta. This concept of pressure recovery is further discussed in Chapter 8.

**FIGURE 14.16.** Doppler evaluation of a normally functioning St. Jude bileaflet prosthesis (A) and a porcine prosthesis (B). In both cases, contour of the flow signal and maximal velocity are within the expected range. Note the opening and closing valve clicks that are associated with the mechanical but not the tissue prosthesis. AV, aortic valve.
FIGURE 14.17. An example of moderate paravalvular aortic regurgitation from a patient with a recent TAVR procedure. The regurgitation (arrows) is visualized from multiple views including the long-axis (A), short-axis (B) and four-chamber (C). In the short-axis, the circumferential extent of the regurgitant flow is indicated by the arrows.

Video 14-17a
FIGURE 14.18. A transesophageal echocardiogram from a patient with a St. Jude mitral prosthesis demonstrates the appearance of the discs during diastole (A) and systole (B). This technique is ideal to record opening and closing of the hemidisks. C: Flow through one of the larger semicircular orifices is recorded using transthoracic Doppler imaging.

FIGURE 14.19. A: A Starr–Edwards mitral prosthesis (arrow). B: Doppler imaging demonstrates flow through the valve. The mean pressure gradient is approximately 10 mm Hg.
Another unique aspect of prosthetic valve function is the presence of normal, or physiologic, regurgitation. This occurs with virtually all types of mechanical prostheses and is actually part of the design of the valve. Physiologic regurgitation can be divided into two types: closure backflow and leakage. Closure backflow occurs because of the flow reversal required to close the occluding mechanism. This results in a small amount of regurgitation that ends once the occluder mechanism is seated in the sewing ring (Fig. 14.22). Leakage backflow occurs after the prosthesis has closed and is the result of a small amount of retrograde flow between and around the occluding mechanism. It is often part of the design of the prosthesis to provide a washing mechanism and prevent thrombus formation on its upstream side. Because leakage backflow may be holosystolic (or
holodiastolic, depending on valve location), it must be distinguished from pathologic regurgitation. This depends on the severity and the pattern of regurgitation. For example, leakage through a bileaflet valve often results in two symmetric narrow jets directed obliquely from the edges of the valve. This type of physiologic regurgitation is illustrated in Figure 14.23. Normal bioprosthetic valves may also exhibit mild regurgitation.

FIGURE 14.21. The concept of pressure recovery. **A:** Flow through a tapered stenosis results in significant pressure recovery downstream from the obstruction. In this case, sampling within the obstruction (SV1) yields a higher velocity compared with a sample site downstream (SV2) where pressure recovery has occurred. At this site, the recovery of pressure is associated with a lower velocity. **B:** In the absence of pressure recovery, different locations for sample volume (SV) measurement yield fairly similar velocities.
For example, some pericardial valves demonstrate mild central regurgitation that resolves 4 to 6 weeks after implantation as the cusps develop increased mobility.

Despite these differences in flow characteristics, the basic Doppler principles applied to native valves are also relevant to the study of prosthetic valves. For example, Doppler imaging can be used to measure both the maximal and mean pressure gradient across prostheses (Fig. 14.24). The assumptions that are critical to the modified Bernoulli equation apply to prosthetic valves as well. Thus, the correlation between pressure gradients obtained by the Doppler technique compared with cardiac catheterization is generally very good. However, because flow velocity through normally functioning prosthetic valves is typically low (<2.5 m/sec), the simplified Bernoulli equation, \( \Delta P = 4v_2^2 \), may lead to overestimation of the true gradient. This is due to the fact that \( v_1 \) and \( v_2 \) are similar enough that \( v_1 \) cannot be ignored, so the more complete formula, \( \Delta P = 4(v_2^2 - v_1^2) \), should be used. For practical purposes, it is sufficient to remember that flow velocity through “normal” prostheses is typically higher than native valves and a modest gradient does not necessarily imply clinically significant stenosis.
Furthermore, because of the existence of multiple jets through many types of prosthetic valves, more than one velocity pattern can often be recorded. As noted previously, the phenomenon of pressure recovery may also lead to overestimation of the pressure gradient. Figure 14.25 illustrates flow through different types of mitral prostheses. Note the variability in the contour and velocity among the four examples. Gradients across “normal” prosthetic valves vary across a wider range compared with native valves. For this
reason, it is often helpful to obtain a baseline Doppler imaging study in all patients at a time when the valve is known to be functioning normally, such as during the first postoperative clinic visit. This can then be used as a reference for future evaluations to help determine whether a given pressure gradient is normal or abnormal for the individual. In addition, tables have been published providing a range of normal values for different types of valves in the various positions.

**FIGURE 14.24.** Doppler imaging is used to record flow through an aortic prosthesis. The peak and mean gradients are indicated. Note the presence of valve clicks at the time of opening and closing.
The continuity equation can also be used to measure the EOA of prosthetic valves. The EOA is defined as the smallest cross-sectional area of the flow profile (the vena contracta) within the prosthesis. This is in contrast to the geometric orifice area (GOA), which is the area derived from the inner dimensions of the sewing ring and can be thought of as the maximum valve area assuming perfect hemodynamics. Since perfect hemodynamics do not exist and some degree of stenosis is inherent, the EOA is always smaller than the GOA, typically by 10% to 25%. As is the case with native valves, calculation of EOA for prosthetic valves offers advantages over pressure gradient alone but also has a greater potential for measurement error.

For prosthetic mitral and tricuspid valves, the pressure half-time technique has also been used to quantify the severity of stenosis. However, pressure half-time generally overestimates the valve area in the presence of a mitral prosthesis and may be more appropriate for serial evaluation. Again, having a baseline study and using the patient as his or her own control is essential for future management.
APPLICATION OF ECHOCARDIOGRAPHY TO PATIENTS WITH PROSTHETIC VALVES

In patients with prosthetic valves, the role of echocardiography begins with the initial evaluation of the patient and contributes importantly to decisions regarding the assessment of severity, the type of procedure (e.g., surgical or transcatheter), the size and type of prosthesis, and the determination of risk. Echocardiography may also be an essential element in the operating suite at the time of surgery. A comprehensive transesophageal evaluation of the diseased valve(s), if not performed previously, is essential for optimal intraoperative management. Thus, echocardiography should be used prior to valve surgery (to make decisions regarding type of prosthesis, feasibility of repair, etc.), during surgery (to assess the success and completeness of the procedure), and following surgery (to establish a new baseline and to document a successful procedure). Some of the specific indications for intraoperative transesophageal echocardiography are listed in Table 14.3. Its value in this setting is well documented. Clinical series indicate that intraoperative echo results change the operative plan in up to 15% of cases and identify a problem of sufficient magnitude to warrant revision in approximately 5% of patients. This is especially true in valve surgery, particularly valve repair procedures. As expected, the potential value of echocardiography is directly related to the complexity of the procedure. Valve repair, replacement of multiple valves, valve surgery involving complicated endocarditis, and valve replacement involving stentless valves or homografts are examples of technically challenging operative procedures where value of intraoperative echocardiography is well established.

Although specific guidelines do not exist, the utility of echocardiography during nonsurgical valve procedures, including aortic valve replacement and mitral valve repair, is also well established. As experience with TAVR has grown, the necessity of transesophageal echocardiographic guidance has declined. Currently, transesophageal echocardiography is performed during difficult deployment or when complications are suspected. At the end of the case, transthoracic echocardiography is usually adequate to confirm successful prosthesis function. In contrast, current percutaneous mitral valve procedures rely heavily on transesophageal echocardiography for device positioning and deployment, as well as determining the degree of success of
the intervention. These topics are covered in more detail later in the chapter.

### Table 14.3 INTRAOPERATIVE ASSESSMENT USING TEE

**Class I**
1. Intraoperative transesophageal echocardiography is recommended for valve repair surgery. *(Level of Evidence: B)*
2. Intraoperative transesophageal echocardiography is recommended for valve replacement surgery with a stentless xenograft, homograft, or autograft valve. *(Level of Evidence: B)*
3. Intraoperative transesophageal echocardiography is recommended for valve surgery for infective endocarditis. *(Level of Evidence: B)*

**Class IIa**
1. Intraoperative transesophageal echocardiography is reasonable for all patients undergoing cardiac valve surgery. *(Level of Evidence: C)*


Following discharge, the role of echocardiography consists of defining baseline function and serial assessment for evidence of dysfunction. Both American College of Cardiology/American Heart Association Management Guidelines and Appropriate Use Criteria have been published to provide guidance in this area (*Table 14.4*). There is general consensus that echocardiography should be performed soon after valve surgery as part of the initial evaluation of the patient during the recovery phase. This serves to establish a baseline, with which future studies can be compared. This examination should focus on an assessment of left and right ventricular function, determination of pulmonary artery pressure, and, of course, a thorough evaluation of the repaired or replaced valve. Because all prosthetic valves have some degree of obstruction, a critical part of the evaluation is to determine the pressure gradient and EOA. Careful assessment of regurgitation is also important. Mild valvular regurgitation is normally
present in many prosthetic valves. On the other hand, paravalvular regurgitation is an abnormal finding that requires thorough assessment and follow-up. Thus, the initial postoperative echocardiogram should clearly document the presence and severity of regurgitation and differentiate normal from abnormal forms.

Following this initial echocardiographic study, subsequent assessment must be individualized. According to the guidelines, there is agreement that echocardiography should be considered if there is a change in clinical status, evidence of infection, or reason to suspect valve dysfunction. Routine (e.g., annual) echocardiographic studies, in the absence of one of the indications listed in the guidelines, are not recommended. However, once dysfunction is documented, serial evaluation, including clinical and echocardiographic monitoring, should be undertaken. This would include, for example, patients with bioprosthetic valves that exhibit early signs of primary tissue degeneration. Finally, in children who are still growing, the possibility of developing prosthesis–patient mismatch mandates, particularly, close follow-up. This occurs because the EOA of the prosthesis remains fixed while the child’s stroke volume increases with age. Monitoring for worsening hemodynamics as a result of normal growth is essential.

<table>
<thead>
<tr>
<th>Number</th>
<th>Indication</th>
<th>Score (1–9)</th>
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<tbody>
<tr>
<td>47</td>
<td>Initial postoperative evaluation of prosthetic valve for establishment of baseline</td>
<td>A (9)</td>
</tr>
<tr>
<td>48</td>
<td>Routine surveillance (&lt;3 yrs after valve implantation) of prosthetic valve if no known or suspected valve dysfunction</td>
<td>I (3)</td>
</tr>
<tr>
<td>49</td>
<td>Routine surveillance (≥3 yrs after valve implantation) of prosthetic valve if no known or suspected valve dysfunction</td>
<td>A (7)</td>
</tr>
<tr>
<td>50</td>
<td>Evaluation of prosthetic valve with suspected dysfunction or a change in clinical status or cardiac exam</td>
<td>A (9)</td>
</tr>
<tr>
<td>51</td>
<td>Reevaluation of known prosthetic valve dysfunction when it would change management or guide therapy</td>
<td>A (9)</td>
</tr>
<tr>
<td></td>
<td>Description</td>
<td>Evidence Level</td>
</tr>
<tr>
<td>---</td>
<td>-----------------------------------------------------------------------------</td>
<td>----------------</td>
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<tr>
<td>52</td>
<td>Initial evaluation of suspected infective endocarditis with positive blood cultures or a new murmur</td>
<td>A (9)</td>
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<td>I (2)</td>
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<tr>
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<td>I (3)</td>
</tr>
<tr>
<td>55</td>
<td>Reevaluation of infective endocarditis at high risk for progression or complication or with a change in clinical status or cardiac exam</td>
<td>A (9)</td>
</tr>
<tr>
<td>56</td>
<td>Routine surveillance of uncomplicated infective endocarditis when no change in management is contemplated</td>
<td>I (2)</td>
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**TTE for evaluation of intracardiac structures and chambers**

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<td>Suspected cardiac mass</td>
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</table>

**TEE as initial or supplemental test**

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<th></th>
<th>Description</th>
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</tr>
</thead>
<tbody>
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<td>Use of TEE when there is a high likelihood of a nondiagnostic TTE due to patient characteristics or inadequate visualization of relevant structures</td>
<td>A (8)</td>
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<td>103</td>
<td>Guidance during percutaneous noncoronary cardiac interventions including but not limited to closure device placement and percutaneous valve procedures</td>
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FIGURE 14.26. Transesophageal three-dimensional echocardiography is useful to visualize prosthetic valves. In this example, the data set are rendered to show the bileaflet disc valve (arrow) from the left atrial perspective, sometimes called the “surgeon’s view.”

GENERAL APPROACH TO PROSTHETIC VALVES

Transthoracic two-dimensional imaging is generally adequate to distinguish among the various types of prosthetic valves and to assess their function. However, the high reflectance of the prosthetic material creates challenges for the echocardiographer. Because the speed of sound changes as it passes through prosthetic materials, size and appearance can be distorted. Some decrease in gain setting is generally necessary to compensate for these differences. The high reflectance also leads to shadowing behind the prostheses. Reverberations frequently appear behind the prosthetic structures which may obscure targets of interest. To overcome these problems, multiple echocardiographic windows must be used to fully interrogate the areas around prosthetic valves. A thorough anatomic assessment is also facilitated by the use of three-dimensional techniques. For example, a properly oriented three-dimensional image will provide a complete, circumferential view of a sewing ring, so that any abnormal masses that might be present will be
visualized. In other cases, transesophageal echocardiography, often with three-dimensional imaging, will be necessary to provide a thorough examination (Fig. 14.26). In this example, by displaying en face views of the mitral prosthesis, disc motion and sewing ring integrity can be evaluated.

The two-dimensional echocardiographic appearance of bioprosthetic leaflets more closely approximates that of native valves. In fact, newer stentless aortic prostheses can be nearly indistinguishable from a normal native aortic valve. For stented valves, imaging is ideally performed with the ultrasound beam aligned parallel to flow to avoid the shadowing effects of the stents and sewing ring. The leaflets themselves are quite similar to native valve tissue, both in texture and excursion. Over time, bioprostheses tend to thicken and become fibrotic, leading to increased echogenicity and reduced excursion on two-dimensional imaging (Fig. 14.27). Such valves can become stenotic and/or regurgitant. This illustration demonstrates a brittle, fibrotic porcine mitral valve with partial rupture of one cusp leading to severe mitral regurgitation. In all cases, a combination of two-dimensional and Doppler imaging is required to thoroughly assess bioprosthetic valves (Fig. 14.28).

For the reasons noted above, mechanical valves can be quite difficult to assess with two-dimensional echocardiography. Although gross abnormalities can be detected, more subtle changes are often missed, especially with transthoracic imaging. The primary goals of two-dimensional echocardiography in this setting are to confirm stability of the sewing ring, determine the specific type of prosthesis, confirm the opening and closing motion of the occluding mechanism, and evaluate for gross structural abnormalities such as vegetations and thrombi. Assessing the mobility of the occluding mechanism can be difficult. However, through careful interrogation, the rapid motion of the leading edge of the disk or ball generally can be recorded. In normal prostheses, the motion is brisk and consistent with each beat (Figs. 14.8 to 14.12). M-mode imaging can be useful in this case to more precisely define the brisk opening and closing and the degree of excursion of the occluder. For bileaflet prostheses, it is important to search for both hemidisks, which often have slightly out-of-phase motion as they open and close in close proximity (Figs. 14.9 and 14.18).
FIGURE 14.27. An example of primary tissue degeneration involving a porcine mitral valve. The leaflets are thickened and fibrotic with decreased mobility (left). Right: Color Doppler imaging demonstrates severe mitral regurgitation with an eccentric jet (arrows).
FIGURE 14.28. A 14-year-old bioprosthetic mitral valve is characterized by leaflet thickening and calcification (A). Continuous wave Doppler recorded from the two-chamber view shows evidence of increased gradient (B).

Video 14-28

As with two-dimensional imaging, the Doppler examination also faces unique challenges in the setting of a prosthetic valve. Because of the variability of flow through and around the different prostheses, color flow imaging is often helpful to define the location and direction of the various flow patterns. Some prosthetic valves have more than one orifice and, consequently, a complex flow profile. Once the desired flow patterns are localized with color flow imaging, pulsed and continuous wave Doppler imaging can be oriented to quantify flow velocity. As already noted, velocities will always tend to be higher through prosthetic valves, depending in part on the size of the specific prosthesis. Whenever velocity is higher than expected, consider the possibility of pressure recovery, as discussed previously.

Assessing valvular regurgitation is primarily limited by the shadowing effect of the prosthetic valve itself. Because the signal-to-noise ratio for Doppler imaging is lower compared with two-dimensional echocardiographic imaging, the shadowing effect is even more pronounced and the ability to record a Doppler signal “behind” a prosthetic valve is very limited. Multiple views must be used to fully interrogate the regurgitant signal. Figure 14.29 demonstrates how the shadowing effect of an aortic prosthesis obscures the left atrium from the parasternal window. It is also important to distinguish
transvalvular from paravalvular regurgitation. This is best accomplished using transesophageal color flow imaging to interrogate the circumference of the sewing ring on the upstream side of the valve (Fig. 14.30). With the increased sensitivity of modern equipment, a small amount of paravalvular regurgitation may be recorded in the immediate postoperative period that will often disappear or diminish over time (Fig. 14.31). Three-dimensional transesophageal imaging will likely prove to be the most sensitive method for this determination. Figure 14.32 is an example of paravalvular regurgitation of a mechanical mitral prosthesis recorded with real-time three-dimensional imaging. In this example, both disc motion and the location and extent of the regurgitation are demonstrated. One advantage of this approach is the ability to distinguish flow through the various orifices of a mechanical prosthesis. Spectral Doppler recordings of prosthetic valve flow will also include brief, high-velocity signals referred to as “clicks.” These are intense recordings associated with both the opening and closing of the occluder mechanism. They provide useful information on timing and are particularly helpful to identify the various phases of filling and ejection. In Figure 14.33, both normal and abnormal St. Jude aortic prostheses are shown. In Figure 14.33A, note the valve clicks marking opening and closing of the normal valve. Figure 14.33B is taken from a patient with a prosthesis that is partially obstructed by a thrombus on the sewing ring. Note that the opening valve click is absent, and the closing click is very faint. The high velocity is evidence of the increased pressure gradient across the partially obstructed valve.

PROSTHETIC AORTIC VALVES

Transthoracic M-mode and two-dimensional echocardiography have relatively low sensitivity for detecting dysfunction of aortic prostheses. Gross abnormalities, such as valve dehiscence or large thrombi or vegetations, can be identified using two-dimensional echocardiography. Thickened and fibrocalcific leaflets of bioprostheses can also be visualized, but assessing the functional significance of such changes is difficult. Thus, most of the diagnostic information related to aortic prostheses depends on a thorough and quantitative Doppler study. Both the peak instantaneous and mean pressure
Gradients across the prosthesis should be recorded from multiple views. The correlation between Doppler gradients and values obtained with cardiac catheterization is quite high, especially when the tests are performed simultaneously. Agreement between Doppler imaging and catheterization tends to be highest for mean gradient. The correlation between the two techniques for peak gradient is not as good, likely due to the inherent differences between peak instantaneous and peak-to-peak gradients.

**FIGURE 14.29.** The presence of a St. Jude aortic prosthesis (arrows) creates a pattern of reverberations that extends into the left atrium. This creates a shadowing effect and can obscure the presence of mitral regurgitation.
The range of normal values depends primarily on the size of the prosthesis (Table 14.5). For example, a 29-mm St. Jude aortic prosthesis will generally have a maximal velocity of less than 2.5 m/sec, whereas a normally functioning 19-mm St. Jude prosthesis may have a maximal velocity as high as 4 to 4.5 m/sec. Consequently, the mean gradient across a 19-mm valve is roughly twice the mean gradient across a 29-mm prosthesis of the same type. Differences among the various types of prosthetic valves (assuming a similar size) are much less. The exceptions to this are aortic homografts and stentless valves, which consistently have lower gradients and exhibit hemodynamics that more closely approximate the native valve.
FIGURE 14.31. This St. Jude mitral prosthesis was evaluated in the operating room immediately after implantation when a mild degree of paravalvular regurgitation may be present. In most cases, this resolves over time. Color Doppler imaging indicates both central and peripheral jets, consistent with mild mitral regurgitation.

When the continuity equation is used to estimate the EOA of a prosthetic valve, it should be remembered that this area corresponds to the vena contracta of flow rather than the actual orifice. The equation itself is identical to the one used in the setting of native valve stenosis (Fig. 14.34). If the outflow tract dimension cannot be accurately measured, some investigators suggest substituting the sewing ring outer diameter for this value. Again, the most important point is that the Doppler recording and the diameter measurement be obtained at the same level. Figure 14.35 is an example of a stenotic bioprosthetic aortic valve.
FIGURE 14.32. An example of paravalvular regurgitation in a bileaflet disc mitral valve. Transesophageal three-dimensional echocardiography is ideal to identify the location and extent of the flow (arrows).
FIGURE 14.33. Examples of flow through two different St. Jude aortic prosthetic valves. **A:** Flow velocity is normal and crisp valve clicks are present. **B:** Jet
velocity is increased, indicating a peak pressure gradient of approximately 77 mm Hg. Valve clicks, especially at the time of valve opening, are diminished.

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FIGURE 14.34. The continuity equation can be used to calculate the effective valve area across prostheses. **A:** The diameter of the left ventricular outflow tract is measured. **B:** Time velocity integral (TVI) of the outflow tract is calculated using planimetry. **C:** Using continuous wave Doppler imaging, flow through the prosthetic valve is recorded. Because of a hyperdynamic left ventricle, the TVI\textsubscript{OT} and the maximal pressure gradient are quite high. Despite the maximal gradient of 65 mm Hg, the aortic valve area is approximately 1.9 cm\textsuperscript{2}. The calculations used to measure valve area are provided. AVA, aortic valve area; CSA, cross-sectional area; D\textsubscript{LVOT}, left ventricular outflow tract diameter.

\[
\begin{align*}
\text{CSA}_{\text{OT}} &= 0.785 \times D^2 = 3.8 \, \text{cm}^2 \\
\text{AVA} &= \frac{\text{CSA}_{\text{OT}} \times \text{TVI}_{\text{OT}}}{\text{TVI}_{\text{AV}}} \\
\text{AVA} &= \frac{3.8 \times 34}{69} = 1.87 \, \text{cm}^2
\end{align*}
\]
FIGURE 14.35. The leaflets of a stenotic bioprosthetic aortic valve appear thick, immobile, and echogenic as seen from the long-axis view in this example (A). Doppler is essential to quantify the degree of obstruction and to follow changes over time (B).

The Doppler velocity index (DVI) is a simple and useful alternative for evaluating stenosis, particularly involving the aortic valve. DVI is dimensionless and is calculated as the ratio between the outflow tract peak velocity (OT) and the maximal velocity through the prosthesis (PV):

\[ \text{DVI} = \frac{V_{\text{OT}}}{V_{\text{PV}}} \]  

[Eq. 14.1]

In the absence of any gradient, the two velocities would be the same, yielding a ratio of 1. Because all prostheses are somewhat stenotic, a DVI of less than 1 is consistently obtained. The expected range for normally functioning aortic prostheses is 0.3 to 0.5. Although this dimensionless number has limited utility in isolation, it can be obtained reproducibly and provides a useful parameter to detect changes over time. In addition, it avoids the challenges of measuring the outflow tract diameter, as described above.

Assessing regurgitation is similar in prosthetic and native aortic valves with two exceptions. First, it must be remembered that some degree of regurgitation is a normal finding for most prostheses. Distinguishing physiologic from pathologic regurgitation is generally a matter of degree. Second, shadowing from the prosthesis can obscure significant regurgitant jets, mandating the use of multiple windows (and often transesophageal echocardiography) to completely interrogate the left ventricular outflow tract. However, this is far less a problem for aortic prostheses (compared to mitral)
and in most cases, transthoracic imaging is adequate to characterize prosthetic aortic regurgitation (Fig. 14.36). Distinguishing valvular from paravalvular regurgitation is also important. Using either the transthoracic or the transesophageal approach, a short-axis view at and immediately below the level of the sewing ring often allows this distinction to be made (Fig. 14.37). For most patients, however, transesophageal imaging is a more accurate way of detecting the presence and extent of paravalvular regurgitation. Figure 14.38 demonstrates mild paravalvular regurgitation associated with a stentless aortic prosthesis.

**FIGURE 14.36.** A bioprosthetic aortic valve and a mitral annuloplasty ring are demonstrated in this study from a patient with a dilated cardiomyopathy. Both aortic valve prosthesis and the mitral ring are apparent in the long-axis view (A). Color Doppler (B) reveals mild aortic regurgitation from the four-chamber view (B, arrow).
**FIGURE 14.37.** An example of an aortic root abscess. **A:** In the short-axis view, an echo-free space is seen posterior to the aortic root (arrows). **B:** Color Doppler imaging demonstrates flow within the abscess cavity (arrows) and associated paravalvular regurgitation.

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**TRANSCATHETER AORTIC VALVES**

Echocardiographic assessment of transcatheter aortic valves has several components. During the procedure, transesophageal echocardiography may be used to guide position of the device prior to deployment (Fig. 14.39). Echocardiography, along with fluoroscopy, assures that the device is neither too low, where it can result in paravalvular regurgitation, mitral valve interference, or conduction abnormalities, nor too high, where regurgitation,
coronary ostia obstruction, or device embolization can occur. Once deployed, echocardiography is used to assure proper seating of the valve, normal motion of the leaflets, and to assess for both valvular and paravalvular regurgitation (Fig. 14.40). As experience with TAVR has grown and conscious sedation has replaced general anesthesia for many cases, the routine use of transesophageal echocardiography has declined. Instead, postprocedure transthoracic echocardiography is typically performed to confirm procedure success.

Patients should undergo a postprocedure transthoracic echocardiography prior to hospital discharge. Important data to be recorded on this study include mean and peak gradients, EOA, and the presence and severity of central and paravalvular regurgitation (Fig. 14.41). In addition, assessing for mitral regurgitation is also important. Stent interference, or even perforation, of the anterior mitral leaflet can occur, leading to significant mitral regurgitation.

When determining EOA, proper placement of the Doppler sample volume for outflow tract velocity is critical. It should be positioned just below (i.e., proximal to) the lower edge of the stent. If the sample volume is within the stent, some degree of flow acceleration may be present, leading to overestimation of EOA (potentially underestimating any degree of prosthesis dysfunction). Normally functioning TAVR valves will usually have a mean gradient of 10 to 15 mm Hg and a calculated EOA of 1.3 to 1.8 cm². Aortic regurgitation, especially paravalvular, should be carefully and thoroughly evaluated. Paravalvular aortic regurgitation may involve multiple eccentric jets and requires a comprehensive approach, including color and continuous wave Doppler, descending aortic flow, pressure half-time determination, and, if possible, determination of regurgitant volume. A recommended approach to paravalvular regurgitation involves the measurement of the circumferential extent of the color flow disturbance, mapped from the basal short-axis view. If more than 30% of the annular circumference is involved, severe regurgitation is present. Clinical trials have demonstrated the important impact aortic regurgitation severity has on patients who have undergone TAVR. Precise assessment, however, remains challenging and an area of active study. Other complications may occur post procedure. Figure 14.42 depicts a perforated anterior mitral valve leaflet due to erosion from a malpositioned transcatheter valve. This resulted in severe mitral
regurgitation.

FIGURE 14.38. A: A transesophageal echocardiogram from a patient with a stentless aortic prosthesis. B: A mild degree of paravalvular aortic regurgitation (arrow) is demonstrated using color Doppler imaging.

FIGURE 14.39. Transesophageal echocardiography is often used to guide the insertion of a transcatheter aortic valve. In A, a long-axis view helps to properly
position the catheter containing the device (arrow) prior to deployment. In B, a short-axis image shows the deployed prosthesis within the aortic annulus. This view is used to ensure proper expansion and to look for regurgitation. In C, the long-axis view is shown following deployment, demonstrating the position of the lower end of the stent relative to the annulus and the anterior mitral leaflet.

FIGURE 14.40. Intraprocedural guidance during TAVR implantation is demonstrated. In A, the device (arrow) has just been deployed within the aortic valve. The short-axis view (B) shows underexpansion of the stent within the annulus, with resulting paravalvular aortic regurgitation (C, arrows). Following further balloon dilation (D), proper expansion and positioning of the prosthetic valve are shown.

FIGURE 14.41. An example of paravalvular aortic regurgitation (arrows) following
TAVR implantation is shown from the long-axis (A) and short-axis (B) views.

Video 14-41b

PROSTHETIC MITRAL VALVES

Visualizing mitral prostheses with transthoracic echocardiography is somewhat easier than visualizing aortic prostheses. This is because the prosthetic mitral valve is seated within the mitral annulus and can be easily visualized from both the parasternal and apical windows. In contrast, aortic prostheses may be partially obscured by the walls of the aorta (from the parasternal view) and by the prostheses itself from the apical view. Evaluating the stability of the mitral prosthesis, excluding dehiscence, and visualizing the motion of leaflets or the occluding mechanism are generally possible with transthoracic imaging.
FIGURE 14.42. A complication of TAVR implantation is demonstrated. The valve was deployed too low relative to the aortic annulus, resulting in the lower end of the wire stent impinging on the anterior mitral leaflet (A). This led to a perforation of the mitral leaflet, seen in the long axis (A, arrow). With three-dimensional echocardiography (B), an en face view of the perforated anterior leaflet is shown (arrows). In C, color Doppler reveals severe mitral regurgitation through the perforation, requiring surgical repair.

Video 14-42a
FIGURE 14.43. Examples of normally functioning (A, B) and stenotic (C, D) bioprosthetic mitral valves. In A, transesophageal echocardiography demonstrates mild thickening but normal motion of the leaflets. Absence of any significant gradient is confirmed using pulsed Doppler (B). The second patient demonstrates severe thickening, calcification, and reduced leaflet mobility (C). With spectral Doppler, severe stenosis is demonstrated with a 31 mm Hg mean mitral gradient.
Video 14-43c

Using Doppler imaging, the antegrade flow through the prosthesis can be accurately recorded (Fig. 14.43). Normal values for the various types and sizes of mitral prosthetic valves are provided in Table 14.6. The mean mitral pressure gradient is derived by planimetry of the mitral envelope, taking care to align the Doppler beam as close as possible to the direction of inflow (Figs. 14.28 and 14.44). Because of the orientation of the prosthesis and the resulting transprosthesis flow direction, nonstandard views may be necessary for optimal alignment of the Doppler beam. Note in Figure 14.44 that the mitral flow recording is obtained from the parasternal long-axis view. The pressure half-time method can also be performed in the setting of prosthetic valves. With native valves, it was empirically determined that mitral valve area was approximated by the equation:

\[
\text{MV area} = \frac{220}{P_{\frac{1}{2}}t} \quad \text{[Eq. 14.2]}
\]

When the same approach is applied to prosthetic valves, the formula tends to overestimate the EOA. Despite this limitation, prolongation of the pressure half-time, especially when a baseline has been established, is a reliable marker of obstruction and is less flow-dependent than gradient alone. In most patients, both mean gradient and pressure half-time should be assessed to determine whether prosthetic valve stenosis is present. Alternatively, the continuity equation can be applied (in the absence of mitral regurgitation) according to the following formula in which MV is the mitral valve, LVOT is the left ventricular outflow tract, and TVI is the time velocity integral:
FIGURE 14.44. Doppler recording of flow through a porcine mitral valve. Both the peak and mean gradients are derived by planimetry. Note that the recording was obtained from the parasternal window. In this case, this view provided optimal alignment with mitral inflow.

Table 14.6  
RANGE OF NORMAL VALUES FOR DOPPLER EVALUATION OF MITRAL PROSTHESES

<table>
<thead>
<tr>
<th>Category</th>
<th>Specific Type</th>
<th>Size (mm)</th>
<th>Gradient (mm Hg)</th>
<th>Peak Velocity (m/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stentless</td>
<td>Biocor</td>
<td>27</td>
<td>13 ± 1</td>
<td>13 ± 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>29</td>
<td>14 ± 2</td>
<td>14 ± 2</td>
</tr>
<tr>
<td>Type</td>
<td>Model</td>
<td>n</td>
<td>Isovolumic (cm/s)</td>
<td>Transvalvular (cm/s)</td>
</tr>
<tr>
<td>--------------------</td>
<td>------------------------------</td>
<td>----</td>
<td>------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td>Bioprosthetic stented</td>
<td>Carpentier-Edwards</td>
<td>27</td>
<td>6 ± 2</td>
<td>98 ± 28</td>
</tr>
<tr>
<td></td>
<td>Hancock I</td>
<td>29</td>
<td>5 ± 2</td>
<td>92 ± 14</td>
</tr>
<tr>
<td></td>
<td></td>
<td>31</td>
<td>4 ± 2</td>
<td>92 ± 19</td>
</tr>
<tr>
<td></td>
<td></td>
<td>33</td>
<td>6 ± 3</td>
<td>93 ± 12</td>
</tr>
<tr>
<td></td>
<td>Ionescu–Shiley</td>
<td>27</td>
<td>2 ± 1</td>
<td>115 ± 20</td>
</tr>
<tr>
<td></td>
<td></td>
<td>29</td>
<td>2 ± 1</td>
<td>95 ± 17</td>
</tr>
<tr>
<td></td>
<td></td>
<td>31</td>
<td>2 ± 1</td>
<td>90 ± 12</td>
</tr>
<tr>
<td></td>
<td>Tilting disk</td>
<td>25</td>
<td>5 ± 1</td>
<td>93 ± 11</td>
</tr>
<tr>
<td></td>
<td>Omnicarbon</td>
<td>27</td>
<td>3 ± 1</td>
<td>100 ± 28</td>
</tr>
<tr>
<td></td>
<td>Björk–Shiley</td>
<td>29</td>
<td>2 ± 1</td>
<td>85 ± 8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>31</td>
<td>4 ± 1</td>
<td>100 ± 36</td>
</tr>
<tr>
<td>Bileaflet</td>
<td>St. Jude Medical</td>
<td>25</td>
<td>6 ± 2</td>
<td>102 ± 16</td>
</tr>
<tr>
<td></td>
<td>Carbomedics</td>
<td>27</td>
<td>5 ± 2</td>
<td>105 ± 33</td>
</tr>
<tr>
<td></td>
<td></td>
<td>29</td>
<td>5 ± 2</td>
<td>120 ± 40</td>
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<tr>
<td></td>
<td></td>
<td>31</td>
<td>4 ± 1</td>
<td>134 ± 31</td>
</tr>
<tr>
<td>Caged ball</td>
<td>Starr–Edwards</td>
<td>28</td>
<td>3 ± 1</td>
<td>99 ± 27</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30</td>
<td>3 ± 1</td>
<td>89 ± 28</td>
</tr>
<tr>
<td></td>
<td></td>
<td>32</td>
<td>3 ± 1</td>
<td>79 ± 17</td>
</tr>
<tr>
<td></td>
<td></td>
<td>33</td>
<td>2 ± 2</td>
<td>70 ± 14</td>
</tr>
</tbody>
</table>

Detecting regurgitation through or around a mitral prosthesis using transthoracic echocardiography is limited by the shadowing effect of the prosthetic material. Whether imaging is performed from the parasternal or the apical view, the prosthetic valve will always obscure a portion of the left atrium so that the sensitivity of this method is reduced (Figs. 14.45 and 14.46). In the presence of both aortic and mitral prostheses, most of the left atrium is shadowed and the detection of mitral regurgitation in such patients is very limited. In contrast, the transesophageal approach offers an excellent opportunity to assess the entire left atrium in the presence of prosthetic valves (Fig. 14.47). Differentiating between physiologic and pathologic mitral regurgitation is based on a variety of factors. Using the transesophageal approach, some degree of regurgitation is detected in as many as 90% of normally functioning mitral prostheses. Characteristics of “normal” prosthetic regurgitation include a jet area less than 2 cm$^2$ and a jet length less than 2.5 cm. In addition, the patterns of regurgitant flow are typical for each individual prosthesis. For example, a St. Jude mitral prosthesis often displays one central and two peripheral small jets, whereas a Medtronic-Hall valve typically has a single central regurgitant jet. Transesophageal echocardiography is also well suited for distinguishing valvular from paravalvular regurgitation. An example of how transesophageal three-dimensional imaging can be used for this purpose is provided in Figure 14.48. In this case, two-dimensional color flow imaging demonstrates regurgitant flow originating in the area of the sewing ring. In three-dimensional views, the spatial orientation provided by this approach permits the origin of the regurgitant jet to be precisely located outside of the ring, confirming the presence of paravalvular regurgitation.
Mitral valve repair typically involves a combination of leaflet resection, chordal reimplantation, and placement of an annular ring. An example of a successfully repaired mitral valve is shown in Figure 14.49. Note that the posterior leaflet is relatively fixed by virtue of the position of the ring. In Figure 14.50, a recently repaired mitral valve demonstrates a significant gradient on Doppler.

Transcatheter Mitral Valve Repair

Percutaneous repair of mitral regurgitation can be performed using nonsurgical techniques, including the MitraClip procedure. This device is currently approved in the United States for treatment of moderately severe or severe primary mitral regurgitation. Other transcatheter therapies for treatment of both mitral stenosis and regurgitation are being developed but currently still considered investigational. In virtually every aspect of this procedure, from patient selection to follow-up, echocardiography plays a key role. The use of echocardiography to determine the cause (primary vs. secondary, or functional) and severity of mitral regurgitation is covered in detail in Chapter 11. In addition, however, echocardiography, and particularly transesophageal echocardiography with three-dimensional imaging, is essential to fully characterize the anatomic detail in order to plan and execute a transcatheter repair. Specifically, the location of the prolapsed and/or flail leaflet scallops must be determined prior to the procedure. In addition, certain
specific measures are necessary to ensure that the patient is an acceptable MitraClip candidate. These include the involvement of the A2/P2 segments, precise measures of coaptation length and depth, leaflet length, and mitral valve orifice area.

During the procedure, transesophageal echocardiography is essential at each step, including guiding the optimal location for transseptal puncture, advancing the catheter into the left atrium, and positioning the delivery system within the mitral valve. During this step, multiple imaging planes are used to assure that the device arms are at the proper depth between the leaflets and that the device is rotated so that it is correctly positioned relative to the two leaflets and the coaptation plane (Fig. 14.51).

![FIGURE 14.46. A: Despite the presence of a prosthetic mitral valve, paravalvular mitral regurgitation was detected on this transthoracic study. B: The eccentric regurgitant jet (arrow) can be seen along the anterior wall of the left atrium.](image_url)
FIGURE 14.47. Transesophageal echocardiography is superior to transthoracic imaging to detect prosthetic mitral regurgitation. A: Poor image quality and the shadowing effect of the St. Jude prosthesis prevent mitral regurgitation from being detected on this transthoracic study. B: The proximity of the left atrium to the transesophageal probe facilitates diagnosis of mitral regurgitation (arrow).

FIGURE 14.48. Transesophageal three-dimensional imaging is useful to distinguish valvular from paravalvular regurgitation. In this example, the two-dimensional echocardiogram demonstrated mitral regurgitation in the vicinity of the sewing ring of a St. Jude prosthesis (A). B: Using transesophageal three-dimensional color Doppler imaging, the location of the regurgitant jet outside of the sewing ring (small arrows) is clearly demonstrated. The asterisk identifies the center of the disk structure.
FIGURE 14.49. An example of a successfully repaired mitral valve is shown. From the long-axis view (A), the annuloplasty ring is evident which typically results in some restriction of posterior leaflet excursion. Anterior leaflet motion is normal, ruling out any significant stenosis. This is confirmed with pulsed Doppler (B).
FIGURE 14.50. A known complication of mitral valve repair is the creation of a degree of mitral stenosis. In A, the ring has greatly restricted posterior leaflet motion. Anterior leaflet motion is also limited, resulting in a reduced mitral orifice (B) and mild stenosis, as shown by Doppler (C).
Once properly positioned, echo is used to monitor successful grasping of two opposing scallops. Then, as the clip is gradually closed, continued echocardiographic monitoring is necessary to assure that leaflet insertion into the clips is maintained and that the severity of mitral regurgitation is reduced. This is done prior to final closing and releasing of the MitraClip (Fig. 14.52). Once the clip is released, a final step is the determination of the severity of residual regurgitation and the gradient across the two resulting orifices. Grading mitral regurgitation severity after MitraClip is challenging. Typically, multiple jets may be present and each must be evaluated separately (Fig. 14.53). Shadowing from the clip that may obscure the vena contracta and altered hemodynamics resulting from general anesthesia all contribute to
these challenges. As is shown in this example, three-dimensional imaging can be extremely useful for this purpose.

**SPECIFIC CAUSES OF DYSFUNCTION**

**Obstruction**

The various categories of prosthetic valve complications are listed in Table 14.7. Obstruction to antegrade flow through a prosthetic valve has several possible causes. As has been mentioned previously, all prostheses are inherently stenotic, demonstrating a wide range of pressure gradients that depend on prosthesis size and stroke volume. Thus, a common cause of obstruction results from a mismatch between the valve and the patient. In this situation, the prosthesis functions as intended but is too small to accommodate the necessary flow. When the EOA is small relative to the patient’s body surface area, hemodynamic abnormalities occur. This results in the generation of a significant pressure gradient across the valve. A common reason for prosthesis–patient mismatch occurs in young patients who outgrow their prosthetic valve. In other words, the prosthesis is properly sized for a child but becomes gradually stenotic over time as the child outgrows it. Small patients, especially women, are prone to this condition because of the necessity to implant small prostheses that result in suboptimal hemodynamics. Figure 14.54 is taken from a 32-year-old woman who had undergone aortic valve replacement 4 years previously with a #21 bileaflet prosthesis. Symptoms of dyspnea had improved after her surgery but then became much worse when she became pregnant. Despite normal motion of the hemidisks, a 45 mm Hg mean gradient is demonstrated.

In other cases, for technical reasons, a prosthetic valve that is too small is implanted and the patient is left with a significant transvalvular gradient. A form of prosthesis–patient mismatch often involves a prosthetic valve that functions adequately at rest but is unable to accommodate the hemodynamic demands of exercise. Distinguishing mismatch dysfunction from other causes of acquired prosthesis obstruction can be difficult. The diagnosis depends on a careful assessment of prosthesis function, knowledge of the prosthesis size relative to the patient, a quantitative evaluation of stroke volume, and a careful search to exclude other causes of prosthesis dysfunction. It should be
pointed out that a high flow velocity alone is not proof of an obstructed prosthesis. A high cardiac output and/or severe regurgitation are additional causes of increased velocity without obstruction.

**FIGURE 14.51.** Transcatheter repair of mitral regurgitation using the MitraClip device requires transesophageal echocardiographic guidance. Advancing the device (*arrow*) through the atrial septum is the first step requiring echo guidance. In **A**, the optimal location for transseptal puncture is essential to allow the device (*arrow*) to be subsequently steered through the mitral valve. In **B**, the long-axis view is then used to advance the device (*arrow*) into the mitral orifice. In **C**, the device has been properly oriented relative to the leaflets and the arms are open to grasp the free edge of both leaflets. In **D**, successful grasping of both leaflets (*arrows*) is confirmed.
FIGURE 14.52. Transesophageal echocardiography is essential for positioning the MitraClip device so that both leaflets can be grasped and secured (A). Before release, color Doppler is used to assess the degree of residual mitral regurgitation (B).
Video 14-52b
FIGURE 14.53. Transesophageal echocardiography demonstrates the etiology and severity of mitral regurgitation prior to transcatheter repair. In A, posterior leaflet prolapse with a partially flail segment is shown (arrow). The severity of mitral regurgitation is assessed using multiple criteria (B, C). Following MitraClip placement, three-dimensional echocardiography shows the clip securing the A2 and P2 segments in both diastole (D) and systole (E). Color Doppler is then used to demonstrate antegrade flow through the two orifices (F), as well as any residual regurgitation. Minimal residual mitral regurgitation is confirmed with color Doppler (G).
### Table 14.7

<table>
<thead>
<tr>
<th>Type of Complication</th>
<th>Example</th>
<th>Role of Echocardiography</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary mechanical</td>
<td>Ball variance</td>
<td>Visualize structure; assess gradient and regurgitation</td>
</tr>
<tr>
<td>failure</td>
<td>Strut fracture</td>
<td></td>
</tr>
<tr>
<td>Nonstructural</td>
<td>Patient–prosthesis mismatch</td>
<td>Valve gradient (change over time); visualize tissue in and around sewing ring</td>
</tr>
<tr>
<td>dysfunction</td>
<td>Ingrowth of pannus</td>
<td></td>
</tr>
<tr>
<td>Bleeding event</td>
<td>Intracranial hemorrhage</td>
<td>Source of embolus; presence and mobility of masses</td>
</tr>
<tr>
<td>Condition</td>
<td>Description</td>
<td>Diagnosis/Assessment</td>
</tr>
<tr>
<td>--------------</td>
<td>------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>Vegetation</td>
<td>Detect mass consistent with vegetation</td>
</tr>
<tr>
<td></td>
<td>Abscess</td>
<td>Visualize area around sewing ring, echo-dense or echo-lucent area; paravalvular regurgitation</td>
</tr>
<tr>
<td></td>
<td>Dehiscence</td>
<td>Detect rocking motion; paravalvular regurgitation</td>
</tr>
<tr>
<td>Thrombosis</td>
<td>Thrombus impedes opening/closing of occluder mechanism</td>
<td>Visualize and localize mass; assess gradient; detect regurgitation; assess disc motion</td>
</tr>
<tr>
<td>Embolism</td>
<td>Stroke</td>
<td>Identify and characterize source of embolus</td>
</tr>
</tbody>
</table>

**FIGURE 14.54.** A significant increase in gradient through a bileaflet disc aortic valve occurred in this woman during pregnancy. Despite normal appearance of the prosthesis on two-dimensional imaging (A, B), Doppler reveals a 45 mm Hg mean gradient (C).
coming soon

Video 14-54a

coming soon

Video 14-54b
Intraoperative transesophageal echocardiography can be useful to identify technical problems related to prosthesis insertion. In this example, one of the hemidisks of a St. Jude mitral valve was stuck in the closed position. A, B: Lack of motion of the hemidisk was apparent (arrow). C: Mild mitral regurgitation was detected (arrow) using color Doppler imaging. D: Continuous wave Doppler imaging confirms both an increased gradient (arrow) and regurgitation through the valve. The problem was rectified before leaving the operating room.

Obstruction can occur as a result of technical difficulties encountered while implanting the prosthesis. Figure 14.55 is an example of an intraoperative transesophageal echocardiogram that demonstrates immobility of one hemidisk. The hemidisk was stuck in the closed position, resulting in both stenosis and regurgitation. Other more common causes of obstruction include thrombus and pannus formation that impede proper opening of the occluder mechanism. Thrombotic interference is the most common cause of obstruction of mechanical prostheses. It may develop gradually over time or occur suddenly with catastrophic consequences. Distinguishing pannus from thrombus can be difficult but has important implications for therapy. Thrombus is usually more mobile and less echo dense. Pannus is the result of ingrowth of fibrous tissue at the interface between prosthetic material and
native tissue. It appears more dense and echogenic, is less mobile, and is usually confined to the area around the sewing ring.

A relatively small thrombus in a location that interferes with opening of the ball or disk can result in a substantial increase in the pressure gradient across the prosthetic valve (Figs. 14.56 and 14.57). The abnormality may be either permanent or intermittent and may or may not be associated with regurgitation. In these two examples, the presence of the thrombus predominantly caused obstruction to forward flow, with minimal regurgitation. Transthoracic echocardiography has low sensitivity for visualizing obstructive thrombi affecting mechanical prostheses. Most often, prosthesis dysfunction is suspected when transthoracic Doppler imaging reveals evidence of an increased pressure gradient. Then, the precise cause of the gradient is determined with transesophageal imaging. Occasionally, a larger thrombus can be seen with the transthoracic approach (Fig. 14.58). Careful scrutiny of the motion of the occluder is a key to diagnosis. The range of occluder motion should be assessed from multiple planes. M-mode echocardiography can be helpful in this setting, particularly if the abnormality is intermittent with varying occluder motion from beat to beat. Two-dimensional echocardiography can sometimes demonstrate the absence of motion of one hemidisk of a bileaflet prosthesis. Figure 14.59 is from a patient who presented with heart failure at 28 weeks of pregnancy. She had a 12-year-old St. Jude mitral prosthesis and had been inadequately anticoagulated for much of her pregnancy. A thrombus was seen on the ventricular aspect of the prosthesis, associated with a high transmitral gradient. Frequently, a combination of transthoracic and transesophageal imaging is necessary for a complete diagnosis. Fluoroscopy is a useful alternative method for assessing disk motion. Figure 14.60 is an example of a thrombus within the left atrium affecting the function of a St. Jude mitral valve. In this example, the location of the thrombus prevented one of the hemidisks from opening, thereby resulting in a moderate diastolic gradient.
FIGURE 14.56. The most common cause of prosthesis obstruction is the presence of a thrombus. In this example, a small thrombus was barely visible on transesophageal imaging (A). B: Color Doppler imaging demonstrates increased turbulence but no significant mitral regurgitation. C: Doppler imaging confirms obstruction by demonstrating a very high mean pressure gradient of 29 mm Hg.
FIGURE 14.57. A transesophageal echocardiogram from a patient with a St. Jude mitral prosthesis. A: From the four-chamber view, restricted motion of one of the hemidisks was apparent on real-time imaging (arrow). In B, only mild mitral regurgitation is present (arrow). In C, using continuous wave Doppler, a mean pressure gradient of approximately 8 mm Hg is demonstrated. The obstructed hemidisk was due to a small thrombus and the result of inadequate anticoagulation.

FIGURE 14.58. In this example, a large thrombus was visualized on transthoracic
(A) and transesophageal (B) imaging. The thrombus can be seen on the left atrial aspect of the mitral prosthesis (arrows). B: Multiple thrombi were demonstrated (arrows) adjacent to the sewing ring.

**FIGURE 14.59.** A large thrombus is demonstrated on a St. Jude mitral prosthesis in a pregnant woman (arrows). The thrombus can be seen in the long-axis (A), short-axis (B), and four-chamber (C) views. The location of the thrombus-restricted disc opening resulting in a 25 mm Hg mean mitral valve gradient (D).
Sometimes the obstruction is not apparent from two-dimensional imaging, but Doppler imaging reveals a significant increase in gradient. Figure 14.61 is taken from a patient who developed heart failure 4 months after insertion of a St. Jude aortic prosthesis. Although a thrombus could not be visualized, significant regurgitation and stenosis were demonstrated. The patient had discontinued his warfarin 3 weeks before presentation. Figure 14.62 shows a case of obstruction suspected on transthoracic imaging and then confirmed using transesophageal echocardiography. In this case, the unusual pattern and direction of the mitral inflow jet, recorded from the apical four-chamber view, was the first indication of abnormal prosthetic valve function. Pulsed Doppler imaging confirmed a significant diastolic gradient, but transesophageal imaging was required to fully demonstrate the obstructed hemidisk. Less often, obstruction is due to the presence of a vegetation within
the sewing ring, restricting antegrade flow through the prosthesis. An example of this is provided in Figure 14.63.

Echocardiography may also play a role in selecting patients for thrombolytic therapy, which is sometimes used to treat prosthetic valve thrombosis, and in assessing its success. This therapy has an overall success rate of 80% to 90% but carries a 20% risk of serious complications. Selecting candidates for thrombolytic therapy must take into account several factors. As noted above, it is essential to differentiate thrombus from pannus (which would not respond to thrombolysis). Poor overall clinical status, previous stroke, extension of thrombus beyond the valve, and large thrombus size are risk factors for complications. In one large multicenter registry (Tong et al., 2004), a thrombus area (measured using transesophageal echocardiography) more than 0.8 cm$^2$ and history of stroke were the most powerful predictors of poor outcome after thrombolytic therapy. Because the decision to proceed with thrombolysis depends in part on the size and location of the thrombus, transesophageal echocardiography plays a key role in decision-making. In addition, serial studies are helpful to evaluate the progress of therapy and determine whether prosthesis function has improved.

Bioprosthetic valves may become obstructed through the process of fibrocalcific degeneration, a primary degenerative process that occurs slowly and leads to prosthesis obstruction, almost always with a component of regurgitation (Figs. 14.27, 14.43, 14.64, and 14.65). In Figure 14.64, serial studies demonstrate progression of prosthetic aortic valve stenosis due to gradual degeneration and calcification of the leaflets. In this patient, with a 2-year-old porcine bioprosthesis, the mean aortic valve gradient more than doubled over an 18-month period. Up to 35% of porcine prostheses fail within 10 to 15 years of implantation, most with a component of primary tissue degeneration. Pericardial valves appear somewhat more durable. The risk of significant fibrocalcific degeneration is greater for valves in the mitral position and much higher in younger versus older patients. Two-dimensional imaging demonstrates increased echogenicity and decreased mobility of the leaflets, and Doppler imaging can be used to confirm an abnormally high pressure gradient across the valve. The degree of degeneration in these valves is often striking on two-dimensional imaging. The fibrocalcific changes may mimic endocarditis, and distinguishing vegetation from degeneration may be impossible on the basis of appearance alone. Figure 14.65 is an example of
this type of appearance in which the possibility of endocarditis cannot be excluded without clinical data.

**FIGURE 14.61.** Even a small thrombus, if properly located, can result in obstruction. **A:** A St. Jude aortic prosthesis is shown. A thrombus was not visualized. **B:** Color Doppler imaging demonstrates increased turbulence and significant aortic regurgitation (arrow). **C:** From the transthoracic study, a peak pressure gradient of 95 mm Hg confirms the presence of significant obstruction.
FIGURE 14.62. Thrombus formation leading to partial obstruction of mitral inflow in a patient with a St. Jude mitral prosthesis. **A:** Abnormal function of the prosthesis is suggested on the basis of the direction of the mitral inflow jet. **B:** An increased gradient confirms partial obstruction. **C:** Transesophageal echocardiography demonstrated abnormal motion of the disks (*arrows*). **D:** Failure of one hemidisk to open properly is shown (*arrows*).
Acute rupture or fracture of a calcified leaflet can lead to sudden and severe regurgitation, often a medical emergency requiring urgent surgery. This can often be visualized with two-dimensional imaging from a window that records the bioprosthesis from the upstream side. Typically, this results in an unusual flow pattern on pulsed Doppler interrogation, illustrated in Figure 14.66. This striated signal generally indicates the presence of a torn or perforated leaflet.

Thrombus formation has also been reported on transcatheter valves. Figure 14.67 is from a patient who had undergone a TAVR procedure. Six weeks later, a follow-up echocardiogram showed an increase in the valve gradient compared to his baseline study. The change in mean gradient was modest, from 9 to 21 mm Hg, and there were no other signs of dysfunction. A CT
scan (Fig. 14.67D,E) demonstrated small, layered thrombi on the prosthetic valve surfaces. He was subsequently anticoagulated with warfarin. One month later, the velocities across the aortic prosthesis had returned to baseline.

**Infective Endocarditis**

Infective endocarditis is a potentially catastrophic complication of prosthetic valves. As with native valves, an early and accurate diagnosis is essential to a favorable outcome. In contrast to native valve endocarditis, infection involving prostheses is more variable and more difficult to diagnose. Because of the reflectance of the prosthetic material, as well as its shadowing effect, detecting vegetations is challenging. Like thrombi, they are easily obscured and require imaging from multiple windows to detect. Although the most common site for attachment of a vegetation is at the base or sewing ring of the prosthetic valve (Fig. 14.68A), other locations can also serve as a site for infection (Fig. 14.68B). Small vegetations can be missed. Pannus or loose suture material can be confused with small vegetations and are sources of false-positive findings. Furthermore, distinguishing vegetation from thrombus is nearly impossible from echocardiographic criteria alone. The distinction relies heavily on the clinical situation—that is, the presence of fever and the results of blood cultures. Figure 14.69 is an example of a large vegetation on a bioprosthetic tricuspid valve in a patient with a history of intravenous drug use. Figure 14.70 shows an atypical location for a vegetation, attached to the stents of a porcine mitral prosthesis. The unusual location of this mass suggests other possible diagnoses, such as thrombus. In this case, the diagnosis was established on the basis of clinical grounds and then confirmed at surgery. In patients with prosthetic valves in whom endocarditis is being considered, transesophageal echocardiography is recommended in the majority of cases (Fig. 14.71). A combination of transthoracic and transesophageal imaging provides the most complete interrogation of the prosthesis, taking advantage of all available windows to secure a diagnosis.

An ominous complication of prosthetic valve endocarditis is the development of an abscess. As is the case with native valves, transesophageal echocardiography is significantly more sensitive for detecting abscesses.
However, because of the reflectance of the sewing ring and the tissue changes that occur after valve surgery, this diagnosis can be difficult even when transesophageal imaging is performed. A careful interrogation that focuses on a distortion of the tissue subjacent to the sewing ring is critical. Abscesses may be either echo dense or echo lucent, and color flow imaging may reveal evidence of flow within the abscess cavity (Figs. 14.72 and 14.73). Paravalvular regurgitation and/or rupture of the abscess into an adjacent chamber or space may occur in association with abscess formation. These complications are best detected with color Doppler imaging. Figure 14.74 illustrates a case of recurrent endocarditis in a young woman undergoing therapy for acute leukemia. Four months after a pericardial bioprosthetic aortic valve was placed, fever recurred. Echocardiography demonstrated a valvular vegetation and a periannular abscess.
FIGURE 14.64. Serial studies from a patient with a 2-year-old bioprosthetic aortic valve demonstrate rapid deterioration with a steadily increasing gradient over an 18-month period. Fibrocalcific degeneration of the leaflets led to a more than doubling of the gradient over that interval. The prosthetic leaflets could be seen in the long-axis view (A). In panels B through E, serial increase of the valve gradient is recorded over an 18-month period.
FIGURE 14.65. A 12-year-old porcine mitral prosthesis is recorded using transesophageal echocardiography. In A, severe fibrocalcific degeneration is demonstrated by the arrows. In B, color Doppler imaging indicates turbulent antegrade flow through the calcified leaflets. In C, continuous wave Doppler
confirms obstruction with a high mean transmitral pressure gradient. This is the result of primary tissue degeneration of the prosthetic valve.

**FIGURE 14.66.** This particular signal may be recorded in the presence of a flail bioprosthetic valve. The unusual Doppler pattern may be the result of coarse fluttering of the flail leaflets.

Although paravalvular regurgitation may occur as a technical complication after implantation, its development late after valve surgery suggests an infectious etiology (Fig. 14.75). If the degree of destabilization of the sewing ring reaches a certain point, dehiscence of the prosthesis may occur (Fig. 14.76). This leads to a characteristic rocking of the sewing ring within the implantation site. Dehiscence is a serious complication of prosthetic valve endocarditis and is almost always associated with significant paravalvular regurgitation. It is, in fact, one of the major features of the Duke diagnostic criteria. Establishing the diagnosis of dehiscence is relatively straightforward in the mitral position where rocking of the prosthesis relative to the mitral annulus is easy to detect. Dehiscence of an aortic prosthesis may be more difficult to establish because of the shadowing effect of the aortic root (Fig.
In this example, dilation of the aortic root makes the diagnosis of dehiscence easier to establish. More often, transesophageal imaging is required to confirm this diagnosis (Fig. 14.78). In this example, note the severity of the paravalvular aortic regurgitation that results from the dehisced valve. An extreme example of dehiscence, resulting from endocarditis, is provided in Figure 14.79. Once formed, an abscess may also rupture. In Figure 14.80, from a patient with porcine aortic and mitral prostheses, endocarditis recurs in the setting of ongoing intravenous drug use. Both prosthetic valves are involved and there is a periaortic abscess, with a fistula between the aortic root and left atrium.
FIGURE 14.67. Thrombus formation on a recently placed TAVR device. A modest increase in gradient 1 month after implantation (B, C) raised concerns about prosthesis function. A CT scan (D, E) demonstrated multiple small thrombi (darker areas, see arrows) on the leaflets. After 1 month of anticoagulation, the gradient had returned to baseline (F).

FIGURE 14.68. Vegetations can form anywhere on an implanted prosthetic valve. The most common site is the sewing ring. In A, multiple masses (arrows) are seen attached to the atrial side of the sewing ring of a St. Jude mitral valve. A less common site of infection is shown in B, from a patient with Starr–Edwards mitral valve. The small vegetation is attached to the top of the cage (arrow).
FIGURE 14.69. A large fungal vegetation is seen on a bioprosthetic tricuspid valve, using transesophageal echocardiography.

Video 14-69
FIGURE 14.70. An atypical location for a vegetation. The vegetation is attached to the distal edge of the stents of a bioprosthetic mitral valve. The valve leaflets (small arrows) and the vegetation (large arrow) are shown. The leaflets themselves appeared free of infection.

FIGURE 14.71. In patients with prosthetic valves, the combination of
transthoracic and transesophageal imaging is often necessary. In this example, transthoracic echocardiography (TTE) (A) was unable to identify the large vegetation present on this St. Jude mitral prosthesis. B: The large mass (arrows) was recorded in the left atrium using transesophageal echocardiography (TEE).

**FIGURE 14.72.** Although transesophageal echocardiography is more sensitive, an annular abscess can sometimes be detected on transthoracic imaging. In the long-axis view (A), a large vegetation on a bioprosthetic aortic valve is seen (arrow). The short-axis view (B) shows thickening and heterogeneity of the annulus and sewing ring (arrows), consistent with abscess formation.

*Video 14-72a*
FIGURE 14.73. Transesophageal echocardiography in a patient with a dehisced, infected porcine aortic valve is shown. In A, the prosthesis has separated from the annulus, resulting in a large anterior clear space (*) within the aortic root. Vegetations are present on the sewing ring (arrows). Color Doppler (B) reveals paravalvular regurgitation, from the aorta into the left ventricular outflow tract.
FIGURE 14.74. A recently placed bioprosthetic aortic valve develops endocarditis. In A, a large vegetation (arrow) is seen on the valve leaflets. In addition, thickening in the area of the posterior annulus (small arrowheads) suggests ring abscess formation. The relationship of the vegetation to the prosthetic leaflets is best shown in the magnified view (B).
FIGURE 14.75. A ring abscess occurring in a patient with a stentless aortic prosthesis. The abscess (arrows) is clearly visualized in both long-axis (A) and short-axis (B) views. C: Color Doppler imaging reveals flow within the abscess cavity (arrows).
FIGURE 14.76. Dehiscence of a bioprosthetic aortic valve results in an unstable, rocking motion of the sewing ring. Systolic (A) and diastolic (B) frames illustrate the exaggerated motion of the prosthesis (arrow) throughout the cardiac cycle.

Video 14-76
FIGURE 14.77. Extensive infection and subsequent dehiscence of a bioprosthetic aortic valve are shown. Systolic (A) and diastolic (B) frames demonstrate the extent of mobility and the presence of vegetations (arrows). In C, severe aortic regurgitation is present.

Video 14-77a

Although transesophageal echocardiography is very accurate for detection
of abscess formation in the presence of a prosthetic valve, errors in diagnosis may occur. Figure 14.81 is an example of a recently placed stentless aortic valve that was implanted using an inclusion technique. In this type of implantation, the porcine aortic valve and root are inserted inside the native aortic root, creating a double-density appearance of the two walls. With time the walls become adherent but until that happens, the presence of two walls separated by an echo-free space can easily be confused with an annular abscess. When doubt about the diagnosis of an abscess persists, other imaging modalities, including CT and MRI, are useful in this setting. In addition, positron emission tomography (PET) has been employed to detect abscess formation as well as peripheral emboli in patients with complicated endocarditis.

Although endocarditis in patients with prosthetic valves usually involves the prosthesis, this is not always the case. Figure 14.82 is from a patient with *Staphylococcus aureus* endocarditis who had undergone mitral valve replacement with a St. Jude prosthesis 1 year ago. Although a vegetation was not detected in association with the prosthetic valve, transesophageal echocardiography showed a perforation of a cusp of the aortic valve, with significant aortic regurgitation. An additional example is shown in Figure 14.83 from a patient with a 22-year-old Starr–Edwards mitral prosthesis, who presented with fever and positive blood cultures. Although no evidence of infection was found in association with the prosthetic valve, a tricuspid valve vegetation is noted. This emphasizes the importance of a careful and thorough echocardiogram when endocarditis is suspected in patients with prosthetic valves.

**Mechanical Failure**

Primary mechanical failure or defects in manufacturing are increasingly rare causes of prosthesis dysfunction. In the past, several recognized defects occasionally developed in some specific types of prostheses. For example, a gradual change in the shape of the occluder of Starr–Edwards prosthesis, termed ball variance, sometimes resulted in dysfunction as the ball intermittently became stuck within the cage. Older models of the Björk–Shiley valve occasionally developed fractured struts that resulted in embolization of the disk. Disk fracture has also been reported, although it is
quite rare. Each of these types of abnormality can be assessed with echocardiography. Fortunately, improvements in design and manufacture have made such catastrophic failures exceedingly uncommon.

FIGURE 14.78. A dehisced aortic prosthesis is evaluated using transesophageal echocardiography. In A, a long-axis view demonstrates that a large echo-free space is present between the aorta and the left atrium indicated by the asterisk. In B, color Doppler imaging demonstrates significant turbulent flow through this space, consistent with a dehisced prosthetic valve and paravalvular regurgitation. In C, the circumferential extent of dehiscence can be evaluated. In this case, the echo-free space is limited to the area posterior to the aortic root (*) in the region of the left atrium. This is confirmed using color Doppler imaging (D).
FIGURE 14.79. A transesophageal echocardiogram recorded during diastole (A) and systole (B) from a patient who had undergone aortic valve and aortic root replacement for treatment of endocarditis. One month after the surgery, the patient presented with fever and heart failure. The transesophageal echocardiogram demonstrates a markedly dilated aortic root with complete dehiscence of the aortic valve. A portion of the prosthetic aortic root (*) can be seen within the dilated native aorta. The arrow indicates the highly mobile mass consistent with a vegetation.

FIGURE 14.80. From a patient with bioprosthetic aortic and mitral valves, extensive infection involving both valves is demonstrated in the transesophageal long axis (A). In the short-axis view (B), a large abscess is seen between the
aortic root and left atrium, leading to a perforation. The jet into the left atrium is clearly shown with color Doppler (C).

FIGURE 14.81. A recently implanted stentless aortic valve is evaluated with transesophageal echocardiography. The valve is shown in the long-axis (left) and short-axis (right) views. Thickening of the aortic root and an echo-free space (arrows) are the result of inclusion of the porcine aortic root within the patient's aortic root, creating the appearance of a double-walled aorta (see text for details).
FIGURE 14.82. From a patient with St. Jude mitral prosthesis and bacteremia, transthoracic imaging reveals no evidence of prosthetic valve dysfunction (A), but moderate aortic regurgitation is detected with color Doppler (B, arrow). Transesophageal echocardiography also demonstrates no vegetations associated with the discs (C, arrows), although mild paravalvular regurgitation is noted (D, arrow). However, close inspection of the aortic valve reveals a perforation (E, arrow) as the cause of the aortic regurgitation (F, arrows).

RIGHT-SIDED PROSTHETIC VALVES

Most prosthetic valves inserted in the tricuspid position are bioprosthetic. Doppler echocardiographic evaluation of prosthetic tricuspid valves follows
an approach similar to that of mitral prostheses. Using a combination of the medially angulated parasternal view and the apical four-chamber view, prosthetic tricuspid valves can be adequately interrogated from the transthoracic approach (Fig. 14.84). Experience with right-sided prosthetic valves is significantly less compared with left-sided valves, so published data regarding range of normal function are limited. Flow through right-sided prosthetic valves normally occurs at low velocities, thereby increasing the risk of thrombus formation. In assessing tricuspid prostheses, the normal respiratory variation that characterizes right heart flow must be taken into account. More commonly, repair of the tricuspid valve is undertaken and an annuloplasty ring is implanted. On two-dimensional echocardiography, these rings appear as dense echogenic structures within the annulus. Echocardiographic evaluation focuses on documenting stable positioning of the ring, excluding functional stenosis from an improperly placed ring, and assessing residual tricuspid regurgitation that might be present.

**FIGURE 14.83.** A 22-year-old Starr–Edwards mitral prosthesis is examined for evidence of endocarditis. Although no abnormalities of the prosthetic valve are detected, a tricuspid valve vegetation is demonstrated (arrow).
Video 14-83

Prosthetic valves in the pulmonary position are even less common. The parasternal short-axis view at the base of the heart and subcostal views are most helpful in their assessment. A form of aortic valve surgery, the Ross procedure, involves replacement of a dysfunctional aortic valve with the patient’s own pulmonary valve (autograft) followed by implantation of a homograft in the pulmonary position. Both the valve and the proximal pulmonary artery are replaced. After a successful Ross procedure, a mild pressure gradient across the pulmonary valve is often present, sometimes associated with a minor degree of pulmonary regurgitation. Progressive stenosis, often due to degeneration of the proximal pulmonary artery, has been reported and may lead to a significant pulmonary artery gradient that is readily detected with Doppler imaging (Fig. 14.85). In Figure 14.86, from a patient who had undergone a Ross procedure 12 years previously, only a modest increase in pulmonary valve gradient is demonstrated with Doppler.
FIGURE 14.84. A bioprosthetic tricuspid valve is recorded from the basal short-axis view (A). The supporting struts are indicated by arrows. In B, Doppler demonstrates tricuspid regurgitation and a 7 mm Hg gradient across the prosthesis.
Repairing, rather than replacing, a dysfunctional mitral valve has several advantages and is being performed with increasing frequency. Selecting patients for mitral valve repair depends heavily on etiology, morphology, and severity of the valve disease as well as on the status of the left ventricle. For all these reasons, echocardiography is critical to patient management and is generally the primary factor in the decision to attempt valve repair. Because the surgical approach must be individualized, clinicians rely on a precise and thorough assessment of valve anatomy and function to plan the procedure.
The success rate of repair in patients with myxomatous degeneration and mitral valve prolapse is linked to factors that are assessed echocardiographically before and during surgery. For example, posterior leaflet prolapse carries a greater likelihood of successful repair than does anterior or bileaflet prolapse. The location and extent of leaflet excision and the decision to shorten the chordae and/or perform a ring annuloplasty also rely on echocardiographic guidance. **Figure 14.87** illustrates an excellent result of repair of mitral valve prolapse with a Carpentier ring. The ring is well positioned and effectively improves the coaptation of the leaflets during systole. At the same time, mobility of the anterior leaflet is maintained with adequate excursion during diastole to allow unimpeded left ventricular filling. This appearance of preserved anterior leaflet mobility and restricted posterior leaflet motion is typically seen following successful repair. Reduced posterior leaflet mobility following repair should not be misinterpreted as a failed repair. Instead, Doppler imaging should be employed to exclude a significant gradient. **Figure 14.88** is another example of successful mitral valve repair. Mild mitral regurgitation is present, but normal mitral inflow is preserved.

**Figure 14.89** illustrates an unsuccessful attempt to repair a regurgitating mitral valve. The mitral ring has become dislodged and appears partially detached within the left atrium. Severe mitral regurgitation is present. Another form of unsuccessful mitral valve repair involves creation of functional mitral stenosis. **Figures 14.50** and **14.90** are examples of significant diastolic gradients due to mitral valve repair. **Figure 14.90** was recorded from a patient who complained of exercise intolerance following mitral valve repair. Although the valve appears structurally and functionally normal at baseline, a significant increase in gradient is apparent following low-level exercise. This accounted for the patient’s symptoms and resolved following subsequent surgery. The topic of mitral valve repair is discussed further in **Chapter 11**.
FIGURE 14.86. Twelve years after a Ross procedure, transthoracic echocardiography demonstrates turbulent flow across the pulmonary homograft (A). Doppler reveals an 18 mm Hg mean gradient across the prosthesis (B).

FIGURE 14.87. Mitral valve repair typically involves placement of a prosthetic annular ring. In A, prior to surgery, posterior leaflet prolapse is noted. Following successful repair, the ring is seen in cross-section in diastole (B) and systole (C).
FIGURE 14.88. Some degree of regurgitation may remain after mitral valve repair. This study demonstrates a stable ring in the mitral position during systole (A, D) with well-preserved leaflet excursion during diastole (B, E). C: Mitral regurgitation (arrows) is present. F: Doppler imaging demonstrates no evidence of obstruction across the repaired mitral valve.
FIGURE 14.89. Unsuccessful mitral valve repair. A: The ring has become detached from the annulus and appears to float within the left atrial cavity (arrow). B: Color Doppler imaging demonstrates severe mitral regurgitation. These findings were confirmed using transesophageal echocardiography (C) and severe mitral regurgitation was documented (D, arrows).
FIGURE 14.90. These images were recorded in a patient following mitral valve repair, who presented with worsening dyspnea. On transthoracic echocardiography, the mitral ring is visualized and mitral leaflet excursion appears normal. This is apparent in both the long-axis (A) and four-chamber (B) views. Left ventricular systolic function is preserved. In C, continuous wave Doppler at rest demonstrates a mean pressure gradient of 8 mm Hg. However, with low-level exercise (D) the mitral gradient increases to 18 mm Hg, providing a plausible explanation for the exercise intolerance.

Suggested Readings


Wilkins GT, Gillam LD, Kritzer GL, Levine RA, Palacios IF, Weyman AE. Validation of continuous-


CLINICAL OVERVIEW

Coronary artery disease is the most common form of heart disease encountered in adults. Clinical presentations include syndromes of stable and unstable angina, acute myocardial infarction, ischemic cardiomyopathy with congestive heart failure, and sudden cardiac death. The role of echocardiography in ischemic heart disease includes diagnosing acute syndromes of angina and myocardial infarction, detecting complications, and assessing prognosis. The American College of Cardiology and the American Heart Association have established areas for which echocardiography is an appropriate diagnostic tool in patients with known or suspected coronary artery disease (Table 15.1).

Many of the echocardiographic studies evaluating myocardial ischemia, acute myocardial infarction, and complications were published prior to the area of routine emergent intervention. Urgent reperfusion strategies have dramatically altered the natural history of acute myocardial infarction as well as the prevalence of classic complications. Furthermore, the current strategy of aggressive revascularization and modern evidence-based medical therapy has changed the prognosis and rate of complications for these patients. Many of the echocardiographic studies outlining the prevalence of complications and the prediction of complications need to be taken in context of rapidly changing clinical management strategies which have significantly altered the natural history of coronary disease. In addition, modern terminology defines acute coronary syndromes and acute myocardial infarction by the presence or
absence of ST-segment elevation on the presenting electrocardiogram (STEMI vs. non-STEMI). While accurate and appropriate for decision making early in the course of acute myocardial infarction, these designations do not define the anatomic aspects of acute myocardial necrosis as being either transmural or nontransmural. Many echocardiographic and anatomical/physiologic sequelae of myocardial infarction are determined by the transmurality of infarction, rather than by the initial electrocardiographic appearance. For this reason, where appropriate older terminology of transmural and nontransmural myocardial necrosis will be employed, as it is relevant to echocardiographic findings.

**Pathophysiology of Coronary Syndromes**

Normal left ventricular wall motion consists of simultaneous myocardial thickening and inward endocardial excursion so that the cavity decreases in size in a relatively symmetric manner (Figs. 15.1 and 15.2). Interruption of normal myocardial contraction, due to ischemia or infarction, results in regional abnormalities of thickening and endocardial motion.

### Table 15.1

<table>
<thead>
<tr>
<th>Indication</th>
<th>Appropriateness Score (1–9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Symptoms or conditions potentially related to suspected cardiac etiology including but not limited to chest pain, shortness of breath, palpitations, TIA, stroke or peripheral embolic event</td>
<td>A (9)</td>
</tr>
<tr>
<td>2. Prior testing that is concerning for heart disease or structural abnormality including but not limited to chest x-ray, baseline scout images for stress echocardiogram, ECG, or cardiac biomarkers</td>
<td>A (9)</td>
</tr>
<tr>
<td>3. Frequent VPCs or exercise-induced VPCs</td>
<td>A (8)</td>
</tr>
<tr>
<td>4. Sustained or nonsustained atrial fibrillation, SVT, or VT</td>
<td>A (9)</td>
</tr>
<tr>
<td>5. Routine surveillance of ventricular function with known CAD and no change in clinical status or cardiac exam</td>
<td>rA (3)</td>
</tr>
<tr>
<td>6. Evaluation of LV function with prior ventricular function evaluation showing normal function (e.g., prior echocardiogram, left ventriculogram, CT, SPECT MPI, CMR) in patients in whom there has been no change in clinical status or cardiac exam</td>
<td>rA (1)</td>
</tr>
</tbody>
</table>
7. Routine perioperative evaluation of cardiac structure and function prior to noncardiac solid organ transplantation  

8. Hypotension or hemodynamic instability of uncertain or suspected cardiac etiology  

9. Acute chest pain with suspected MI and nondiagnostic ECG when a resting echocardiogram can be performed during pain  

10. Evaluation of a patient without chest pain but with other features of an ischemic equivalent or laboratory markers indicative of ongoing MI  

11. Suspected complication of myocardial ischemia/infraction, including but not limited to acute mitral regurgitation, ventricular septal defect, free-wall rupture/tamponade, shock, right ventricular involvement, HF, or thrombus  

12. Initial evaluation of ventricular function following ACS  

13. Reevaluation of ventricular function following ACS during recovery phase when results will guide therapy  

14. Respiratory failure or hypoxemia of uncertain etiology  

15. Respiratory failure or hypoxemia when a noncardiac etiology of respiratory failure has been established  

16. Initial evaluation or reevaluation after revascularization and/or optimal medical therapy to determine candidacy for device therapy and/or to determine optimal choice of device  

A, appropriate; U, uncertain; rA, rarely appropriate.

FIGURE 15.1. Anatomic rendering of a short-axis view of the left ventricle in diastole (A) and systole (B). Note the circular geometry of the left ventricle in both diastole and systole and the crescent-shaped geometry of the right ventricle. In the real-time image, note the symmetric wall thickening and inward endocardial
There is a well-defined hierarchy of abnormalities that occurs as a consequence of myocardial ischemia. This has been termed the “ischemic cascade” and is schematized in Figure 15.3. Resting blood flow to the myocardium is preserved until a coronary stenosis approaches 90% diameter narrowing. It should be emphasized that simple diameter narrowing is only one component of a complex anatomic and physiologic abnormality that results in reduced coronary flow. Lesion eccentricity, length, and number of sequential lesions, as well as vasomotor tone, all play crucial roles. At lesser degrees of stenosis, rest flow is preserved, but coronary flow reserve may be reduced. At times of increasing demand such as exercise, a supply–demand mismatch occurs. Creation and detection of a supply–demand mismatch is the underlying principle of stress echocardiography and other stress-testing techniques designed to unmask occult coronary artery stenoses (see Chapter 16).

With the above hierarchy of functional abnormalities in mind, one can then appreciate the predictable sequence of events that can be detected with echocardiographic imaging in the presence of a coronary stenosis. Experimentally, immediately after coronary artery occlusion, abnormalities in diastolic function occur. The easiest and most commonly identified abnormality is abnormal mitral valve inflow, with reduction in E-wave velocity and an increase in A-wave velocity which occurs within seconds of total coronary occlusion (Fig. 15.4). Early diastolic abnormalities may also be detectable with strain and strain rate imaging. There also may be a visibly abnormal relaxation pattern to the wall, mimicking a conduction abnormality. Analysis of Doppler tissue imaging or other advanced techniques has demonstrated that this abnormality often is the result of postsystolic contraction. This is followed almost immediately by loss of systolic wall thickening and decreased endocardial excursion in the region perfused by the obstructed coronary artery (Fig. 15.5).
FIGURE 15.2. Parasternal short-axis view of the left ventricle at the papillary muscle level. As with the accompanying schematic (Fig. 15.1), note the circular geometry of the left ventricle and the symmetric endocardial inward motion and wall thickening from diastole (A) to systole (B).

Video 15-2

If coronary obstruction persists for a threshold period of time (typically ≥4 hours), myocardial necrosis ensues and a persistent wall motion abnormality develops. If flow is restored before the onset of myocardial necrosis, variable degrees of recovery of function can be expected. In most instances, total occlusion of 4 to 6 hours results in irreversible myocardial necrosis. Below this threshold, varying degrees of nontransmural necrosis, predominantly involving the subendocardial layers of the myocardium, occur. The severity and extent of wall motion abnormalities depend in part on the amount of transmural versus nontransmural infarction present in a given segment.
If ischemia is the result of transient occlusion of 20 to 60 minutes, recovery of function may not be immediate but delayed due to myocardial stunning. Myocardial stunning is a phenomenon easily demonstrated experimentally and represents persistent wall motion abnormalities after restoration of coronary flow. These abnormalities recover over a variable
time period. Typically, with brief occlusions of 5 minutes or less, recovery of function occurs within 60 to 120 seconds. With coronary occlusions of 30 to 60 minutes, there may be a 24- to 72-hour delay in recovery of function. In clinical practice, there is substantial variability in the time course over which myocardial stunning recovers, and recovery of function occasionally may be delayed for weeks. Regional and global diastolic stunning also occurs. A phenomenon of repetitive stunning has also been described in which myocardium is subject to repetitive, brief episodes of ischemia. No single episode of ischemia is sufficient to result in postischemic dysfunction; however, the combined effect of multiple episodes over time may result in prolonged postischemic dysfunction that mimics myocardial hibernation.

FIGURE 15.4. Pulsed Doppler recording of mitral inflow in a canine model of myocardial ischemia. Top panel: Note the normal E/A ratio and the reversal of the E/A ratio within seconds of coronary occlusion in the bottom panel.
FIGURE 15.5. Parasternal short-axis view recorded in diastole (A) and in systole (B) in a patient with acute left anterior descending coronary artery occlusion and myocardial infarction. B: Note the lack of wall thickening and the dyskinesis of the anterior septum (outward-pointing arrows) and the normal motion of the posterior wall (inward-pointing arrows) (©).
Over a period of roughly 6 weeks, necrotic myocardium is replaced by fibrosis and scar, which is thinner and denser than normal myocardium but which has similar tensile strength, rendering it less likely to rupture (Figs. 15.6 and 15.7). There may be regional dilation in the area of the scar that results in a ventricular aneurysm (Figs. 15.8 and 15.9). An aneurysm is defined as a regional area of akinesis or dyskinesis and scar that has abnormal geometry in both diastole and systole. This is in contrast to a regional wall motion abnormality that has normal geometry in diastole and the distortion occurs exclusively in systole (dyskinesis).

Although the location of a wall motion abnormality is an accurate marker for the site of ischemia or infarction, the size of the wall motion abnormality may either under- or overestimate the anatomic extent of ischemia or infarction due to tethering. Myocardial tethering refers to the impact that an abnormal segment has on a normal adjacent segment. Tethering occurs on both a horizontal and a vertical basis. Horizontal tethering occurs when there is akinesis or dyskinesis of a segment that reduces endocardial excursion in the normal boundary tissue. The impact of horizontal or lateral tethering is that the extent of a wall motion abnormality will overrepresent the anatomic extent of myocardial necrosis because the wall motion abnormality includes not only the infarcted tissue but also a variable amount of the adjacent nonischemic boundary tissue. In general, a wall motion abnormality will overestimate the anatomic extent of a myocardial infarction by approximately 15% due to this phenomenon (Fig. 15.10). Conversely, if myocardial
ischemia or necrosis involves a very limited region, tethering by the adjacent normal (and frequently hyperdynamic) myocardium may mask the limited region of abnormal wall motion.

**FIGURE 15.6.** Schematic depiction of remodeling. The upper left schematic depicts a recent anterior and anteroseptal myocardial infarction (shaded areas) encompassing approximately 40% of the ventricular circumference. The remaining 60% is normal nonischemic, noninfarcted myocardium. The schematic
in the middle depicts progressive thinning and dilation of the infarct segment so that it now represents approximately 50% of the ventricular circumference. The schematic at the bottom represents the long-term impact of the dilated infarct segment on the remaining normal, noninvolved myocardial segments. Over time, the dilation of the infarct segment results in progressive tethering of the adjacent normal border zone with subsequent secondary myocardial dysfunction and progressive dilation and malfunction of the previously noninvolved myocardium.

**FIGURE 15.7.** Parasternal long-axis view recorded in a patient with a remote posterior wall myocardial infarction. In the central image note the relatively thin and dense posterior wall (arrows) compared to the normal thickness and myocardial texture of the ventricular septum. Akinesis is appreciated in the real-time image. At the upper left is a two-dimensionally directed M-mode echocardiogram through the anterior septum and posterior wall, again demonstrating a thin akinetic posterior wall (arrow).
Both the velocity and the magnitude of contraction are greater in the subendocardial than in the subepicardial layers. As such, a contraction abnormality in the subendocardium has a disproportionate impact on overall wall thickening. This phenomenon is known as vertical tethering. Vertical tethering has been demonstrated both experimentally and clinically and has relevance for the determination of myocardial infarction size, based on wall motion abnormalities. In general, ischemia or infarction of the inner 25% of the myocardial wall will result in akinesis or dyskinesis of that segment. As such, nontransmural involvement (either infarction or ischemia) results in malfunction of the entire wall thickness, and thus the wall motion abnormality, as evaluated by standard wall motion analysis, is indistinguishable from that seen with full transmural infarction or ischemia.
FIGURE 15.8. Anatomic rendering in the four-chamber view depicts a left ventricular apical aneurysm. **A:** Diastole. **B:** Systole. Note in diastole the abnormal geometry of the apex with localized apical and septal dilation and the relative thinning of the wall compared with the thickness in the proximal walls. **B:** The preserved thickening of the proximal walls and a lack of thickening in the aneurysmal segment in all segments distal to the arrows are shown. This abnormal geometry in both diastole and systole with wall thinning is the hallmark of true ventricular aneurysm.
FIGURE 15.9. Apical four-chamber view recorded in a patient with a remote anteroapical myocardial infarction and subsequent aneurysm and scar formation. In this end-systolic view note the altered geometry with dilation of the apical segments. Preserved function at the base of the heart can be appreciated in the real-time image. See Figure 15.15 for the extracted three-dimensional image.
DETECTION AND QUANTITATION OF WALL MOTION ABNORMALITIES

Regional left ventricular wall motion and global ventricular function can be analyzed and quantified using a number of schemes. These can be classified as purely qualitative, semiquantitative, and quantitative assessments. Table 15.2 outlines many of the schemes that are either commonly used today or have been proposed in the past for evaluation of regional wall motion abnormalities. Although detailed quantitative schemes, which measure regional or global function as a percentage of anticipated normal, may be useful for serial studies and investigational protocols, they are not necessary for clinical diagnosis. M-mode left ventricular measurements provide only limited information on patients with coronary artery disease, largely because of the regional nature of the wall motion abnormality (Fig. 15.11).

**FIGURE 15.10.** Schematic representation of horizontal tethering. This diagram represents posterior dyskinesis without translational motion. The true extent of the infarct is as noted in the darkly shaded area encompassing radian 5 and parts of radians 6 and 4. Note that there is a border zone (lightly shaded areas) adjacent to the infarct area that is anatomically normal but has abnormal motion due to the
tethering effect of posterior dyskinesis. In the schematic, the true anatomic defect represents 20% of the circumference of the left ventricle, with the tethered border zone giving an apparent total extent of 30%.

<table>
<thead>
<tr>
<th>Table 15.2 WALL MOTION ANALYSIS METHODS (CURRENTLY CLINICALLY APPLICABLE)</th>
</tr>
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<tbody>
<tr>
<td><strong>Regional</strong></td>
</tr>
<tr>
<td>Qualitative (“Eyeball” assessment)</td>
</tr>
<tr>
<td>Normal–hypokinetic–akineti–dyskinetic</td>
</tr>
<tr>
<td>Presence of scar/aneurysm</td>
</tr>
<tr>
<td>Semiquantitative</td>
</tr>
<tr>
<td>Wall motion score/score index</td>
</tr>
<tr>
<td>Quantitative</td>
</tr>
<tr>
<td>Fractional shortening</td>
</tr>
<tr>
<td>Radial shortening</td>
</tr>
<tr>
<td>Cavity/fractional cavity area change</td>
</tr>
<tr>
<td>Chordal centerline analysis</td>
</tr>
<tr>
<td>Tissue tracking based</td>
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<tr>
<td>Wall velocity/displacement</td>
</tr>
<tr>
<td>Myocardial strain</td>
</tr>
<tr>
<td>Longitudinal, radial, circumferential</td>
</tr>
<tr>
<td><strong>Global parameters of left ventricular function</strong></td>
</tr>
<tr>
<td>Ventricular geometry</td>
</tr>
<tr>
<td>Short-axis area change</td>
</tr>
<tr>
<td>Left ventricular volumes</td>
</tr>
<tr>
<td>Diastole</td>
</tr>
<tr>
<td>Systole</td>
</tr>
<tr>
<td>Ejection fraction</td>
</tr>
<tr>
<td>Doppler forward flow (TVI_{LVot})</td>
</tr>
<tr>
<td>Annular displacement (DTI)</td>
</tr>
<tr>
<td>Myocardial performance index</td>
</tr>
<tr>
<td>Left ventricular $dP/dt$ (from mitral regurgitation)</td>
</tr>
</tbody>
</table>

DTI, Doppler tissue imaging; TVI_{LVot}, Doppler time velocity integral in the left ventricular outflow tract.

Determination of global ventricular function provides diagnostic and prognostic information in patients with ischemic syndromes. Many of the methods for determining global function are discussed in Chapter 5 which deals with assessment of left ventricular systolic function. The most commonly used assessment of left ventricular systolic function is the ejection
fraction. As a matter of convenience, many echocardiographic laboratories give an “eyeball” or visually estimated qualitative assessment of the ejection fraction. Although there are data supporting this approach, it is subjective and highly observer dependent. One can measure left ventricular diastolic and systolic volumes, from which ejection fraction is then calculated. The volumes are frequently indexed to body surface area to allow normalization of data for investigational purposes.

An additional qualitative indicator of abnormal ventricular function involves assessment of ventricular geometry. The normal left ventricle is best described as a cylinder with an apical cone resulting in “bullet”-shaped geometry. This bullet-shaped geometry is noted in the apical four- and two-chamber views as well as in the subcostal view. In the short-axis view, normal left ventricular geometry is circular. In the parasternal long-axis view, normal geometry involves a slight concave curvature of both the ventricular septum and the inferoposterior wall, with the direction of concavity for each wall pointing toward the center of the ventricle. Figure 15.12 schematizes normal and abnormal geometry. Abnormal geometry is often most apparent in the apical four-chamber view and may involve rounding of the apex or asymmetry of apical shape as opposed to smooth bullet-like tapering (Figs. 15.8 and 15.9). When evaluating an echocardiogram for an ischemic wall motion abnormality, it is important to quickly assess the left ventricular geometry because it often provides a very rapid clue to the presence of abnormal regional function.

The most commonly used method for quantitation of left ventricular volume is the Simpson rule, or the rule of disks method. For this method, endocardial borders in diastole and systole are outlined. A series of disks of identical height, each of which corresponds to one of multiple, equally spaced, minor-axis dimensions of the ventricle, are generated. The volume of the individual disks is summed to provide a volume. If a regional wall motion abnormality is not visualized in the plane of examination, this technique will overestimate the ejection fraction. For this reason, when dealing with patients with coronary disease in whom regional abnormalities are anticipated, biplane methodology is necessary.
FIGURE 15.11. A: A two-dimensionally guided M-mode echocardiogram through the mid left ventricle in a normal subject. Note the symmetric contraction of both the anterior septum and the posterior wall (PW). B: Recorded in a patient with an anteroseptal myocardial infarction and extensive areas of scar. At the base, the anterior septum has normal contraction but at the level of the mitral valve (upward-pointing arrow), there is an abrupt loss of wall thickness and endocardial
motion (rightward arrows) of the anterior septum.

The majority of current generation ultrasound platforms utilize tissue tracking methodology to automatically identify boundaries as well as to track myocardial velocities throughout the cardiac cycle. When used for tracking myocardial velocities, parameters of myocardial deformation such as strain and strain rate can be derived. Similar algorithms allow detection of the endocardial/blood pool boundary from which diastolic and systolic volumes can be calculated. When used in conjunction with three-dimensional volumetric scanning, a full volume of the left ventricle can be calculated at multiple time points throughout the cardiac cycle. It is important to recognize that none of these systems are 100% accurate and that multiple interactions by the operator are commonly necessary to obtain accurate volumes.

Quantitation of regional left ventricular function is more complex. There are multiple schemes for regional wall motion assessment (Table 15.2). The assessment can be undertaken on purely qualitative terms such as an “eyeball” assessment of wall motion as being normal or abnormal or further characterized as hypokinetic, akinetic, or dyskinetic. At the other end of the spectrum, analysis can be undertaken by detailed quantitative schemes in which shortening of multiple endocardial chords around the circumference of the ventricular cavity is calculated.

While a number of different detailed, quantitative techniques have been developed and validated in the animal laboratory for quantitation of wall motion abnormalities, the majority of these are not utilized in routine clinical practice. They are limited by the ability to accurately identify endocardial borders and/or myocardial thickening as well as rotational and translational motion and the effects of tethering. As such, while in theory highly accurate for identification of wall motion abnormalities, they have seen little application in clinical practice. (See Chapter 5 for a more detailed discussion of quantitative techniques.)
FIGURE 15.12. Schematic representation of normal and abnormal left ventricular geometry shows varying degrees of regional dilation, including a classic apical aneurysm and less typical regional dilation, which may also be a manifestation of myocardial ischemia or infarction. Note that in the schematic depicting lateral wall regional dilation the posterolateral papillary muscle has been laterally displaced as well. This may result in mitral valve malcoaptation and functional mitral regurgitation. In each schematic, the dotted line represents the normal geometry.
It is important to recognize that normal myocardial motion in systole consists of two interrelated events. The first is myocardial thickening during which all layers of the wall contract, resulting in augmentation of the thickness of the myocardium from its normal 8 to 11 mm in diastole to 14 to 16 mm at end systole. This typically represents a 35% to 40% change in wall thickness. The left ventricular myocardium consists of two layers of myocardial fibers oriented circumferentially around the left ventricle. The contraction of these layers results in both apex to base shortening and circumferential shortening of the left ventricle. The two fiber layers are oriented in opposing directions such that the left ventricle contracts with a “wringing” motion which can be quantified as twist or torsion. When viewed from the apex, the base of the heart rotates clockwise and the apex in a counterclockwise direction. The nature of this wringing motion can be detected with techniques such as Doppler tissue imaging or speckle tracking. While deviation from this normal clockwise–counterclockwise wringing motion has been noted in ischemic heart disease, the incremental benefit of this analysis has not been demonstrated in clinical practice. It should also be pointed out that the apex has limited motion during the ejection and filling phases of the left ventricle. The appearance of significant apical motion on an apical view suggests that the transducer is not over the true apex.
FIGURE 15.13. Schematic representation of the currently recommended 17-segment model of the left ventricle. The parasternal and apical views are depicted. The circled numbers correspond to the current segment numbers recommended by the American Society of Echocardiography. For each segment, the coronary distribution most likely responsible for the wall motion abnormality in that area is noted. When more than one coronary territory is listed, overlap
between coronary distributions is anticipated in that segment. The apex is most often perfused by the left anterior descending coronary artery; however, in the presence of a dominant right coronary artery or circumflex coronary artery, it may also be perfused by that artery.

Because of the sequence of electrical activation of the heart, not all regions contract at the identical rate or time. In addition to substantial temporal and mechanical heterogeneity of contraction in the normal setting, ischemia accentuates heterogeneity. Although abnormal wall motion is typically described as being hypokinetic, akinetic or dyskinetic, detailed analysis of the sequence of contraction often reveals temporal variations of these contraction abnormalities. One such variation is early systolic contraction followed by dyskinetic motion rather than dyskinesis throughout the entire duration of systole. A second is marked delay in onset of contraction but with nearly normal excursion (tardokinesis). Both findings are nonspecific and can be seen as a normal variant, as a manifestation of ischemia, or in the postischemia period. As a general rule, if the wall motion abnormality is very brief (<50 ms), it is more likely to be a normal variant than a manifestation of myocardial ischemia.

When dealing with coronary disease, it is imperative to adopt a regional approach to the description of wall motion abnormalities, whether that description is a highly detailed quantitative scheme or a simple “eyeball” approach. Figure 15.13 schematizes the recommended 17-segment scheme of the left ventricle which is commonly employed for analysis, as well as the coronary arteries that usually perfuse those segments. In general, the anterior septum and anterior wall are perfused by the left anterior descending coronary artery and its branches, and the inferior wall in the area of the posterior interventricular groove by the right coronary artery. There can be substantial overlap in the inferior, lateral, and anterolateral segments, depending on the dominance of the right and left circumflex coronary arteries. The inferoapical segment represents an overlap zone between the distal left anterior descending coronary artery and the distal right coronary artery, and the apical lateral wall represents an overlap between the circumflex and the left anterior descending coronary arteries. This type of scheme that attributes the coronary artery territories to different regions can be superimposed on any of the semiquantitative or quantitative schemes to assist in linking regional wall motion abnormalities to the coronary artery.
responsible for wall motion abnormality.

The simplest assessment of wall motion consists of description of wall motion as being normal or abnormal, typically further characterized as hypokinetic, akinetic, and dyskinetic in each region of the myocardium. This assessment suffices for the immediate detection of an ischemic event but does not provide information that can be readily communicated with respect to the size of myocardial infarction or the size of an area in jeopardy.

The next level of complexity for quantitation of wall motion abnormalities involves generation of a wall motion score or score index (Fig. 15.14). This methodology involves describing the wall motion characteristics of each of the predefined segments as being normal, hypokinetic, akinetic, dyskinetic, or aneurysmal. A numerical score, typically 1 to 5, is then applied to each of these segments (Table 15.3), and the total score is divided by the number of segments evaluated to create a wall motion score index. A ventricle with completely normal wall motion has a wall motion score index of 1.0 (total score divided by the number of segments), with higher scores representing progressively greater degrees of ventricular dysfunction. This global score, representing overall left ventricular wall motion, can then be subdivided into an anterior score, representing the distribution of the left anterior descending coronary artery, and a posterior score, representing the right plus circumflex coronary artery territories. Often, because of the tremendous overlap in the posterior circulation, an effort is not made to separate the independent contribution of the right coronary artery and the circumflex coronary artery. It is often helpful to also calculate the percentage of segments with normal motion.
FIGURE 15.14. Wall motion score diagrams from two patients, one with an extensive anteroapical myocardial infarction (A) and the second with a limited inferior wall myocardial infarction (B). In the upper panel note the akinesis and dyskinesis of all segments associated with a typical left anterior descending coronary artery (LAD) infarct. Wall motion is preserved in the inferior and lateral walls. The table at the left demonstrates a markedly abnormal wall motion score in the LAD territory of 3.44 with normal scores (1.0) in the circumflex and right coronary territories. The percent of normally functioning muscle (% FM) is 43% and the global left ventricular wall motion score index (LVSI) is 2.37. In B, note the limited wall motion abnormality confined to the proximal inferior wall consistent with a limited inferior wall myocardial infarction.

<table>
<thead>
<tr>
<th>Standard Scores</th>
<th>Optional Scores</th>
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<tbody>
<tr>
<td>Normal</td>
<td>0</td>
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<tr>
<td>Hypokinetic</td>
<td>1</td>
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<td></td>
<td>1.5</td>
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<td>Akinetic</td>
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<td>2.5</td>
</tr>
<tr>
<td>Dyskinetic</td>
<td>3</td>
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<tr>
<td>Aneurysm</td>
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<sup>a</sup>Descriptive numbers only. The actual numeric value added to the global score is that corresponding to the motion pattern (i.e., 1 to 5).

Additional modifications of the wall motion score index have included an additional descriptive score for scar. Typically, the number assigned for scar is used only for descriptive purposes and the numeric value corresponding to the wall motion abnormality (i.e., 2, 3, or 4) is used for calculation purposes. For example, an akinetic-scarred segment will receive a descriptive value of 6, but when calculating the wall motion score index, it is given a value of 3 because it is akinetic. Although allowing for the description of the scar and its extent, it avoids attributing a greater functional deficit to a segment than is actually present.
A final modification involves using a score of 0 for hyperdynamic. As with the aneurysm score, this allows description of walls with compensatory hyperkinesis; however, it may result in relative underestimation of the deficit attributable to the infarct because the global numeric score now allows the compensatory hyperkinesis to reduce the impact of the wall motion abnormality. By using a score of 1.0 for calculation purposes, the regional wall motion score will remain abnormal even if overall left ventricular function is normal due to compensatory hyperkinesis. Further modifications of a wall motion score scheme have included intermediate scores of 1.5 and 2.5 for mild and severe hypokinesis, respectively, which provide additional quantitative information when evaluating patients during cardiovascular stress or in following recovery of function after myocardial infarction.

**Role of the Three-Dimensional Echocardiography**

Three-dimensional echocardiography provides an incremental method for evaluating left ventricular wall motion and for extraction of detailed parameters of left ventricular function. One clinically relevant application of three-dimensional echocardiography relies on an automated or semiautomated extraction of the left ventricular border from which a “shell” model of the left ventricular volume can be created (Fig. 15.15). Carefully done clinical studies have demonstrated the superiority of left ventricular volumes determined from three-dimensional echocardiography with respect to absolute accuracy and reproducibility. The three-dimensional volume can be automatically divided into subvolumes, corresponding to either a 16- or a 17-segment model of regional wall motion, analogous to that used for generation of a wall motion score. In theory, this method for analysis of regional ventricle function should provide information equivalent to that from visual analysis of left ventricular wall motion. In reality, technical parameters, such as dropout of the endocardial border and deficiencies in the algorithms used to identify precise boundaries, may reduce the actual impact of this technology in clinical practice especially in the setting of poor image quality. Multiple two-dimensional image planes can be extracted from a three-dimensional data set allowing simultaneous visualization of a wall motion abnormality from two or more imaging perspectives (Fig. 15.16). While technically feasible, real-time or reconstructed images from three-
dimensional data sets remain limited by frame rate, and image quality generally is not equivalent to that obtained from dedicated two-dimensional transducers.

**FIGURE 15.15.** Volume-rendered three-dimensional image recorded from the patient presented in Figure 15.9. This image was recorded at end systole. In the upper portion the wire mesh denotes the boundaries of the left ventricular wall in diastole. The colored full volume depicts the end-systolic position of the walls. Note the dyskinesis of the apical segment and the reduced ejection fraction of 39.4%. In the lower portion of the figure the individual segmental volumes are individually tracked.
FIGURE 15.16. Multiple two-dimensional imaging planes have been extracted from a single, three-dimensional volume allowing simultaneous visualization of wall motion in apical four-chamber, apical long-axis, and short-axis views of the left ventricle for simultaneous assessment for regional wall motion abnormalities in multiple orthogonal planes.
FIGURE 15.17. Doppler tissue-based strain imaging recorded in a patient with an apical myocardial infarction. Basal and distal septal regions of interest have been analyzed for strain imaging. Note the normal strain pattern in the basal septum and the substantially delayed contraction and pathologically reduced strain at the border zone of the apical myocardial infarction.
Doppler Tissue Imaging and Speckle Tracking

The basic techniques for determining strain and strain rate were discussed in Chapter 5 dealing with evaluation of left ventricular function. From a clinical perspective, the clinician should be cognizant of the fact that the algorithms for determining strain and strain rate are technique dependent and absolute values of normal vary with location in the myocardium and from patient to patient, making analysis of a subtle deviation from “normal” problematic. Scrupulous attention to detail is essential with respect to placement of regions of interest to provide data that are accurate and reproducible. The technique has shown promise in stress echocardiography where serial changes in strain are tracked in predefined regions in a given patient. As discussed earlier, normal torsion of the left ventricle is related to the opposing direction of contraction of endocardial and epicardial layers. Selective ischemia of one layer (typically the endocardial) can be detected and will alter normal ventricular torsion and may be a specific marker of selective subendocardial ischemia. Figures 15.17 and 15.18 are examples of Doppler and tissue tracking techniques used to quantify regional wall motion abnormalities.

Other Methods for Evaluating Ischemic Myocardium

Several other ultrasound methods have shown promise for evaluating patients with acute ischemic syndromes. Tissue characterization has shown promise for providing incremental information regarding myocardial contractility. This technique relies on evaluating the cyclic variation in backscatter
(returning signals from the myocardium). In the absence of myocardial ischemia, the overall intensity of returning signals within the myocardium varies phasically with the cardiac cycle. The presence of even mild myocardial ischemia results in a reduction in this cyclic variation of intensity.

Contrast echocardiography using new perfluorocarbon- or nitrogen-based agents can evaluate the integrity of capillary level flow in the myocardium. Reduction in contrast intensity correlates with reduced myocardial blood volume and has been used to identify the presence and severity of coronary stenosis. Demonstration of preserved microvascular perfusion with myocardial contrast echocardiography has correlated with myocardial viability and subsequent recovery of function in both experimental and clinical myocardial infarction. This topic was discussed in Chapter 3.

Cardiac computed tomography and magnetic resonance imaging play an increasing role in evaluation of patients with acute and chronic coronary artery presentations. Because of its complexity and other issues, cardiac magnetic resonance imaging plays a greater role in management of patients with chronic coronary disease than in the acute setting. Cardiac computed tomography is accurate for identifying complication such as pericardial effusion, aneurysm, and pseudoaneurysm formation. Cardiac magnetic resonance imaging can play a similar role and also identify mitral regurgitation and ventricular septal defect. Both techniques are highly dependent on local availability and expertise and their role needs to be taken in context of local factors. Cardiac magnetic resonance imaging with gadolinium enhancement plays a valuable role in identification of scar and has the resolution to identify subendocardial fibrosis which may be a marker of clinically unrecognized myocardial infarction (Fig. 15.19).
FIGURE 15.18. Apical two-chamber view recorded in a patient with a remote inferior wall myocardial infarction. This image was recorded with tissue tracking for determination of longitudinal strain. In the upper portion of the figure, which was recorded at end systole note the reduction of strain in the basal inferior wall (BI) compared to normal strain in the remaining segments. This is graphically demonstrated in the lower portion of the figure where the longitudinal strain of the individual seven segments is depicted. Note the reduced strain in the basal inferior wall (arrow).
FIGURE 15.19. Cardiac magnetic resonance imaging performed in a patient with an inferior myocardial infarction. A short-axis image at the base of the heart is presented. Note the abrupt thinning of the inferior wall (upward-pointing arrows) with mild aneurysmal dilation. The downward-pointing arrow denotes a laminar
Angina Pectoris

For patients with exertional chest pain, stress echocardiography can play an instrumental role in establishing the diagnosis of coronary artery disease. This is discussed in Chapter 16. In rare instances, a patient may experience an episode of spontaneous chest pain while imaging is taking place or in a situation in which imaging can be undertaken immediately. If this fortuitous timing occurs, detection of a regional wall motion abnormality during or shortly following an episode of pain is evidence that the pain is due to myocardial ischemia. The specificity of this observation is obviously greatest if the wall motion abnormality is transient and resolves simultaneously with resolution of chest pain or electrocardiographic changes.

A subset of patients with chronic coronary artery disease, but without documented myocardial infarction, has regional wall motion abnormalities on a resting echocardiogram. The potential mechanisms are repetitive stunning due to recurrent ischemia, myocardial hibernation in the presence of severe coronary stenosis, or clinically unrecognized previous nontransmural infarction. Detection of a resting regional wall motion abnormality in a patient with clinical suspicion of coronary disease is circumstantial evidence that significant underlying coronary artery disease is present.

Conversely, by detecting other forms of organic heart disease, echocardiography can play an exclusionary role in evaluating patients with chest pain. When the resting echocardiogram reveals evidence of severe valvular heart disease such as aortic stenosis or of other diseases such as pulmonary hypertension, dilated or hypertrophic cardiomyopathy, this may provide a definitive diagnosis and a plausible explanation for the presenting symptoms. In this instance, the echocardiogram is used to establish an alternative diagnosis, and coronary artery disease may become a less likely alternative.

Acute Myocardial Infarction
Urgent transthoracic two-dimensional echocardiography can play a crucial role in establishing the diagnosis of acute myocardial infarction and determining its location, extent, and prognosis. The hallmark of acute myocardial infarction or ischemia is a regional wall motion abnormality in the presence of full-thickness myocardium. The wall motion abnormality may range from hypokinetic to frankly dyskinetic. Systolic thickening of the myocardium is absent. In the presence of chest pain with electrocardiographic changes, detection of a regional wall motion abnormality is direct evidence of myocardial ischemia, and the extent of the wall motion abnormality is directly related to the volume of myocardium in jeopardy. On the basis of the fundamentals previously noted, including the disproportionate impact of subendocardial ischemia, one should appreciate the independence of the wall motion abnormality from electrocardiographic changes, as wall motion abnormalities may be seen in the absence of ST-segment elevation or Q-wave infarct.

Classic inferior myocardial infarction with ST-segment elevation and/or Q waves in electrocardiographic leads II, III, and AVF typically involves segments bordering the posterior interventricular groove with variable amounts of involvement of the inferoposterior wall and interior septum. Classic anterior and anterolateral myocardial infarction with ST-segment elevation and/or Q waves in the anterior precordial leads involves the anterior septum, anterior wall, and apex. Commonly the distal inferior wall may be abnormal in patients with left anterior descending coronary occlusion. This is due to commonly present anatomy in which the distal left anterior descending coronary artery wraps around the apex and perfuses the apical aspect of the inferior wall. Circumflex coronary artery occlusion presents with variable electrocardiographic changes, most often presenting as an inferior or posterior myocardial infarction or with exaggerated R waves in the anterior precordium. The location of wall motion abnormalities in this instance is predominantly in the inferior, posterior, and posterolateral walls. Apical involvement on echocardiography can be seen in any of the classic electrocardiographic distributions of myocardial infarction and is not limited to the anterior infarct pattern. As such, detection of an apical abnormality in the presence of an inferior or posterolateral wall motion abnormality does not necessarily imply multivessel coronary disease or concurrent anterior myocardial infarction but rather can be the effect of a single posterior
dominant coronary territory. Figures 15.20 through 15.23 were recorded in patients presenting with classic acute ST-segment elevation myocardial infarction.

As noted in the section on pathophysiology, it is not necessary to render the entire thickness of the myocardium ischemic to result in a wall motion abnormality. Ischemia involving more than 25% of wall thickness will result in akinesis or dyskinesis of the entire wall. This is in large part due to vertical tethering. As such, nontransmural necrosis, typified by “non–ST-segment elevation” myocardial infarction, results in wall motion abnormalities identical to those seen in ST elevation myocardial infarction. Because the extent of the wall motion abnormality reflects the distribution of the ischemic territory, two-dimensional echocardiography provides incremental information compared with electrocardiography for determining the amount of myocardium in jeopardy, which in turn is related to prognosis and the likelihood of complications. Figure 15.24 was recorded in a patient with non-STEMI myocardial infarction with only isolated T-wave inversion noted on the presenting electrocardiogram. Note that the extent of wall motion abnormalities in this patient is similar to that seen in ST-segment elevation infarction. A not uncommon clinical scenario is the presentation of a patient with known coronary artery disease and previous myocardial infarction who now has an acute chest pain syndrome. In this situation, it can be problematic to identify additional wall motion abnormalities on the background of a pre-existing wall motion abnormality, especially if the pre-existing abnormality is extensive. Finally, in the presence of a left bundle branch block on the presenting electrocardiogram, identification of typical wall motion abnormalities may allow the diagnosis of myocardial infarction or ischemia to be established. Various aspects of distinguishing ischemic from conduction-related wall motion abnormalities are discussed in Chapter 5, which deals with evaluation of left ventricular systolic function.
FIGURE 15.20. Parasternal long-axis echocardiogram recorded in a patient with an acute anteroseptal and apical myocardial infarction. Note the normal geometry of the left ventricle in diastole (A). B: In systole, note the normal motion of the proximal posterior wall and the lack of thickening and dyskinesis of the distal 90% of the anterior septum (arrows).

Video 15-20
FIGURE 15.21. Apical four-chamber view recorded in the same patient depicted in Figure 15.20. **A:** Recorded in diastole, note the relatively normal left ventricle geometry and biatrial enlargement, evidence of long-standing hypertensive cardiovascular disease. **B:** In the systolic panel, note the normal motion at the base of the heart (larger arrows) including the ventricular septum and lateral wall and dyskinetic and apical segments (arrows).
FIGURE 15.22. Apical four- (A) and two-chamber (B) views recorded in a patient with an acute anterior septal myocardial infarction. Both images are recorded at end systole. In A, note the dyskinesis of the mid and distal septum and apex with preserved function in the basal segments (arrows). The inset at the right are mitral Doppler and lateral annular velocities suggestive of grade 2 diastolic dysfunction. In B, also recorded in systole, note the dyskinesis of the distal inferior wall and apex (arrows) which is not uncommon in a left anterior descending coronary artery occlusion when the artery wraps around the apex and perfuses the distal inferior segments.
FIGURE 15.23. Apical two- and four-chamber views recorded in a patient with an acute inferior wall myocardial infarction related to occlusion of the right coronary artery. A is an apical two-chamber view recorded at end systole. Note the
dyskinetic motion of the proximal inferior wall (leftward-pointing arrows) and the normal motion of the more distal inferior wall segments (rightward-pointing arrows). B is an apical four-chamber view recorded in the same patient. Note the dyskinesis of the proximal one-third of the ventricular septum (leftward-pointing arrows) and normal motion of the more distal septal wall (rightward-pointing arrows). The septal wall motion abnormality is common in right coronary artery occlusion as the proximal inferior septum is perfused by the right coronary artery as is the proximal inferior wall.

Once a myocardial infarction is established, there is loss of myocardial tissue with replacement by variable degrees of scar which may be either transmural or only partial thickness. As such the involved wall is thinner than the adjacent normal myocardial segments (Figs. 15.25 and 15.26).
Multiple studies have evaluated the clinical utility of transthoracic two-dimensional echocardiography for detecting wall motion abnormalities in suspected acute myocardial infarction. In general, 80% to 95% of patients with documented myocardial infarction have detectable wall motion abnormalities. Experimentally, there is a threshold of myocardium required to produce a wall motion abnormality. The transmural threshold was discussed previously. It also appears that there is a total myocardial burden that must be rendered ischemic before a wall motion abnormality develops. Older animal models have suggested that involvement of 1.0 g of myocardium or more is necessary before any wall motion abnormality is detectable with standard echocardiography. For this reason, myocardial infarction or ischemia involving exceptionally small territories may not result in a detectable wall motion abnormality.

FIGURE 15.24. Apical two-chamber view recorded in a patient presenting with atypical chest pain, nonspecific T-wave changes on the electrocardiogram, and a mild troponin elevation. The central illustration was recorded with contrast for left ventricular opacification. In this end-systolic view, note the dyskinesis of the apical segments (arrows) consistent with ischemia and/or infarction in the territory of the distal left anterior descending coronary artery. The inset is the same image.
recorded prior to injection of contrast for left ventricular opacification. At the time of catheterization the patient had subtotal occlusion of the mid left anterior descending coronary artery.

The majority of studies correlating wall motion abnormalities with the presence of acute myocardial infarction were done prior to the advent of the ultrasensitive enzymatic assays for myocardial injury such as the currently employed troponin assays. These modern assays may detect levels of myocardial injury well below the threshold required to result in a regional wall motion abnormality or even electrocardiographic abnormalities. As such, the “sensitivity” of echocardiographic techniques for identification of an acute coronary syndrome needs to be put in context of these new markers,
which may be a marker of a clinical ischemic syndrome without detectable myocardial dysfunction. Myocardial strain and strain rate imaging may detect more subtle degrees of myocardial dysfunction than are apparent by visual analysis. Caution is advised however, as a reduction in strain or strain rate is nonspecific and can develop for reasons other than acute ischemia. Additionally, nonoptimal tracking will introduce substantial error. As such isolated reduction in strain has not seen clinical acceptance as an independent marker of ischemia or infarction.

FIGURE 15.25. Parasternal long-axis view recorded in a patient with a remote posterior wall myocardial infarction. This image was recorded at end systole. Note the relative thinning of the posterior wall compared to the ventricular septum and the dyskinetic motion in its proximal portion (arrows).
FIGURE 15.26. Apical four-chamber view recorded in a patient with a remote, limited anteroapical myocardial infarction. This image was recorded at end systole. Note the normal thickening of the proximal two-thirds of the septum and lateral wall and the abrupt area of thin-scarred myocardium in the apical septum (arrows) consistent with a remote limited anteroapical myocardial infarction.
In contemporary practice, resting transthoracic echocardiography is rarely used as a standalone technique in patients presenting with chest pain syndromes. Many centers have adopted an approach of early stress echocardiography in patients with normal resting wall motion who have presented with chest pain, suggesting an acute coronary syndrome. The safety of this approach has been demonstrated in numerous studies. The accuracy of combined rest and stress echocardiography for detecting underlying coronary artery disease likewise has been demonstrated and appears equivalent to the capability of competing radionuclide techniques. The use of stress echocardiography is discussed in Chapter 16.

**Natural History of Wall Motion Abnormalities**

Once the diagnosis of acute myocardial infarction has been established, transthoracic echocardiography can be used to follow the progression of remodeling or regression of wall motion abnormalities. If successful reperfusion is obtained either by catheter-based or pharmacologic strategies, wall motion recovers fully or in part depending on timeliness and completeness of reperfusion. Because reperfusion strategies often are not completed within the critical time window to avoid all myocardial necrosis, many patients are left with varying degrees of myocardial necrosis. Figure 15.27 was recorded in a patient with an extensive acute anteroapical myocardial infarction at the time of presentation (panel A) and at a time of follow-up (panel B).
Without successful restoration of flow, the natural course of acute myocardial infarction is for a variable degree of transmural necrosis to occur depending on the location of coronary occlusion and the presence of collateral circulation. In many cases, there will be no recovery of function in the infarct zone. Border zones that may have compromised myocardial perfusion acutely, and hence abnormal wall motion, may show recovery of function; however, the central transmural infarct zone will remain akinetic. Over approximately a 6-week period, myocardial necrosis is replaced by fibrosis and scar. Both pathologically and echocardiographically, the wall becomes thinner and denser. Figures 15.7, 15.9, 15.25, and 15.26 were recorded in patients with nonintervened myocardial infarction in which thinning of the wall and frank akinesis can be seen. More chronically, aneurysm formation and remodeling may occur that can have deleterious effects on ventricular performance. These issues are discussed further in the section on chronic coronary artery disease.

Several large clinical trials have evaluated the impact of pharmacologic therapy with either beta-blockers or angiotensin-converting enzyme inhibitors for preventing adverse remodeling following myocardial infarction. Depending on the size of the initial infarction, degree and success of reperfusion, and, in some instances, presence or absence of active treatment, adverse remodeling can be minimized. Long-term prognostic studies have demonstrated that patients with adverse ventricular remodeling are more likely to develop ventricular arrhythmias, congestive heart failure, and diastolic dysfunction, and, in general, have a substantially worse prognosis than patients in whom adverse ventricular remodeling has not occurred. Remodeling has been quantified by a number of techniques including assessment of endocardial surface area and calculation of diastolic and systolic volumes.

**Prognostic Implications**

A substantial amount of the data demonstrating the prognostic relevance of echocardiographic findings in acute myocardial infarction was developed prior to the era of aggressive emergent reperfusion strategies and current evidence-based medical therapy. As such many of the prior studies currently are less relevant. These older observations may be relevant only to those
patients with late presentations, or who for other reasons do not receive acute reperfusion. Additionally as access to aggressive interventional strategies becomes more widespread, and utilization of repeat procedures for percutaneous or other strategies becomes more common, the value of an echocardiographic finding at a single time point needs to be placed in context of the ongoing patient care.

FIGURE 15.27. Apical four-chamber view recorded in a patient with a proximal left anterior descending coronary artery obstruction and subsequent extensive anteroapical myocardial infarction. A was recorded in the acute phase immediately prior to percutaneous intervention. Note the abnormal geometry of the left ventricle and the dyskinetic motion of the distal septum, apex, and distal lateral wall (arrows). B was recorded 1 year after A. In this end-systolic view, note the relatively localized area of apical dyskinesis (arrows) compared to the extent of wall motion abnormalities in the acute phase prior to reperfusion.

Video 15-27a
FIGURE 15.28. Global longitudinal strain from an apical four-chamber view recorded in a patient presenting with an anteroapical myocardial infarction, associated with distal septal and apical akinesia. Overall left ventricular systolic function was visibly mildly reduced. In the image depicting global longitudinal strain, recorded at end systole, note the preserved mechanical function at the base of the heart with dilation and akinesia at the apex. Left ventricular ejection fraction is calculated 44.6%. Note however the markedly reduced strain in the noninvolved segments with the pathologically reduced global longitudinal strain of –6.1% in spite of preserved visible function at the base of the heart and only mild reduction of left ventricular ejection fraction.
Early studies demonstrated the adverse prognosis of wall motion abnormalities in patients presenting with acute myocardial infarction. In general, the more extensive the wall motion abnormality, whether determined by a wall motion score, ejection fraction, or more detailed quantitative techniques, the greater is the likelihood of complications such as congestive heart failure, arrhythmia, and death. The extent of regional wall motion abnormalities, as well as parameters of global ventricular dysfunction such as end-diastolic and end-systolic volume index and ejection fraction, all correlate with the likelihood of an adverse outcome in both the short and long term. More recently, a reduction in global strain has been shown to confer a worsened prognosis (Fig. 15.28).

Several other echocardiographic observations have direct relevance to prognosis. In the presence of isolated, single-vessel coronary artery disease, resulting in an acute ischemic syndrome, normally there is compensatory hyperkinesis of the remaining segments (Fig. 15.21). This mitigates against the overall adverse impact of the ischemic regional wall motion abnormality and serves to protect global function. Failure to develop compensatory hyperkinesis may be noted as a marker of multivessel coronary artery disease.

Doppler interrogation of mitral inflow, Doppler tissue velocities, and strain imaging have been used to evaluate left ventricular diastolic function at the time of acute myocardial infarction. Assuming normal diastolic properties before myocardial infarction, there is an immediate reduction in left
ventricular compliance at the time of acute ischemia or myocardial infarction. This typically results in a reduced mitral E/A ratio with a prolonged deceleration time. Clinical studies have suggested worse outcomes in individuals with restrictive (or pseudonormal) patterns in the presence of acute myocardial infarction, compared with patients with delayed relaxation (Fig. 15.29). Confounding the evaluation of mitral valve inflow patterns is the wide range of inflow patterns that may exist immediately before myocardial infarction. Although normal diastolic properties can be assumed in an otherwise healthy young patient before myocardial infarction, in the elderly population with coexistent left ventricular hypertrophy or other disease (including previous myocardial infarction), one cannot assume that the patient began with a normal baseline diastolic function. Nevertheless, detection of a classic restrictive inflow pattern does convey an adverse prognosis irrespective of the presence, nature, and degree of previously existing underlying abnormalities.

**COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION**

Virtually all mechanical complications of acute myocardial infarction can be diagnosed with two-dimensional echocardiography. In most instances, transthoracic scanning suffices for this assessment. Obviously, color Doppler flow imaging is an integral part of the comprehensive examination in patients with acute myocardial infarction and is crucial for detection and quantitation of lesions such as mitral and tricuspid regurgitation and ventricular septal defect. If transthoracic imaging is suboptimal, as may be the case in a critically ill patient, transesophageal echocardiography usually provides the necessary diagnostic information.
FIGURE 15.29. Apical four-chamber view recorded in a patient with a limited apical infarction. In this, in systolic view, note the dyskinesis of the distal septum (arrows). This is associated with grade 2 diastolic dysfunction as noted by the normal E/A ratio in the upper right and reversal of the lateral annular velocity seen in the lower right.

Video 15-29

The classic mechanical complications of acute myocardial infarction have
become increasingly infrequent. The majority were the result of transmural necrosis. In the current area of acute revascularization, patients with transmural necrosis have become increasingly less common as have the complications of infarct expansion, chronic aneurysm, free-wall rupture and ventricular septal defect.

**Pericardial Effusion**

Transient pericardial effusion is not uncommon after acute myocardial infarction. It typically is seen in transmural or ST elevation myocardial infarction and only rarely in non-STEMI. Careful surveillance studies performed in the preinterventional era demonstrated that 30% to 40% of patients with acute ST-segment elevation infarction have a transient small pericardial effusion (Fig. 15.30). The genesis of this effusion is assumed to be epicardial inflammation, and it may be seen in the absence of any symptoms of acute pericardial disease. Larger amounts of fluid, or fluid accumulation sufficient to result in hemodynamic compromise, are rare in uncomplicated myocardial infarction. Larger effusion or effusions with a hemorrhagic appearance should always prompt consideration of myocardial rupture.
FIGURE 15.30. A, B: Parasternal long-axis and short-axis echocardiograms recorded in a patient with acute anteroapical myocardial infarction and a small pericardial effusion (arrows).
FIGURE 15.31. Parasternal short-axis view recorded in a patient 72 hours after acute anterolateral myocardial infarction. In the central image, note the distorted left ventricular geometry with compression of the anterolateral wall (arrows) and the vague echodense mass which compresses the wall. This represents hematoma and hemorrhage related to free-wall rupture. In the image at the upper left note the systolic color Doppler flow signal communicating between the left ventricular cavity and the extracardiac hematoma. [Video 15-31 CFD]
Delayed pericarditis (Dressler syndrome) has also been described after myocardial infarction but appears to be less prevalent than originally described. This syndrome consists of recurrent pain with pericardial fluid, typically occurring 6 weeks to 3 months after myocardial infarction. The appearance and behavior of the effusion are similar to that due to any other cause, and as with the small effusions accumulating during the acute phase of myocardial infarction rarely leads to hemodynamic compromise.

The most worrisome scenario in which pericardial effusion is seen occurs with impending or partial rupture of the ventricular wall. This will be seen in the presence of transmural necrosis. Clinically recurring chest pain and dynamic electrocardiographic changes are seen in the absence of additional enzyme increases. Fluid accumulation in this instance is due to inflammation of the thinned, expanded wall and/or direct extravasation of blood through a partial myocardial rupture. The pericardial fluid may have a cloudy appearance or contain vague homogeneous echodensities suggesting hemorrhage. Figure 15.31 was recorded in a patient who developed recurrent pain and electrocardiographic changes 3 days after a transmural anterolateral wall myocardial infarction. Partial left ventricular free-wall rupture has occurred, resulting in an acute compressive pericardial effusion.

**Mechanical Complications of Acute Myocardial Infarction**

Even in the presence of acute infarction, myocardium with normal thickness
has nearly normal tensile strength. Infarct expansion is defined as acute thinning of the ventricular wall with aneurysmal dilation, occurring 24 to 72 hours after transmural myocardial infarction. It represents an acute remodeling phenomenon and carries significant prognostic implications. This complication is not seen in the absence of transmural necrosis. It is more common after anteroapical than posterior infarction. Echocardiographically, one detects a fairly typical aneurysmal bulge of the myocardium but without the appearance of dense scar. The wall in the area of infarct expansion consists of necrotic myocardial tissue, which, because it has expanded or been stretched over a larger endocardial surface area, may be only 3 to 5 mm in thickness, rather than the normal 8 to 10 mm. The thin necrotic wall has low tensile strength and is the precursor to most mechanical complications. Figure 15.32 schematizes this phenomenon. Figure 15.33 was recorded in a patient 48 hours after acute anteroapical myocardial infarction and shows acute infarct expansion. This complication should be recognized because it is the precursor to mechanical complications such as free-wall rupture, ventricular septal rupture, and papillary muscle rupture. Early studies suggested a short-term, in-hospital mortality as high as 40% for patients with this phenomenon.
FIGURE 15.32. Schematic representation of infarct expansion. Left: Normal left ventricular geometry with acute transmural mitral infarction of the apical portions of the left ventricle is schematized. In the acute setting, there is similar thickness of both the infarct and the noninfarct tissue. For this hypothetical example, an initial endocardial surface area of 200 cm$^2$ is assumed with uniform wall thickness of 1 cm, resulting in a left ventricular mass of 200 g. At the time of acute myocardial infarction, the total endocardial surface area is 200 cm$^2$, which is composed of 125 cm$^2$ of normal tissue and 75-cm$^2$ infarct tissue. Right: Because of infarct expansion, apical dilation has occurred so that the total endocardial
surface area is now 250 cm\(^2\), which consists of 125-cm\(^2\) normal tissue and 125-cm\(^2\) infarct tissue. Because the total amount of myocardium has not increased, there is an obligatory thinning of the infarct tissue such that the wall thickness is now 6 mm in the infarct area versus 1 cm in the normal areas. The expanded area consists of necrotic myocardium with reduced tensile strength, which is the precursor for mechanical complications such as myocardial rupture.

**FIGURE 15.33.** Apical four-chamber view recorded in a patient 48 hours after presentation with an anteroapical myocardial infarction associated with total occlusion of the mid left anterior descending coronary artery. In this somewhat off-axis four-chamber view, note the dilation of the apical segments with apical expansion and dyskinesis (arrows). In this setting, this degree of acute dilation represents infarct expansion and has been considered a precursor to mechanical complications such as ventricular septal defect.
FIGURE 15.34. Apical long-axis view recorded in a patient presenting with an extensive anteroapical myocardial infarction. Note the pedunculated mobile thrombus which has formed acutely (arrows).
Free-Wall Rupture

Rupture of the free wall of the left ventricle is usually fatal. In exceptional cases, rupture occurs with a timing such that immediate cardiovascular surgery and repair can be undertaken. As such, there are few recorded echocardiograms of patients with acute free-wall rupture. Figure 15.31 was recorded in a patient with free-wall rupture in which a large hemorrhagic pericardial effusion can be seen. Free-wall rupture most often results in instantaneous accumulation of massive compressive pericardial hemorrhage and death.

Ventricular Thrombus

Before the era of acute reperfusion strategies for myocardial infarction, ventricular thrombus was reported in 25% to 40% of patients with anterior myocardial infarction. It was infrequently reported in inferior myocardial infarction. Figure 15.34 was recorded in a patient with acute myocardial infarction and early thrombus formation. Several studies have examined the timing with which thombi occur. The peak timing of early thrombus formation appears to be approximately 72 hours; however, in larger myocardial infarctions with large areas of apical akinesis and stagnant blood flow, they may form within hours of the acute event. A thrombus in acute myocardial infarction has the same characteristics as it does in chronic myocardial infarction and may be laminar, pedunculated, or mobile. The likelihood of subsequent embolic events is greatest for thombi that are either
pedunculated or mobile and highest when a combination of features is seen. The use of transpulmonary contrast for left ventricular opacification is useful to exclude or confirm the presence of ventricular thrombosis if the diagnosis is in question.

**Right Ventricular Infarction**

Right ventricular infarction occurs most commonly (>90%) in conjunction with inferior myocardial infarction. On rare occasions, patients are noted with left anterior descending coronary artery distribution infarction and concurrent right ventricular involvement. This typically is due to a variation of coronary anatomy in which right ventricular branches arise from the left anterior descending coronary artery. The overwhelming majority of right ventricular infarctions, however, will be seen in the presence of inferior myocardial infarction due to occlusion of a proximal right coronary artery. In some instances, the inferior wall motion abnormality may be relatively small and overall left ventricular systolic function may appear preserved. Figure 15.35 was recorded in a patient with a limited inferior myocardial infarction and concurrent right ventricular infarction. With scrupulous attention to detail, variable degrees of subtle right ventricular systolic dysfunction can be appreciated in many patients with acute inferior wall myocardial infarction related to occlusion of the right coronary artery. This may include variable degrees of right ventricular dilation and systolic dysfunction as well as functional tricuspid regurgitation (Fig. 15.36). In many instances, especially if an acute reperfusion strategy has been undertaken, these changes are subtle and transient, and recovery of right ventricular function is common.
FIGURE 15.35. Four-chamber subcostal view recorded in a patient with a limited inferior myocardial infarction associated with a right ventricular infarction. In this, in end-systolic view, note the normal motion of the proximal right ventricular free wall *(downward-pointing arrow)* and the dyskinetic motion of the distal two-thirds of the right ventricular free wall *(upper-pointing arrows)*. 

*Video 15-35*
FIGURE 15.36. A, B: Apical four-chamber view recorded in a patient with an inferior myocardial infarction complicated by right ventricular infarction. A: Note the dilation of the right ventricle and in B, the secondary tricuspid regurgitation.
Note (inset) the relatively low tricuspid regurgitation velocity of approximately 2.1 m/s, which would be inconsistent with functional tricuspid regurgitation due to pulmonary hypertension.
Right ventricular infarction results in elevation of diastolic right heart pressures, which are often both subtle and transient. An early manifestation of elevated right heart pressures is persistent bowing of the atrial septum from right to the left suggesting that right atrial pressures are elevated over left atrial pressures. Additionally, because of the right atrial dilation a patent foramen ovale may manifest and transient right-to-left shunting may be
demonstrated on saline contrast echocardiography (Figs. 15.37 and 15.38). On occasion, the magnitude of right-to-left shunting results in clinically relevant hypoxemia. Interventional techniques for closing the patent foramen ovale have been employed with variable success, largely because overall outcome is more related to the magnitude of right ventricular dysfunction.
FIGURE 15.38. Transesophageal echocardiogram recorded in a patient with a limited inferior myocardial infarction and preserved left ventricular function complicated by substantial right ventricular infarction. The patient's course was
complicated by profound refractory hypoxia. A: Note the dilation of the right atrium and the persistent bowing of right to left (arrow) consistent with marked elevation of right atrial pressure. B: Note the patent foramen ovale with right-to-left shunting on color flow imaging (arrow).

Evidence of right ventricular involvement may be transient because the dysfunction is related to transient ischemia. Concurrent mitral regurgitation or ventricular septal defect increases the work of the right ventricle acutely, and the combination of right ventricular involvement with either of these entities confers a substantially worse prognosis.

For instances in which right ventricular dysfunction does not recover one can anticipate a range of persistent right ventricular dilation and reduction in
systolic function. Depending on the severity and location this may be associated with functional tricuspid regurgitation. In the absence of concurrent pulmonary hypertension the tricuspid regurgitation jet will have a relatively low velocity. There can be substantial discrepancies in the amount of left ventricular systolic dysfunction and right ventricular systolic dysfunction in the chronic phases.

Occasionally one encounters a patient presenting with evidence of right heart failure and volume overload who is incidentally noticed to have an unrecognized prior inferior infarct with right ventricular involvement (Fig. 15.39). In these instances, the right ventricular involvement with volume overload, rather than left ventricular dysfunction, is responsible for the presentation. When patients present with occult right heart failure (in the absence of pulmonary hypertension) this scenario should be considered.

**Acute Mitral Regurgitation**

Mitral regurgitation occurs after acute myocardial infarction due to several mechanisms (Fig. 15.40). The first, a mechanical complication, is rupture or partial rupture of the papillary muscle. Acute rupture of a papillary muscle may involve either the entire body of the papillary muscle or one of the subheads of the papillary, resulting in a flail leaflet. Papillary muscle rupture more commonly involves the posteromedial papillary muscle, which has single blood supply from the posterior descending coronary artery rather than a dual blood supply, which is present for the anterolateral papillary muscle. There is a wide range of myocardial infarction size which can be associated with papillary muscle rupture, and many patients present with relatively limited overall left ventricular dysfunction. Because of the acute severe mitral regurgitation, the left ventricle may be hyperdynamic, masking a limited wall motion abnormality. Unless the mitral regurgitation develops on the background of chronic diastolic dysfunction or previous mitral regurgitation, the left atrium is often normal in size.
FIGURE 15.39. Transthoracic echocardiograms recorded in a patient with a previously unrecognized inferior myocardial infarction with associated right ventricular infarction and moderate to severe functional tricuspid regurgitation. The patient presented with edema and volume overload without previously known ischemic heart disease. 

A: Recorded in the apical four-chamber view demonstrating right atrial dilation and failure of tricuspid leaflet coaptation (arrows) related to underlying right ventricular systolic dysfunction. In the inset note the moderate to severe tricuspid regurgitation. 

B: Apical two-chamber view confirming the presence of an inferior wall myocardial infarction with basal or scarring (arrows). The inset is an end-systolic short-axis view again showing evidence of an inferior myocardial infarction.
Papillary muscle rupture should be suspected in a patient with acute myocardial infarction who develops a new holosystolic murmur and congestive heart failure. The differential diagnosis is obviously between papillary muscle rupture and acute ventricular septal defect. Figures 15.41 to 15.45 were recorded in patients with papillary muscle rupture in the setting of acute myocardial infarction. On occasion, one may image a patient with papillary muscle necrosis but without frank rupture. In these instances, one may note an abnormal shape, myocardial texture, or motion of the papillary muscle.

With transthoracic imaging, visualization of the actual ruptured papillary muscle head is often problematic and transesophageal echocardiography is often essential to visualize the anatomical rupture and leaflet. A partial flail leaflet due to papillary muscle rupture most often results in an eccentric
mitral jet, the direction of which is most often opposite to that of the involved leaflet. As such a posterior flail leaflet usually results in an anteriorly directed jet. The opposite is true for an anterior flail leaflet. Color flow Doppler is crucial for evaluation of possible papillary muscle rupture. Color flow Doppler imaging allows clear separation of mitral regurgitation from ventricular septal defect in most instances. While the actual ruptured papillary muscle head often cannot be directly visualized from a transthoracic window, detection of an eccentric mitral regurgitation jet with a relatively normal-sized left atrium is indirect evidence that acute mitral regurgitation is present.

**FIGURE 15.41.** Apical two-chamber view recorded in a patient with an inferior wall myocardial infarction and acute mitral regurgitation related to papillary muscle rupture. **A:** Note the soft tissue density within the left atrium, immediately behind the mitral leaflet (arrow), representing the ruptured head of a papillary muscle. **B:** Note the highly disorganized mitral regurgitation jet, representing moderate to severe mitral regurgitation.
FIGURE 15.42. Transesophageal echocardiogram recorded from a low esophageal view in the longitudinal imaging plane. There is frank rupture of a papillary muscle. The rightward-pointing arrow denotes the detached papillary muscle head. The downward-pointing arrows denote the location of the actual left ventricular wall. The inset at the upper right is recorded with color Doppler and confirms an eccentric mitral regurgitation jet related to rupture of the papillary muscle head. [Video 15-42]
In addition to anatomic disruption of the mitral valve apparatus, mitral regurgitation can be the result of functional disturbances in mitral valve coaptation. This is typically due to apical displacement of a papillary muscle, which tethers the leaflet tip and interferes with normal coaptation. Depending on the degree of displacement and which leaflet is involved, the mitral regurgitant jet may be central or eccentric and range from mild to severe (Fig. 15.46).
FIGURE 15.43. Transesophageal echocardiogram recorded in a longitudinal view in a patient with acute myocardial infarction and papillary muscle rupture. The central image was recorded at end systole and shows the head of the papillary muscle (arrow) attached to the posterior mitral valve leaflet prolapsing into the left atrium in systole consistent with a ruptured papillary muscle head. The inset was recorded with color Doppler in the same orientation and confirms the severe mitral regurgitation in an eccentrically directed jet. 

Video 15-43
coming soon

Video 15-43CFD
FIGURE 15.44. Transesophageal echocardiogram recorded in a patient who
developed a new holosystolic murmur and shock 3 days following posterior wall infarction. Color Doppler flow imaging confirms the presence of severe mitral regurgitation with a fairly complex bidirectional jet in the left atrium. Note the prolapse of the posterior mitral leaflet and the vague echodensity of the left ventricular cavity (arrow) representing the ruptured head of the papillary muscle.

Video 15-44CFD

Video 15-44
FIGURE 15.45. Transesophageal echocardiogram recorded in a patient with acute myocardial infarction and cardiogenic shock related to papillary muscle rupture. In this instance, there is rupture of both papillary muscles with soft tissue
densities attached to both the anterior and the posterior mitral leaflets seen in the left atrium and systole (arrows). Color Doppler imaging confirms severe mitral regurgitation.

Ventricular Septal Rupture
In the preinterventional era, ventricular septal defect occurred in 3% to 5% of transmural or Q-wave myocardial infarction. It can occur at any point along the ventricular septum from the base to the apex and is seen in both left anterior descending and right coronary distribution myocardial infarction. The posterior septal perforator arteries at the base of the heart arise from the right coronary artery and right coronary artery occlusion can result in
infarction of the proximal inferior septum and ventricular septal defect at the base of the heart. Figures 15.47 to 15.50 were recorded in patients with acute myocardial infarction and ventricular septal defect.

When evaluating patients for ventricular septal defect, it is often necessary to use nonconventional imaging planes. It is often most advantageous to first scan using color flow imaging in an effort to identify the pathologic left-to-right flow rather than scanning looking for the anatomic defect. Once the abnormal flow from the left ventricle to the right ventricle has been identified and its orientation maximized, color can then be turned off and anatomic imaging undertaken. As mentioned previously, the imaging plane in which the color flow jet is best identified may not correspond to traditional imaging planes. Ventricular septal defect after acute anterior myocardial infarction can occur anywhere in the ventricular septum but is most common apically. Occasionally defects may take a serpiginous course through the myocardium, especially if only partial septal rupture has occurred.
FIGURE 15.46. Transthoracic echocardiogram recorded in a patient with an acute myocardial infarction and restricted posterior mitral leaflet related to
abnormalities of the underlying posterior lateral LV wall. Notice the abnormal coaptation pattern of the posterior mitral leaflet behind the tip of the anterior leaflet and the eccentric mitral regurgitation jet. AML, anterior mitral leaflet; PML, posterior mitral leaflet.
FIGURE 15.47. Parasternal long-axis view recorded in a patient with an acute anteroseptal myocardial infarction complicated by acute ventricular septal defect. The central panel is recorded with color Doppler. Note the systolic jet through the ventricular septal defect (arrow). The inset at the upper left is a continuous-wave Doppler through the ventricular septal defect. The peak flow velocity of 4 m/s suggests a relatively restrictive and therefore modest-size ventricular septal defect. [Video 15-47]
FIGURE 15.48. Apical four-chamber view recorded in a patient 72 hours after
presentation with an acute anterior infarction related to total occlusion of the left anterior descending coronary artery. In this case no intervention was feasible. A is an apical four-chamber view recorded at end systole. Note the aneurysmal dilation and dyskinesis of the distal septum and apex (arrows). Also note the acute thinning of the apical myocardium consistent with infarct expansion. B was recorded with color Doppler and demonstrates a ventricular septal defect at the distal apical septal wall (arrow).

Video 15-48a

coming soon

Video 15-48b

coming soon

Three-dimensional echocardiography, either from transthoracic or transesophageal window, can be used to further characterize the defect with respect to location and size (Figs. 15.51 and 15.52).

Once the diagnosis of ventricular septal defect has been established, there are several echocardiographic features which impact prognosis. These
include the status of overall left ventricular function, the presence of pulmonary hypertension, and the function of the right ventricle. When ventricular septal defect occurs as a consequence of a limited myocardial infarction and single-vessel disease, the remaining walls typically become hyperdynamic. Conversely, if a previous infarction has occurred, or if multivessel ischemia or infarction is present, the left ventricle may have global systolic dysfunction. The latter confers a substantially worse prognosis than does preserved left ventricular function. Additionally, small apical defects are substantially easier to approach from a surgical standpoint than are the large posterior ventricular septal defects and as such carry a more favorable surgical mortality. Concurrent ventricular septal defect and right ventricular infarction, which typically will be seen in inferior infarction, also carries a substantially worse prognosis.

Definitive therapy for infarct-related ventricular septal defect traditionally has involved surgical closure. Because of the acute nature of the infarct, the adjacent tissue is frequently friable and necrotic, and repair is subject to failure related to poor tissue integrity. More recently percutaneous approaches to closure of ventricular septal defects have been undertaken. This is most often and successfully accomplished in small apical defects. Echocardiography plays a valuable role in identifying those patients with appropriately located ventricular septal defects for percutaneous closure and may play a role in online guidance of closure.
FIGURE 15.49. Echocardiograms recorded in a patient with an acute inferior wall myocardial infarction complicated by ventricular septal defect. A is a transthoracic short-axis view of the left ventricle recorded with (right panel) and without color Doppler flow imaging. In the left panel, note the abrupt discontinuation of the
ventricular wall between the inferior septum and inferior wall with remnants of an intracardiac pseudoaneurysm (arrow) protruding into the right ventricle. With color-flow Doppler, note the flow between the left ventricle and the pseudoaneurysm cavity as well as the ventricular septal defect. B is a transesophageal echocardiogram recorded in the same patient from a low esophageal perspective. In this view note the roughly 1.5 cm break in the ventricular wall (arrows) allowing communication between the left and right ventricles. In the color Doppler image at the lower left, note the systolic flow from the left ventricle into the right ventricle.  

Video 15-49a

Video 15-49b CFD
FIGURE 15.50. Parasternal long-axis view recorded in a patient with an anteroseptal myocardial infarction and partial rupture of the ventricular septum. Note the lucent area within the mid anterior septum (arrows) representing partial rupture without frank ventricular septal defect. The inset at the upper left was recorded with contrast for left ventricular opacification. Note the contrast within the lucent space suggesting communication into the partially ruptured area which does not yet communicate with the right ventricle.
An additional presentation of coronary artery disease is cardiogenic shock, either occurring at the time of an acute coronary syndrome or developing subsequent to previous coronary events. Clinically, these patients will present with a combination of congestive heart failure and malperfusion, which can be traced to a specific cardiac etiology. The etiology may be isolated severe left ventricular pump failure or any of the previously mentioned mechanical complications of acute infarction, including acute severe mitral regurgitation or ventricular septal defect, right ventricular infarction, or cardiac tamponade, as well as other less common abnormalities such as acquired dynamic
outflow tract obstruction. All of these abnormalities can be rapidly identified with echocardiography. If the diagnosis is not easily established with a transthoracic imaging, transesophageal echocardiography is usually diagnostic. For patients presenting in cardiogenic shock, survival is directly related to the degree of pump dysfunction as well as the severity of mitral regurgitation.

FIGURE 15.51. The central illustration is a real-time partial volume three-dimensional echocardiogram recorded in a patient with an acute anteroapical myocardial infarction and ventricular septal defect. The upper-pointing arrow points to the area of actual tissue disruption. The panel at the left is a three-dimensional real-time echocardiogram recorded with color Doppler imaging in which the ventricular septal defect flow can easily be appreciated.
FIGURE 15.52. Transthoracic three-dimensional echocardiogram recorded in a patient with an acute infarct-related VSD. In the central figure, note the obvious ventricular septal defect. The smaller inset has been “cropped” providing an en face view of the actual ventricular septal defect.
CHRONIC CORONARY ARTERY DISEASE

Chronic complications of coronary artery disease include left ventricular aneurysm, pseudoaneurysm, chronic ventricular remodeling, chronic ischemic dysfunction (“ischemic cardiomyopathy”), functional mitral regurgitation, and chronic right ventricular dysfunction, all of which can be evaluated with echocardiography.

Left Ventricular Aneurysm

Before the era of urgent reperfusion strategies, left ventricular aneurysm developed following approximately 40% of anterior and 20% of inferior and posterior myocardial infarctions. Both pathologically and echocardiographically, an aneurysm is defined as a distinct break in the geometry of the left ventricular contour that is present in both diastole and systole with replacement of myocardium by fibrous scar tissue. By definition, it does not occur after nontransmural infarction. Approximately 6 weeks is required for scar formation. Acute infarct expansion may have a similar appearance but is seen within the 1- to 4-day time frame. Figures 15.53 through 15.56 were recorded in patients with left ventricular aneurysms after myocardial infarction. Note the broad range of aneurysm size. In general, a true aneurysm has a relatively wide mouth communicating with the aneurysmal cavity compared with a narrow neck that is seen in
pseudoaneurysm. This results in a fairly broad gradual opening to the aneurysm as opposed to a distinct shelf-like opening.

There are several echocardiographic features that should be recorded if aneurysm resection is contemplated. The clinical indications for resection of a ventricular aneurysm are intractable heart failure and less commonly for control of arrhythmias. Mechanically, the aneurysm acts as a dead space reservoir with no ability to eject blood from its diastolic volume. The remaining myocardial walls may move normally; however, the aneurysmal cavity serves as a second output for ejection and thus compromises stroke volume. When contemplating aneurysm resection, it is essential to ensure that the basal portions of the cardiac walls have normal function. This can be accomplished by calculating an ejection fraction of the basal half of the left ventricle. A simplified method to evaluate basal function is to calculate basal fractional shortening or fractional area change using a two-dimensional short-axis view at the base of the heart. Generally, if basal function is normal and the basal half ejection fraction or fractional shortening is greater than 35% or 18%, respectively, then aneurysm resection is more likely to be of clinical benefit.
**FIGURE 15.53.** Apical four-chamber view recorded in a patient with a remote anteroapical myocardial infarction–related total occlusion of the mid left anterior descending coronary artery. In this systolic frame note the dilation and aneurysmal configuration of the left ventricular apex (arrows) and the overall left ventricular dilation. The *inset* on the right are mitral inflow and annular velocities confirming the presence of grade 2 diastolic dysfunction.

Video 15-53
FIGURE 15.54. Apical four-chamber view recorded in a patient with a massive left ventricular apical aneurysm related to total occlusion of the proximal left anterior descending coronary artery. Note the relatively normal configuration of the left atrium and left ventricle in its proximal third, but the massive aneurysmal dilation of the distal two-thirds of the left ventricle (arrows). In this example, the aneurysmal cavity size exceeds that of the functioning residual left ventricle. [Video 15-54]
FIGURE 15.55. Apical two-chamber view recorded in a patient with a remote inferior wall myocardial infarction and subsequent aneurysm formation (arrows). This image was recorded in systole. Note the abnormal geometry of the left ventricle with dyskinesis and aneurysmal dilation of the proximal 40% of the inferior wall (arrows).
FIGURE 15.56. Apical four-chamber view recorded in a patient with an established anteroapical myocardial infarction and aneurysm. Note the abnormal apical geometry and thin-scarred aneurysmal cavity (arrows) with preserved function of the remaining walls.

Video 15-56

Three-dimensional echocardiography can be used to quantify aneurysm size and provides a unique perspective regarding function of residual myocardium and characteristics of left ventricular geometry (Fig. 15.57). Other approaches which can be considered to control heart failure in patients
with ventricular aneurysm have included reduction myoplasty and Dor myoplasty. In the reduction myoplasty, a large segment of the aneurysmal wall is resected, resulting in immediate remodeling of the left ventricle. In the Dor myoplasty, an intraventricular patch is placed that excludes a portion of the aneurysmal cavity without resecting the wall. The advantage of the Dor myoplasty is that the aneurysmal portion of the ventricular septum can also be excluded from the functional left ventricular cavity. Figure 15.58 was recorded in a patient after Dor myoplasty. The preoperative echocardiogram is presented in Figure 15.56. In the postoperative echo, note the linear echo within the left ventricular cavity due to the intraventricular patch that separates a true functional left ventricle, composed of normally functioning myocardium as well as smaller portions of the aneurysmal wall, from the dead space aneurysm cavity. Echocardiography can play a valuable role in assessing feasibility of either of these approaches by determining the degree to which an aneurysm is located in the anterior septum and apex (which is more favorable for Dor myoplasty) and determining the function of the residual myocardium. After Dor myoplasty, it is not uncommon to see small degrees of residual blood flow into the apical dead space created by the intraventricular patch.

Following surgical correction of the left ventricular apical aneurysm, whether by traditional techniques or a patch repair, it is not uncommon for the echocardiographic appearance to suggest residual aneurysm. It is often not technically possible to exclude the full aneurysmal cavity and success should be based on comparison of pre- and postoperative imaging.

An infrequently encountered abnormality which may be confused for an infarct-related aneurysm is a myocardial diverticulum. These are probably developmental abnormalities and can be noted anywhere in the left ventricular myocardium, but are most commonly encountered in the inferior wall or apex (Fig. 15.59). They are characterized by a very discrete outpouching of the endocardial border, typically less than 1 cm in greatest dimension with otherwise preserved left ventricular geometry and wall motion. They are typically not associated with any symptoms, arrhythmic or thromboembolic risk.
FIGURE 15.57. Transthoracic apical four-chamber view recorded in a patient with a remote anteroapical myocardial infarction and a large apical aneurysm. Both standard two-dimensional (upper panel) and reconstructed three-dimensional (lower panel) images are presented. Both images are recorded at end systole. Note the markedly abnormal left ventricular geometry with a “light bulb” shape to the left ventricle. Also note the spontaneous contrast within the cavity of the aneurysm consistent with stasis of blood.

Video 15-57 3D

Video 15-57
FIGURE 15.58. Apical four-chamber view recorded in the same patient presented in Figure 15.56 after undergoing a Dor myoplasty. Note the abnormal geometry of the apex with a horizontal shelf-like apical cavity which represents the margins of the Dor myoplasty (arrows). Overall left ventricular volume has been reduced and the ratio of functioning to nonfunctioning ventricular wall segments improved.
FIGURE 15.59. Apical two-chamber view recorded in a patient with an incidentally noted inferior wall diverticulum (arrows). Note the very discrete small (less than 1 cm) diverticulum in the proximal one-third of the inferior wall. The location of this isolated abnormality would not be consistent with recognized coronary anatomy and was incidentally noted in an individual subsequently demonstrated to be free of obstructive coronary artery disease.
Left Ventricular Pseudoaneurysm

Left ventricular pseudoaneurysm is the result of a contained rupture of the left ventricular wall. In rare instances, a pseudoaneurysm can occur within the ventricular septum rather than along the free wall. It is important to recognize a pseudoaneurysm because the likelihood of spontaneous rupture is high. Unlike a true aneurysm in which the wall consists of dense fibrous tissue with excellent tensile strength, the wall of a pseudoaneurysm is composed of organizing thrombus and varying portions of the epicardium and parietal pericardium (Fig. 15.60). Pathologically, it is the sequelae of myocardial rupture with hemorrhage into the pericardial space, which then becomes locally compressive. Local tamponade occurs, preventing further hemorrhage into the pericardium. Over time, the intrapericardial thrombus organizes, creating a wall to the pseudoaneurysm, however, with poor structural integrity. As such, it is at risk of spontaneous rupture, which is generally a fatal event.

Figures 15.61 through 15.63 were recorded in patients with pseudoaneurysms. Note the narrow opening to the pseudoaneurysm with an overhanging shelf-like edge. Traditionally, it is thought that if the size of the opening to the left ventricular cavity is less than the maximal dimension of the aneurysm, the defect is more likely to be a pseudoaneurysm. Because the pseudoaneurysm is composed of both a free-aneurysmal cavity and the organizing hematoma, its true size is often underrepresented on echocardiography because the organized hematoma has a soft tissue density similar to that of surrounding structures. It is therefore not uncommon to have the situation of a pericardiac mass on chest radiography or computed tomography in the presence of what appears to be a modest size pseudoaneurysmal cavity detected with echocardiography. This phenomenon also makes it more difficult to assess the ratio of the size of the opening to the left ventricular cavity to the actual aneurysm size because only the blood-filled aspect of the pseudoaneurysm may be easily visualized. This phenomenon is schematized in Figure 16.60. Pseudoaneurysms at the base of the heart most commonly occur after inferior myocardial infarction and may be difficult to separate from a true aneurysm. In this location, they may have a wider mouth than is traditionally taught. Their true nature is often confirmed only at the time of surgical inspection (or autopsy).
Because the body of the pseudoaneurysm may fall outside of the typical echocardiographic imaging planes, further evaluation with cardiac CT or MRI is often essential. Both have the ability to visualize the field of view outside of the actual cardiac silhouette and can provide a more accurate determination of the full anatomical extent of the pseudoaneurysm. Cardiac MRI can uniquely identify tissue structure.
Chronic Remodeling

After transmural myocardial necrosis, a process of ventricular remodeling may occur. Remodeling refers to the tendency of the left ventricle to chronically alter in size and geometry due to adverse effects of the myocardial infarction. Even a well-localized myocardial infarction will be surrounded by a dysfunctional border zone. Within the border zone, myocardial dysfunction is due to a combination of factors including tethering, varying degrees of nontransmural necrosis, and abnormal regional wall stress in the regionally dilated segments. Over time, this results in progressive dilation of the ventricle at the margins of the myocardial infarction, even in the presence of a relatively healthy, normally perfused myocardium. Chronic remodeling is usually a complication of a larger anterior infarction and is less often seen after posterior distribution infarction. By definition it will be seen after transmural rather than nontransmural necrosis. Figure 15.6 schematizes the remodeling process, and Figure 15.64 is an example of a patient who presented with an extensive anteroapical infarction and who has had adverse remodeling over time. Ventricular remodeling is of clinical relevance because it results in dilation of the ventricle and reduced contractile performance with reduction in left ventricular ejection fraction. Remodeling often may also result in malcoaptation of the mitral leaflets and secondary mitral regurgitation due to the apical and lateral displacement of the papillary muscles. Clinical studies have suggested that beta-blockers or angiotensin-converting enzyme blockade may prevent or reduce adverse remodeling.
FIGURE 15.61. Off-axis apical view recorded in a patient with an inferior wall myocardial infarction of uncertain timing. There is evidence of a large inferior wall pseudoaneurysm (PA), the outer boundary of which is noted by the downward-pointing arrows. There is thrombus in the cavity of the pseudoaneurysm. The inset at the lower left is a cardiac magnetic resonance image in a similar plane. The upper-pointing arrow denotes the outer wall of the pseudoaneurysm. The downward-pointing arrows outline a filling defect in the pseudoaneurysm consistent with thrombus. 

Video 15-61
FIGURE 15.62. Transesophageal echocardiogram recorded in the same patient shown in Figure 15.61. The central image was recorded from a low esophageal view in a short axis of the left ventricle. Note the large pseudoaneurysm cavity (PA). The upward-pointing arrows denote the thrombus which partially fills the pseudoaneurysm. The two inward-pointing arrows define the relatively narrow neck of the pseudoaneurysm. The panel at the lower right is a cardiac magnetic resonance image of the same patient showing virtually identical anatomy. Note that the orientation of the cardiac magnetic resonance image is upside down compared to the central image.
FIGURE 15.63. Parasternal short-axis view of the left ventricle recorded at the mitral valve level. Note the break in the continuity of the left ventricular inferior wall (arrows) which communicates with vague extracardiac space representing the pseudoaneurysm (PA). The extent of the pseudoaneurysm is not well appreciated from this echocardiogram. The inset at the lower right is a cardiac magnetic resonance image recorded in the same patient in approximately the same orientation. Again note the wide opening between the left ventricle and pseudoaneurysm (arrows) with the substantially greater dimension of the pseudoaneurysm exterior to the cardiac contour, the full extent of which is clearly seen.
FIGURE 15.64. Apical four-chamber views recorded at two time points in a patient with an anteroapical myocardial infarction. **A** was recorded 4 weeks following myocardial infarction. Note the dilation and dyskinesis of the distal septum and apex (*arrows*). Left ventricular end diastolic volume (LVEDV) and ejection fraction are as noted. **B** was recorded in the same patient 3 years following the event. There is a similar distribution of wall motion abnormalities in the septum and apex (*arrows*) but there has been progressive dilation of the left ventricle with an increase in left ventricular volume and a decrease in ejection fraction. This has occurred in the absence of any further ischemic events and represents adverse ventricular remodeling. 

Video 15-64b
FIGURE 15.65. Apical four-chamber view recorded in a patient with an anteroapical myocardial infarction and thrombus formation. Note the oval-shaped filling defect at the ventricular apex (arrows) consistent with laminar thrombus. Incidental note is also made of right ventricular dilation related to pre-existing pulmonary hypertension. The image at the upper left is an expanded view of the apex in which the pedunculated nature of the thrombus can be appreciated. 🎥

Video 15-65
Mural Thrombus

Chronic thrombus formation is most common after large anterior myocardial infarction, especially with involvement of the apex. Before the interventional era, left ventricular thrombus occurred in 25% to 40% of patients after a first anteroapical myocardial infarction. With the advent of acute reperfusion strategies, there has been a dramatic decline in this prevalence. The major risk of left ventricular thrombus is of subsequent embolization with stroke or major organ loss. Historically, the likelihood of embolic events was greatest in the first 2 weeks after the acute event and tapered off over the ensuing 6 weeks. After this time, there is presumed endothelialization of the thrombus with reduction in its embolic potential. There are several characteristics of ventricular thrombus that should be noted. These include not only size but also whether it is laminar, forming a layer along the akinetic wall, versus being pedunculated and protruding into the ventricle. Thrombi may be mobile, a characteristic which has been associated with a higher embolic potential. Figures 15.65 through 15.68 were recorded in patients with myocardial infarction and illustrate the range of thrombi to be seen. Note in Figure 15.66 that there is an anteroapical wall motion abnormality with a purely laminar thrombus. This is chronic thrombus, likely to be covered fully by an endothelial layer, and presumably has a relatively low embolic potential. Contrast this to the thrombi in Figures 15.67 and 15.68, which are pedunculated and mobile. Both pedunculated character and mobility confer a greater likelihood of embolization with embolic rates reported as high as 40% when both mobility and protrusion into the cavity have been reported. On occasion, fresh thrombi take on a cystic appearance. This is due to a combination of factors including varying degrees of maturity of the clot, and results in acoustic boundaries between relatively fresh and more organized regions. This results in a relative echolucency to the center of the thrombus. When seen in the presence of a wall motion abnormality in which thrombus would be expected, it is important to recognize this as such rather than make the diagnosis of presumed cyst or tumor.
FIGURE 15.66. Apical four-chamber view recorded in a patient with an anteroapical myocardial infarction. Note the laminar filling defect at the apex of the left ventricle (arrows) which represents a chronic mural thrombus. [Video 15-66]
FIGURE 15.67. Off-axis apical view recorded in a patient with a prior limited apical infarction. Note the pedunculated mass attached to the inferoapical wall consistent with thrombus. The inset is a real-time three-dimensional image recorded in the same patient. In the real-time images, note the mobility of the thrombus. [Video 15-67 3d]
In addition to frank thrombus formation, when using newer generation, high-frequency transducers, spontaneous contrast is occasionally noted in the left ventricular cavity (Fig. 15.57). This typically will be seen in the area of a regional wall motion abnormality or aneurysm. The etiology of the spontaneous contrast is presumably stagnant blood in the region of an aneurysmal dilation. Color flow imaging at low velocities and intravenous contrast for left ventricular opacification can also demonstrate abnormal swirling patterns of blood.
FIGURE 15.68. Apical view recorded in a patient with a remote anteroapical myocardial infarction and apical aneurysm. The central figure was recorded in a four-chamber orientation. Note the very large thrombus filling a substantial portion of the aneurysm and ventricular apex (arrows). The inset at the upper right was recorded in an off-angle image of the apex and demonstrates that the thrombus is pedunculated and anchored to the apex with otherwise free margins (arrows).
On occasion, either the vague nature of a thrombus or the technical limitations in the examination render it difficult to either exclude or confirm the presence of ventricular thrombus. The use of higher frequency, short-focus transducers can often result in higher quality imaging in the apex and resolve the dilemma. An additional echocardiographic tool to further evaluate the presence or absence of thrombus is the use of intravenous contrast. Using the newer-generation perfluorocarbon-based agents, which pass into the left ventricular cavity, it is possible to fully opacify the left ventricular apex. In doing so, one may then detect a true fixed filling defect in the apex and thereby confirm the presence of ventricular thrombus.

Mitral Regurgitation

Chronic mitral regurgitation can occur through several mechanisms involving different aspects of the mitral apparatus. Necrosis and subsequent scarring of a papillary muscle may result in retraction of either the anterior or the posterior mitral apparatus but is most common with the posterior leaflet. This results in a malcoaptation process, as shown in Figure 15.40. Figure 15.69 was recorded in a patient with previous myocardial infarction and functional mitral regurgitation. It should be emphasized that “papillary muscle dysfunction” actually represents malfunction not only of the papillary muscle but also of the underlying ventricular wall. As a consequence of remodeling, the wall supporting the papillary muscle and the papillary muscle itself are apically and posteriorly or laterally displaced. This has the effect of
functionally shortening the mitral valve apparatus for that leaflet, thus restricting its ability to close fully. This phenomenon can be quantified as the tenting area of the mitral valve, which is directly related to the severity of the subsequent mitral regurgitation. This results in abnormal coaptation and mitral regurgitation. This is not infrequently accompanied by dilation of the mitral annulus. The degree of mitral regurgitation that results by this mechanism can range from trivial and inconsequential to severe and may be a cause of congestive heart failure. The severity of mitral regurgitation due to this mechanism is graded as for other forms of mitral regurgitation. Because the underlying pathophysiology may involve one leaflet more than the other, eccentric jets are not uncommon and caution regarding grading severity is advised. The issue of chronic ischemic mitral regurgitation is further discussed in Chapter 11.

FIGURE 15.69. Two- and three-dimensional echocardiograms recorded in a patient with chronic ischemic mitral regurgitation. In the three-dimensional image, note the abnormal restricted motion of the posterior leaflet (most easily seen in
the real-time image). Color Doppler imaging recorded from a transesophageal approach confirms a highly eccentric mitral regurgitation jet. 

Chronic Ischemic Dysfunction

Ischemic cardiomyopathy is defined as chronic left ventricular dysfunction due to the sequelae of diffuse coronary artery disease. By definition, it excludes congestive heart failure due to discrete left ventricular aneurysm with otherwise preserved function or acute myocardial infarction. Several recent studies have demonstrated substantial areas of nontransmural infarction and fibrosis in most patients with diffuse left ventricular dysfunction and underlying coronary artery disease. This may be seen in the
absence of clinical evidence of prior myocardial infarction. In the typical ischemic cardiomyopathy, the left ventricle is composed of areas of normal myocardium, areas of transmural scar, and substantial areas of partial-thickness fibrosis (Fig. 15.70). Echocardiographically, a wide range of appearances, from multiple discrete areas of wall motion abnormalities to global hypokinesis, may be encountered. In addition to “ischemic cardiomyopathy” as the result of multiple prior myocardial infarctions with mixed full- and partial-thickness necrosis, a number of patients will have chronic ischemic dysfunction without evidence of discrete myocardial infarction based largely on chronic hibernation. In this situation, chronic low-grade ischemia has occurred with downregulation of contractile elements. If sufficient myocardium is involved in the hibernation process, global dysfunction with full-thickness myocardium will be noted. In the majority of instances, there will be sufficient regional heterogeneity or limited areas of frank akinesia with scar to allow establishing a diagnosis of ischemic substrate. On occasion, virtually all myocardium retains full thickness and only global hypokinesis is noted (Fig. 15.71). Dobutamine stress echocardiography with attention to low-dose augmentation and high-dose deterioration may be a relatively specific task for identifying this scenario as being ischemic in etiology. If substantial viable myocardium is noted, successful reperfusion, most often with coronary artery bypass grafting, often allows significant recovery of systolic function. Because of the chronic nature of ischemic cardiomyopathy, varying degrees of mitral regurgitation are nearly ubiquitous and secondary pulmonary hypertension and tricuspid regurgitation are common. In many instances, there will be substantial areas of viable myocardium that may recover function if successfully reperfused (Fig. 15.72).
FIGURE 15.70. Apical four-chamber view recorded in a patient with a chronic ischemic cardiomyopathy. In this instance, there is an established infarct with scarring in the distal septum and apex (arrows). Additionally, there is global hypokinesis of the remaining left ventricular segments with an overall reduction of left ventricular systolic dysfunction consistent with an ischemic cardiomyopathy. Note the secondary dilation of the left atrium. Incidental note is made of an ICD lead in the right ventricle.  Video 15-70
FIGURE 15.71. Apical four-chamber view recorded in a patient with recently recognized systolic dysfunction and congestive heart failure. A is an apical four-chamber view revealing full-thickness myocardium. This image was recorded at end systole and reveals significant global left ventricular systolic dysfunction. The two insets are mitral valve inflow and Doppler annular velocities suggesting diastolic dysfunction. B was recorded with color Doppler imaging and reveals moderate functional mitral regurgitation. The patient was subsequently documented to have severe multivessel coronary artery disease and underwent multivessel bypass surgery. Figure 15.81 is a follow-up echocardiogram. Video 15-71a
It is often not possible to separate an ischemic from a nonischemic dilated cardiomyopathy. Clues to the former include patient age and cardiovascular risk factors as well as clinical information regarding previous ischemic events. In the absence of clinical evidence of previous infarction, detection of an area of frank scar frequently will establish the diagnosis of an ischemic etiology for chronic dysfunction. In many instances, it will not be possible to accurately separate the two entities and coronary arteriography will be necessary to establish or exclude the diagnosis. In some patients, there will be concurrent coronary disease and primary cardiomyopathy. Typically, these individuals will have significant left ventricular dysfunction and limited coronary artery disease, resulting in a situation in which the degree of left ventricular dysfunction is out of proportion to the severity of coronary disease. These individuals probably have the combination of nonischemic cardiomyopathy and incidental coronary disease.

**DIRECT CORONARY VISUALIZATION**

There are several clinical instances in which direct visualization of the coronary arteries can provide valuable clinical information. The ostia of the left main and right coronary arteries can be visualized in most adults and in virtually all children, using transthoracic and transesophageal echocardiography. Additionally, a variable length of the left main and proximal left anterior descending and right coronary artery can often be
CT angiography of the coronary arteries is considered the “gold standard” for assessing the origin and proximal course of the coronary arteries. Its accuracy exceeds that of echocardiography and often conventional coronary angiography for determining the course of anomalous coronary arteries. Because of radiation exposure its value for serial evaluation, especially in children with Kawasaki syndrome is limited.

To visualize the ostia of the left and right coronary arteries from a transthoracic echocardiogram, scanning is performed in a parasternal short-axis view at the base of the heart (Fig. 15.73). The proximal left main coronary artery is seen arising from the left coronary cusp at approximately the 4 o’clock position. The ostium of the right coronary artery is closer to the sinotubular ridge and arises at approximately the 10 o’clock position. Typically, it is not possible to visualize the proximal portions of both coronary arteries simultaneously because the takeoff of the right coronary artery is more cephalad than that of the left. Additionally, a variable length of the left anterior descending coronary artery can be visualized using a modified parasternal long-axis view along the interventricular groove.

**FIGURE 15.72.** These images were recorded in the same patient depicted in Figure 15.71 3 months following coronary artery bypass surgery. In **A**, recorded in systole, note the improved global systolic function with only residual apical akinesia (arrows). Overall left ventricular size and geometry are markedly improved. The insets are mitral valve inflow and annular velocities demonstrating improved diastolic function. **B** was recorded in systole with color Doppler and reveals marked reduction in the severity of functional mitral regurgitation.
FIGURE 15.73. Parasternal short-axis echocardiogram recorded at the base of the heart demonstrates the origin of the left main coronary artery (arrows) (A) and the right coronary artery (arrows) (B). Note that the takeoff of the two coronary arteries is not simultaneously visualized because the right coronary artery takeoff is slightly more cephalad than that of the left main coronary artery.
Using transesophageal echocardiography, both coronary ostia likewise can
be visualized. Typically, the left main coronary artery is technically easier to
visualize than the right (Fig. 15.74). There are several clinical instances in
which visualization of the coronary arteries is of clinical benefit and other
situations in which it may provide valuable clues to the presence of
underlying disease. Clinical situations in which it is of proven benefit include
identification of anomalous coronary artery takeoff and detection of
aneurysms in Kawasaki disease.

There are several variations of anomalous coronary artery origin, some of
which are schematized in Figure 15.75. Clinically, one should document the
origin of both coronary arteries in childhood cases of cardiomyopathy in
which the origin of a coronary artery from the pulmonary artery may lead to a
cardiomyopathic process and, if possible, in patients for whom
echocardiographic screening as part of an athletic screen is indicated. If both
coronary arteries are identified with normal origins, the likelihood of a
coronary artery anomaly is low. If one or the other main coronary artery is
not visualized, this is indirect evidence that there is a possible anomalous
origin.
FIGURE 15.74. Transesophageal echocardiogram recorded in the short axis (upper panel) and longitudinal axis (lower panel) of the proximal aorta. The takeoff of the left main coronary artery (arrow) and the right coronary artery is clearly seen (arrow). L, left coronary cusp; N, noncoronary cusp; R, right coronary
Common variations on anomalous coronary artery anatomy include origin of the right coronary artery from the left coronary sinus or the left anterior descending or circumflex artery from the right coronary sinus. Less often, the left main coronary artery may arise in an anomalous location. A relatively common coronary anomaly is an anomalous origin of the right coronary artery from the left coronary cusp after which it then courses between the aorta and the pulmonary artery before assuming a relatively normal course. This anomaly has been associated with sudden cardiac death during exercise, presumably because of the acute angle that the coronary artery makes in arising from the left cusp before traversing posteriorly (“malignant course”).

The presumed mechanism of sudden death is acute kinking of the artery with reduction in flow at the time of or immediately after vigorous physical exercise. On occasion, the course of the anomalous coronary artery between the two great vessels can be directly visualized with either transthoracic or transesophageal echocardiography, but is best identified by CT angiography.

Anomalous origin of a coronary artery from the pulmonary artery is an uncommon condition that usually presents as a dilated cardiomyopathy in infancy. Most often, a coronary steal phenomenon occurs in which there is retrograde flow in the anomalous artery. This results in effective bypassing of the myocardium into the low-pressure pulmonary circuit. Chronically, diversion of flow from the coronary arterial system into the low-pressure pulmonary artery results in a myopathic process. Because the anomalous coronary artery, arising from the pulmonary artery, represents a pathologic left-to-right shunt, the vessel typically dilates in response to the high-volume flow. Additionally, because the entire myocardial blood flow volume is provided by the remaining normally connected arteries, they likewise dilate in response to the excess volume flow. Similar dilation of a coronary artery may be seen in cases of a coronary artery atrial fistula in which the low resistance of flow into the atrium results in a pathologic increase in flow volume and subsequent coronary artery dilation (Figs. 15.76 to 15.80).

On occasion, one may directly visualize the abnormal flow into a downstream chamber as a continuous turbulent flow signal (Fig. 15.78).
FIGURE 15.75. Schematic representation of normal and abnormal origins of the coronary arteries. The upper left schematic depicts the normal takeoff of the right coronary artery and the left main coronary artery from the right and left Valsalva sinuses, respectively. The middle schematic depicts the anomalous origin of the right coronary artery from the left Valsalva sinus. The right coronary artery...
courses between the aorta and the RVOT pulmonary artery. This course results in a marked angulation of the right coronary artery near its origin, which may result in coronary flow compromise. The lower left schematic depicts the origin of either the left coronary artery or circumflex from the right coronary artery or right Valsalva sinus. As with the anomalous origin of the right coronary artery from the left Valsalva sinus, the artery courses between the aorta and the right ventricular outflow tract and may have an acute bend near its origin, which may result in compromise of flow. L, left coronary cusp; LAD, left anterior descending coronary artery; LCX, circumflex coronary artery; N, noncoronary cusp; R, right coronary cusp; RCA, right coronary artery.

FIGURE 15.76. Parasternal long-axis view recorded in a patient with marked dilation of the proximal right coronary artery due to a coronary artery fistula to the right atrium. A similar appearance may be noted in the anomalous takeoff of the left coronary artery from the pulmonary trunk due to compensatory high flow in the right coronary artery. RCA, right coronary artery.
FIGURE 15.77. Parasternal long-axis view recorded in a young patient incidentally noted to have mild left ventricular systolic dysfunction and a coronary artery anomaly. In the central figure note the marked dilation of the left coronary artery (dotted line) and the in color-flow image at the upper left the robust flow into the dilated left main coronary artery with a suggestion of continuing flow in a nonanatomical direction. This patient was eventually demonstrated to have a large fistulous connection between the distal left main coronary artery and the right atrium resulting in a coronary steal phenomena and a low-grade cardiomyopathy. Figures 15.78 to 15.80 were recorded in the same patient.
While technically feasible to identify the origin and proximal course of the coronary arteries with transthoracic or transesophageal echocardiography, the utility of this approach in clinical practice is limited. Cardiac magnetic resonance imaging and computed tomographic coronary arteriography are superior for identifying the course of anomalous coronary arteries even compared with traditional coronary arteriography.
FIGURE 15.78. Transesophageal echocardiogram recorded in same patient presented in Figure 15.77 visualizing the left and right atria. In the central figure note the robust color flow which appears to originate in the atrial septum and then empty into the right atrium. The inset at the lower left is the same image recorded without color Doppler which reveals a large circular structure which represents the coronary fistula. The pair of horizontal lines represents the plane of the atrial septum. Video 15-78 IAS
FIGURE 15.79. Apical four-chamber split screen image with and without color Doppler recorded in the same patient presented previously. In the image on the left note the suggestion of a channel communicating between the lateral aspect of the left ventricle and the right atrium. The inset on the right was recorded with color Doppler and clearly demonstrates a communication into the right atrium (arrows) from an arterial source. The actual origin of this communication cannot be ascertained from this view.
FIGURE 15.80. Three-dimensional reconstruction of a coronary CT angiogram recorded in the patient presented in the preceding three figures. Note the marked dilation of the left main coronary artery (LMCA) which has a normal origin from the aorta. Note also the markedly dilated fistulous communication (small arrows) originating from the distal left main coronary artery and the emptying directly into the atrium.
FIGURE 15.81. Parasternal short-axis view recorded at the base of the heart in a child with Kawasaki disease and aneurysmal dilation of the right coronary artery. Note the size and location of the aorta and pulmonary artery and a markedly dilated right coronary artery that measures approximately 8 mm in diameter. LMCA, left main coronary artery; RCA, right coronary artery.

Kawasaki Disease

Kawasaki disease is an infectious/inflammatory disease, typically of childhood. Its major manifestations are arthralgia, rash, and fever, and it is
associated with coronary arterial aneurysms. Detection of aneurysms by echocardiography is one of the clinical features for establishing the diagnosis of Kawasaki disease. Typically, the aneurysms are present in the proximal portions of the coronary arteries and as such can be visualized with transthoracic echocardiography. Because this is a childhood disease, in which coronary visualization is often less problematic, screening the coronary arteries with transthoracic echocardiography provides a reliable tool for establishing or excluding the diagnosis of the disease. The image in Figure 15.81 was recorded in a patient with Kawasaki disease and demonstrates a large right coronary artery aneurysm. Color flow imaging often demonstrates fairly limited color flow areas within the aneurysm. High-frequency scanning can frequently demonstrate thrombus lining the wall of an aneurysm. Two-dimensional echocardiography is used as a tool for follow-up of these aneurysms because their size and appearance may change over time.

Occasionally, one encounters an adult patient with a proximal coronary artery aneurysm of uncertain etiology. Many such aneurysms may represent the sequelae of previously unrecognized Kawasaki disease in childhood. Not infrequently the aneurysm is detected when echocardiography is performed in a patient who is being evaluated for chest pain syndrome. On occasion, the aneurysms encountered in adult patients can reach substantial size, with aneurysms as large as 4 to 6 cm in diameter having been infrequently encountered.

**Suggested Readings**

**GENERAL**

**ACUTE MYOCARDIAL INFARCTION**


**Assessment of Wall Motion and Function**


**Prognosis**


**Miscellaneous**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Stress echocardiography is based on the fundamental causal relationship between induced myocardial ischemia and left ventricular regional wall motion abnormalities. Although the first echocardiographic demonstration of an ischemic wall motion abnormality occurred over 40 years ago, it was not until the 1980s that improvements in image quality and the development of digital acquisition technology, or frame grabbers, made the clinical application of this method a possibility. The ability to digitize echocardiographic images reduced the problem of respiratory interference by permitting selection of cardiac cycles that were devoid of lung interference and the creation of cine loops that permitted side-by-side analysis of rest and stress images. This allowed more accurate interpretation of wall motion, largely by permitting relatively subtle changes in stress-induced wall motion to be detected. Digital technology also shortened the acquisition time for postexercise imaging and facilitated display, storage, and transmission of echocardiographic data. More than any other single factor, the application of digital imaging led to the rapid development of stress echocardiography as a practical, clinical tool.

**PHYSIOLOGIC BASIS**

In the 1930s, Tennant and Wiggers observed the relationship between systolic contraction and myocardial blood supply. With the induction of ischemia, these investigators demonstrated the rapid and predictable development of systolic bulging (or dyskinesis). This observation established the link between induced ischemia and transient regional myocardial dyssynergy, recorded echocardiographically as the development of wall
motion abnormality after the application of a stressor (Fig. 16.1). In the absence of a flow-limiting coronary stenosis, physiologic stress results in an increase in heart rate and contractility that is maintained via an increase in myocardial blood flow. Systolic wall thickening, endocardial excursion, and global contractility all increase, leading to a decrease in end-systolic volume (and an increase in the ejection fraction) compared with baseline. Although this response may be blunted in the setting of advanced age and/or hypertension or in the presence of β-blocker therapy, absence of the hypercontractile state in response to stress should generally be considered an abnormal response.

In the presence of a coronary stenosis, the increase in myocardial oxygen demand that occurs in response to stress is not matched by an appropriate increase in supply. If the supply–demand mismatch persists, a complex sequence of events known as the ischemic cascade will develop (Fig. 16.2). It should be recognized that the ischemic cascade is a generalization. The overlap of the parameters depicted in the schematic is intended to convey the variability that exists. That is, in an individual patient, the sequence and timing of the ischemic markers will vary. For example, ST-segment depression may occur early or later than depicted, or may not occur at all.

Soon after the development of a regional perfusion defect, a wall motion abnormality will occur, characterized echocardiographically as a reduction in systolic thickening and endocardial excursion. The severity of the wall motion abnormality (hypokinesis vs. dyskinesis) will depend on several factors, including the magnitude of the blood flow change, the spatial extent of the defect, the presence of collateral blood flow, left ventricular pressure and wall stress, and the duration of ischemia. Deterioration in regional wall motion, however, is a specific and predictable marker of regional ischemia that generally precedes such traditional manifestations as angina or electrocardiographic abnormalities.
FIGURE 16.1. A transient wall motion abnormality involving the mid and distal septum is demonstrated, during an episode of ischemia (A) and after resolution (B). Video 16-1B
FIGURE 16.2. The ischemic cascade is the term used to describe the sequence of events that occur after the onset of ischemia. The temporal abnormalities develop in a predictable sequence, as demonstrated in this schematic. Wall motion abnormalities detectable by echocardiography generally develop after a perfusion defect but before electrocardiographic changes or angina. Abn, abnormal; dysfcn, dysfunction; MBF, myocardial blood flow.

Once the stressor is eliminated, myocardial oxygen demand decreases and ischemia resolves. Normalization of wall motion may occur rapidly, although typically the complete recovery of normal function takes 1 to 2 minutes, largely depending on the severity and duration of ischemia. Stunned myocardium is the term applied when functional abnormalities persist after transient ischemia for a longer period. Although a reversible process, stunning may last days or even weeks if the ischemia is severe and prolonged.

The utility of echocardiography in conjunction with stress testing is contingent on the ability to record wall motion and left ventricular function at baseline and then to detect changes after the induction of stress, either exercise or pharmacologic (Table 16.1). At baseline, the presence of a regional wall motion abnormality generally implies the presence of previous
myocardial damage, in most cases due to myocardial infarction. Less often, cardiomyopathy and stunned or hibernating myocardium cause resting wall motion abnormalities. Regional deterioration of left ventricular function during stress is a specific marker of ischemia. Although exercise-induced wall motion abnormalities may occasionally occur in normal individuals after prolonged, intense exercise, this type of response during stress testing is usually the result of significant coronary disease. A global decrease in left ventricular function in response to stress, however, may be due to other causes, such as hypertension, valve disease, or cardiomyopathy. Therefore, by comparing regional wall motion at baseline and during stress, the presence of inducible ischemia can be detected and localized.

Although most of the useful information gathered during stress echocardiography is dependent on two-dimensional imaging and the analysis of regional and global left ventricular function, several other useful parameters should also be considered. For example, Doppler techniques can be applied to measure changes in stroke volume that occur during stress. Analysis of mitral inflow velocity and annular tissue Doppler velocity has been used to assess diastolic abnormalities in response to stress. This may be especially helpful in patients with exertional dyspnea. As is discussed later, Doppler imaging has particular utility in the evaluation of patients with valvular heart disease, prosthetic valves, and hypertrophic cardiomyopathy. Stress testing in these patients can provide valuable information and has been used to assess the effectiveness of therapy and to make decisions regarding the timing of interventions.

**METHODOLOGY**

Guidelines for the performance, interpretation, and application of stress echocardiography have been published by the American Society of Echocardiography (Pellikka et al., 2007). Echocardiographic imaging can be applied to both exercise and pharmacologic stress for the detection of myocardial ischemia (Table 16.2). Exercise echocardiography is most often performed using either treadmill or bicycle (upright or supine) exercise. The most common pharmacologic agent used in conjunction with echocardiography is dobutamine. Much less commonly used stressors include
isometric exercise such as handgrip, vasodilators such as dipyridamole or adenosine, and pacing, usually through a transesophageal approach. Modalities may even be combined. For example, handgrip may be used during dobutamine stress to increase workload and improve sensitivity.

**Treadmill Exercise**

Treadmill exercise is the most common form of stress testing in the United States. It provides a plethora of useful clinical information that has both diagnostic and prognostic value. These include exercise capacity, blood pressure response, and arrhythmias. It is safe and well tolerated and can be applied to a large percentage of the patients referred for stress testing. Because clinicians have become comfortable with this form of stress testing and because of the widespread availability of treadmill equipment, it is logical that stress echocardiography should be applied to this technique.

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<tr>
<th>Table 16.1</th>
<th>CAUSES OF WALL MOTION ABNORMALITIES</th>
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<tr>
<td><strong>Wall Motion Abnormalities at Rest</strong></td>
<td><strong>Wall Motion Abnormalities During Stress</strong></td>
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<td>Infarction/scar</td>
<td>Ischemia</td>
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<td>Myocarditis</td>
<td>Marked increase in blood pressure</td>
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<td>Left bundle branch block</td>
<td>Cardiomyopathy</td>
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<tr>
<td>Hypertension/afterload mismatch</td>
<td>Rate-dependent left bundle branch block</td>
</tr>
<tr>
<td>Hibernating myocardium</td>
<td>Pulmonary hypertension</td>
</tr>
<tr>
<td>Stunned myocardium</td>
<td></td>
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<tr>
<td>Toxins (e.g., alcohol)</td>
<td></td>
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<tr>
<td>Postoperative state</td>
<td></td>
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<tr>
<td>Paced rhythm</td>
<td></td>
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<tr>
<td>Right ventricular volume/pressure overload</td>
<td></td>
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<table>
<thead>
<tr>
<th>Table 16.2</th>
<th>TYPES OF STRESSORS USED IN STRESS ECHOCARDIOGRAPHY</th>
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Echocardiographic imaging in conjunction with treadmill exercise is intended not to alter the standard exercise protocol. Imaging is performed before and immediately after treadmill exercise, without affecting the exercise portion of the test. Thus, the advantages of treadmill exercise echocardiography include preserving the additional information already available from treadmill exercise, the widespread availability of this form of stress, and the relatively simple protocol created by the addition of echocardiographic imaging. The primary disadvantage of treadmill echocardiography stems from the difficulty in obtaining images while patients walk in an upright position. For this reason, imaging is performed in the immediate postexercise period. Because ischemia may resolve quickly after termination of exercise, it is incumbent on the operator to complete postexercise imaging as soon as possible, certainly within 1 to 1.5 minutes after exercise. As soon as the exercise test ends, the patient must step off the treadmill and assume a recumbent position so that imaging can be completed quickly.

Although any available transthoracic view can be used in exercise echocardiography protocols, the traditional approach has included the parasternal long- and short-axis and the apical four- and two-chamber views. The apical long-axis, the subcostal four-chamber, and short-axis views may also be included at the discretion of the operator. Image acquisition can be individualized, depending on available ultrasound windows but is always intended to acquire images that provide more than one opportunity to examine each region of the left ventricle. In addition, some attention to right ventricular function and wall motion should also be a part of most stress echocardiographic protocols. Figure 16.3 is an example of a treadmill exercise echocardiogram showing the apical four- and two-chamber views. The resting or baseline images are on the left and the postexercise images are
on the right. Each quadrant contains annotated information about heart rate, stage, time of acquisition, etc.

Resolution of induced wall motion abnormalities before postexercise imaging can be completed is a cause of false-negative results (Fig. 16.4). In this example, with treadmill exercise, anterior ischemia is evident in the long- and short-axis views, less obvious in the four-chamber view, and no longer present in the two-chamber view. This is because the wall motion abnormality resolved over the course of poststress image acquisition. As the heart rate decreases postexercise, wall motion recovers. If an adequate workload is achieved and postexercise images are acquired within 1 minute, the likelihood of a false-negative finding is minimized. Figure 16.5 is another example of rapid recovery, in this case during supine bicycle exercise. Note the obvious apical wall motion abnormality at peak exercise. Postexercise, there is near normalization of wall motion. Why some wall motion abnormalities normalize very quickly is not completely understood. Several investigators have compared peak and postexercise imaging during bicycle protocols and examined the frequency and possible causes of rapid recovery of wall motion abnormalities. Exercise duration, extent of disease, workload achieved, or medical therapy is not predictive of rapid recovery. Conversely, wall motion abnormalities that persist into late recovery generally indicate more severe epicardial coronary disease and/or multivessel disease.
FIGURE 16.3. The standard format to display stress echocardiographic images. This example, from a treadmill exercise echocardiogram, demonstrates the four-chamber view at the top and the two-chamber images at the bottom. The resting study is displayed on the left and the immediate postexercise images are on the right. Note that heart rate and time of image acquisition are displayed for each quad.
FIGURE 16.4. An example of rapid recovery of abnormal wall motion is demonstrated in a patient undergoing treadmill exercise. The resting study is normal. Postexercise, septal, and apical ischemia develops and is evident in the long-and short-axis views. The abnormality is less apparent in the four-chamber view and nearly resolved in the two-chamber view. Image acquisition was completed in approximately 75 s.
FIGURE 16.5. This study demonstrates rapid recovery during supine bicycle exercise. An obvious apical wall motion abnormality develops during exercise and is recorded at peak (right upper and left lower quads). Postexercise (right lower quad) wall motion is nearly normal. This is especially apparent in the two-chamber view (B, see videos). [Video]
Bicycle Ergometry

Stationary bicycle ergometry was the first form of exercise used in conjunction with echocardiography. Initially, upright bicycle ergometers were used and imaging was performed during and after exercise. Later, supine bicycle systems that permit a variety of patient positions became popular. By providing an approximately 30-degree head-up tilt of the patient, a balance between comfort and image quality can be achieved. To perform graded exercise, patients pedal at a constant cadence at increasing levels of resistance.

The primary advantage of bicycle stress echocardiography is the ability to
image throughout exercise, particularly at peak stress. This not only avoids the potential problem of rapid recovery but also permits the onset of a wall motion abnormality to be documented. Exercise-induced wall motion abnormalities are more frequent, more extensive, and more easily visualized at peak compared with postexercise. Imaging at intermediate stages can also be analyzed and this may improve the sensitivity of the test by facilitating the detection of a biphasic response. The application of contrast to stress echocardiography is also easier using bicycle exercise compared with treadmill exercise. The major disadvantage of bicycle exercise echocardiography is the problem of workload. Some patients find bicycling in the supine position very difficult, which may prevent an adequate level of stress to be achieved. However, supine posture appears to facilitate the induction of ischemia, perhaps by increasing venous return and preload or because it is associated with a greater blood pressure response. As a result, ischemia occurs at a lower heart rate during supine versus upright exercise. Again, the newer generation of bicycle ergometers increases the comfort and tolerability of supine exercise.

**Dobutamine Stress Echocardiography**

Dobutamine is a synthetic catecholamine that causes both inotropic and chronotropic effects through its affinity for $\beta_1$, $\beta_2$, and $\alpha$ receptors in the myocardium and vasculature. Because of differences in affinity, the cardiovascular effects of dobutamine are dose dependent, with augmented contractility occurring at lower doses followed by a progressive chronotropic response at increasing doses. Peripheral effects may result in either predominant vasoconstriction or vasodilation, so changes in vascular resistance (i.e., blood pressure) are unpredictable. The net effect of these interactions is a combined increase in contractility and heart rate with an associated increase in myocardial oxygen demand. If coronary flow reserve is limited, myocardial oxygen demands will eventually exceed supply and ischemia will develop.

It should be noted that the mechanism of action of dobutamine is not identical to exercise. For example, the change in venous return that typically accompanies leg exercise is less pronounced with dobutamine. In addition, the autonomic nervous system–mediated changes in systemic and pulmonary
vascular resistance are quite different with exercise compared with dobutamine. These differences have implications for the determinants of the ischemic threshold during exercise and pharmacologic stress. For example, heart rate response is less important with dobutamine compared with exercise, and ischemia can often be induced even if target heart rate is not attained. The lower heart rate achieved during dobutamine infusion is offset by the greater augmentation in contractility. Thus, the two modalities are both capable of producing ischemia but do so by different mechanisms. As a result, the parameters that define an adequate level of stress are also different.

The primary application of dobutamine echocardiography is in patients unable to exercise adequately. The ability of dobutamine to mimic the cardiac effects of exercise, coupled with the safety and versatility of the test, has contributed to the popularity of dobutamine echocardiography. A related application has been for the detection of viable myocardium in the setting of either stunned or hibernating myocardium. As with exercise, the goal is to produce a graded increase in cardiac workload that can be monitored for the development of ischemia. To do this, dobutamine is infused at increasing rates for 3- to 5-minute stages. Although this duration at each stage is insufficient to produce a steady-state effect, it generally yields a gradual and well-tolerated increase in both contractility and heart rate. Atropine is frequently used to augment the heart rate response. The use of atropine for this purpose has been shown to improve sensitivity, especially in patients taking β blockers. Although there is no universally agreed-on protocol for dobutamine administration, a commonly used approach is outlined in Table 16.3.

<table>
<thead>
<tr>
<th>Table 16.3</th>
<th>PROTOCOL FOR DOBUTAMINE STRESS ECHOCARDIOGRAPHY</th>
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</thead>
<tbody>
<tr>
<td>Patient is prepared for standard stress testing</td>
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<tr>
<td>Intravenous access is obtained</td>
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<tr>
<td>Digital images are acquired at baseline (these loops are displayed and used as reference throughout the infusion)</td>
<td></td>
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<tr>
<td>A decision regarding the need for contrast administration is made at this point</td>
<td></td>
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<tr>
<td>Continuous electrocardiogram and blood pressure monitoring are established</td>
<td></td>
</tr>
<tr>
<td>Dobutamine infusion is begun at a dose of 5 (or 10) µg/kg/min</td>
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The infusion rate is increased every 3 minutes to doses of 10, 20, 30, and 40 μg/kg/min. The echocardiogram, electrocardiogram, and blood pressure are monitored continuously. Low-dose images are acquired at either 5 or 10 μg/kg/min (at the first sign of increased contractility).

Atropine in aliquots of 0.5–1.0 mg can be given during the mid- and high-dose stages to augment the heart rate response. Middose images are acquired at either 20 or 30 μg/kg/min. Peak images are acquired before termination of the infusion. Poststress images are recorded after return to baseline.

The patient is monitored until he or she returns to baseline status.

The test may be terminated when one of several end points are reached (Table 16.4). Although such guidelines are essential, the decision to terminate the dobutamine infusion must be individualized. The ability to monitor wall motion is critically important to that decision. For example, atypical symptoms not associated with objective evidence of ischemia (i.e., a new wall motion abnormality) are not necessarily a reason to stop the test. A subtle or limited wall motion abnormality, particularly if well tolerated, also does not mandate termination. To assess the true extent of coronary disease, it is often prudent to continue the test under close monitoring. A decrease in blood pressure is sometimes an indication of extensive ischemia. During dobutamine infusion, however, hypotension may instead indicate the development of a left ventricular outflow tract gradient, and this can be easily recognized using Doppler imaging (Fig. 16.6). Finally, electrocardiographic evidence of ischemia is less reliable during dobutamine infusion than it is during exercise testing. Thus, neither ST-segment depression nor elevation occurring in the absence of a wall motion abnormality or typical symptoms is sufficient reason for terminating the dobutamine infusion.

**Table 16.4** END POINTS AND REASONS TO TERMINATE THE DOBUTAMINE INFUSION DURING STRESS TESTING

<table>
<thead>
<tr>
<th>End Point</th>
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<tbody>
<tr>
<td>Exceeding target heart rate of 85% age-predicted maximum</td>
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<tr>
<td>Development of significant angina&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Recognition of a new wall motion abnormality&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>A decrease in systolic blood pressure &gt;20 mm Hg from baseline&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Arrhythmias such as atrial fibrillation or nonsustained ventricular tachycardia</td>
</tr>
<tr>
<td>Limiting side effects or symptoms</td>
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</table>
Decision may depend on clinical status of the patient and presence/extent of wall motion abnormality.

Decision may depend on clinical status of the patient and extent/severity of the wall motion abnormality.

Decision may depend on clinical status and left ventricular function and/or outflow tract gradient.

FIGURE 16.6. An example of an induced left ventricular outflow tract gradient during dobutamine stress testing. This occurred in a patient with severe left ventricular hypertrophy who developed hyperdynamic wall motion at peak stress. In panel (A), the resting study shows normal wall motion and a small end-systolic cavity. At baseline (B), a 20 mm Hg late-peaking gradient is present. At peak stress (C), there is considerable variability, but the left ventricular gradient has increased to as high as 100 mm Hg.
The safety of dobutamine stress echocardiography has been examined in several series. Because of the short half-life of dobutamine, inducible ischemia can be readily reversed through termination of the infusion. In severe cases or when the ischemic manifestations persist, a short-acting intravenous β blocker (such as metoprolol or esmolol) is effective. The most common side effects associated with dobutamine infusion are minor arrhythmias such as premature ventricular contractions and atrial arrhythmias and minor symptoms such as palpitations or anxiety. Nonsustained ventricular tachycardia occurs in approximately 3% of patients and generally terminates spontaneously or can be successfully treated with an intravenous β blocker. Rare isolated serious complications have been reported.

There are no absolute contraindications to dobutamine stress testing. Unstable patients, such as those with uncompensated heart failure for unstable angina, should rarely be subjected to stress testing of any kind. Dobutamine echocardiography has been safely performed in patients with recent myocardial infarction, extensive left ventricular dysfunction, abdominal aortic aneurysm, syncope, aortic stenosis, hypertrophic cardiomyopathy, history of ventricular tachycardia, and aborted sudden death. In each instance, the value of the expected diagnostic information must be balanced with the individualized risk to the patient. Unlike dipyridamole, dobutamine can be safely used in patients with bronchospastic lung disease.

**Dipyridamole and Adenosine**
Potent vasodilators such as dipyridamole and adenosine have been used in conjunction with echocardiography for the detection of coronary artery disease. Unlike dobutamine, these agents work by creating maldistribution of blood flow, that is, by preventing the normal increase in flow in areas supplied by stenotic coronary arteries. In more extreme cases, flow may actually be diverted away from abnormal regions (the so-called coronary steal), resulting in true ischemia. Adenosine is a potent and short-acting direct coronary vasodilator. Dipyridamole is slower acting and its effects result from inhibition of adenosine uptake. With both agents, the development of a wall motion abnormality is predicated on the ability to create sufficient maldistribution of regional blood flow to result in an ischemia-induced wall motion abnormality. Compared with dobutamine, these changes tend to be more subtle and short-lived.

The safety of dipyridamole and adenosine echocardiography is well established. However, both agents are substantially less popular compared with dobutamine as a pharmacologic stressor during echocardiographic imaging. The primary reason for this relates to the mechanism of action. It is conceivable that redistribution of regional blood flow can occur without an associated wall motion abnormality. Thus, vasodilator stress agents may be better suited to imaging techniques that rely on relative changes in perfusion rather than the development of a wall motion abnormality. This is the reason that dipyridamole and adenosine have been commonly used with nuclear imaging techniques.

**Three-Dimensional Stress Echocardiography**

The application of real-time three-dimensional imaging to stress echocardiography is now feasible and offers several theoretical advantages. A full volume data set can be acquired and then sliced and displayed in variety of views. For example, a series of parallel short-axis scans can be derived and analyzed (called “multislice”). With as many nine short-axis images available for analysis, this approach permits virtually the entire left ventricle to be examined. Alternatively, traditional orthogonal planes can be derived from the volumetric data set, a technique called multiplane imaging. The advantage of this approach is that each plane can be adjusted to ensure that it is properly aligned. These two methods for three-dimensional stress imaging have
recently been compared (Yoshitani et al., 2009). Figure 16.7 is an example of a three-dimensional dobutamine echocardiography analyzed using both multislice and multiplane techniques. Although both methods permitted detection of the anteroapical wall motion abnormality, only the multislice images (Fig. 16.7B) demonstrated the inferior ischemia. This study shows the versatility of three-dimensional techniques which should contribute positively to overall accuracy.

Three-dimensional stress echocardiography has several strengths. With treadmill exercise, the acquisition of the entire left ventricle in a single volume shortens postexercise imaging time. Three-dimensional echocardiography also allows a more complete examination of the left ventricle than would be possible with two-dimensional imaging alone. In addition, this approach permits precise alignment and matching of rest and stress views that facilitates detection of subtle abnormalities. Finally, it is well established that three-dimensional echocardiography is a more accurate means of measuring left ventricular volume and ejection fraction. With stress, the ability to compare, for example, left ventricular end-systolic volume
before and after exercise has both diagnostic and prognostic utility and this determination has been improved through the use of three-dimensional imaging. The major limitations of three-dimensional stress echocardiography continue to be image quality and frame rate. As technology continues to improve, these technical issues should become less of a problem, allowing this modality to develop as a practical approach to stress echocardiography.

**CHOOSING AMONG THE DIFFERENT STRESS MODALITIES**

The wide range of choices in stress testing has the potential to create confusion for the clinician trying to select the optimal test for any given patient. Is the stress test necessary? Is any form of imaging required? Which stress modality is better: exercise or pharmacologic? What type of exercise works best with a given form of imaging? Although some of these decisions must be individualized, general guidelines can be provided. It is well established that all forms of imaging increase the accuracy of stress testing, particularly in those patients who have had or are likely to have a nondiagnostic stress electrocardiogram (ECG). Imaging also provides information on the location and extent of disease, contributing both to the diagnostic and prognostic value of the test. General guidelines for choosing among the various modalities are provided in Table 16.5.

For most patients, exercise is the preferred form of stress, provided the patient is capable of adequately performing either treadmill or bicycle exercise. The additional information available during an exercise stress test provides most of the advantage over pharmacologic testing. When compared in the same group of patients, exercise has generally been a more sensitive test for the detection of coronary disease compared with dobutamine. However, the superiority of exercise is modest and has not been a universal finding. In most clinical situations, exercise is preferred for the reasons listed previously. An exception to this general rule is when myocardial viability is an issue. In such cases, pharmacologic stress testing with dobutamine is preferred. Thus, dobutamine stress echocardiography is generally limited to patients who are unable to exercise adequately or to specifically address the question of viability.
When nonexercise stress is deemed necessary and echocardiography is the imaging modality, the weight of evidence and the general experience support the use of dobutamine as the stress agent. Because dobutamine is more likely to cause true ischemia rather than merely a flow mismatch, the induction of a wall motion abnormality, detectable with echocardiography, is more likely. For the induction of a perfusion abnormality, both vasodilators and dobutamine have been employed. One study (Kowatsch et al., 2007) suggested that dobutamine was equivalent to adenosine for induction of perfusion abnormalities that could be detected with echocardiography. However, because dobutamine is superior to vasodilators for inducing wall motion abnormalities and perhaps equivalent for creating perfusion mismatch, it is likely that dobutamine will remain the preferred pharmacologic stressor for the near future.

Among the various forms of exercise echocardiography, both bicycle and treadmill techniques have been used successfully and are safe and well tolerated. Bicycle exercise has as its primary advantage the opportunity to image throughout exercise. The larger general experience with treadmill stress testing and the comfort that most clinicians have with the methodology and information available during a treadmill test must also be considered. Few studies have directly compared treadmill and bicycle exercise. In one series (Badruddin et al., 1999), in which treadmill exercise and supine bicycle exercise were performed in random order on 74 patients with suspected coronary disease, the bicycle technique was found to be slightly more sensitive, whereas treadmill exercise was slightly more specific. Although mean exercise duration was considerably longer for bicycle exercise, overall workload, expressed as double product, was similar for the two tests. When an ischemic wall motion abnormality was induced, the extent of the defect was greater with bicycle exercise, most likely because imaging was performed during rather than after stress. Thus, both the treadmill and the bicycle are acceptable forms of stress when echocardiographic imaging is used. Methods that permit imaging during exercise may allow both the presence and the extent of disease to be more accurately determined. These advantages must be balanced by patient preference, exercise ability, and the availability of other types of diagnostic and prognostic data.
### Table 16.5 COMPARISON OF THE DIFFERENT STRESS METHODOLOGIES IN VARIOUS CLINICAL SITUATIONS

<table>
<thead>
<tr>
<th>Clinical Question</th>
<th>Treadmill</th>
<th>Bicycle</th>
<th>Dobutamine</th>
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<tbody>
<tr>
<td>Chest pain evaluation</td>
<td>++</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>Postmyocardial infarction risk</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Viability</td>
<td>–</td>
<td>–</td>
<td>++</td>
</tr>
<tr>
<td>Evaluation of dyspnea/fatigue&lt;sup&gt;a&lt;/sup&gt;</td>
<td>+</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>Preoperative risk assessment</td>
<td>+</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Severity of valve disease&lt;sup&gt;a&lt;/sup&gt;</td>
<td>–</td>
<td>++</td>
<td>–</td>
</tr>
<tr>
<td>Pulmonary hypertension&lt;sup&gt;a&lt;/sup&gt;</td>
<td>–</td>
<td>++</td>
<td>–</td>
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<sup>a</sup>Inclusion of Doppler parameters is recommended.

### INTERPRETATION OF STRESS ECHOCARDIOGRAPHY

Most stress echocardiograms are analyzed on the basis of a subjective assessment of regional wall motion, comparing wall thickening and endocardial excursion at baseline and during stress. The rest or baseline echocardiogram is first examined for the presence of global systolic dysfunction or regional wall motion abnormalities. The presence of baseline wall motion abnormalities suggests previous myocardial infarction. Other less likely possibilities include stunned or hibernating myocardium or a form of focal cardiomyopathy. Subtle abnormalities at baseline, such as hypokinesis of the inferior wall, may occur in the absence of coronary artery disease and represent a cause of false-positive results. Interventricular septal motion may be specifically altered in the presence of left bundle branch block (Fig. 16.8), the postoperative state, ventricular pacing, or pressure or volume overload of the right ventricle.
FIGURE 16.8. An example of LBBB during exercise stress testing. The study demonstrates resting global hypokinesis with septal dyskinesis. Post-exercise, there is augmentation of left ventricular function, although the septum remains dyskinetic. This patient had LBBB with nonischemic cardiomyopathy. Video 16-8a
Regardless of the form of stress, the normal response is the development of hyperdynamic wall motion (Fig. 16.9 and Table 16.6). Although this is generally true, some heterogeneity may be expected and not all left ventricular segments will necessarily display the same degree of hypercontractility. When examined quantitatively, this variability in the normal response is apparent and even mild hypokinesis may be present in normal subjects. Despite this caveat, a global increase in contractility should still be regarded as the normal response. Lack of hyperkinesis is abnormal and is most often caused by the development of regional myocardial ischemia. Other factors may also affect the ability to develop hyperkinesis. These include the presence of a nonischemic cardiomyopathy, treatment with β-blocker agents, certain valve diseases, left bundle branch block, and severe hypertension. In addition, submaximal exercise resulting in attainment of a low workload is often associated with the absence of a hyperkinetic response. If postexercise imaging is performed after treadmill exercise, an excessive delay in image acquisition may miss the transient hyperkinesis and lead to a misinterpretation.
FIGURE 16.9. An example of a normal treadmill exercise echocardiogram, demonstrating a hyperdynamic response to stress, is provided. The resting study is on the left and postexercise images are on the right. Mild left ventricular hypertrophy is present. Video 16-9B

Video 16-9B

Table 16.6 COMBINATION OF REST AND STRESS WALL MOTION
Early relaxation is another potentially confusing type of wall motion in which a wall segment moves outward, that is, relaxes, at end-systole earlier than the adjoining segments (Fig. 16.10). This can occur at rest, but is often exaggerated during stress. It is usually a normal variant. Tardokinesis, a term used to describe late inward motion of myocardium at end-systole, is another confounding type of wall motion response to stress (Fig. 16.11). It usually involves the basal inferior or posterior wall where it is typically a normal variant. In this setting, preserved systolic thickening is evidence against ischemia. However, tardokinesis may be a manifestation of ischemia in which case systolic wall thickening is absent. Analyzing wall motion frame by frame or trimming a cine loop to include only the first half of systole will help identify tardokinesis and distinguish it from other wall motion responses.

A limitation of this approach to interpretation is the subjective and nonquantitative nature of wall motion analysis. Several studies have examined the reproducibility of subjective wall motion scoring. In general, experienced interpreters agree in the majority of cases, and overall accuracy is reasonable. More quantitative and objective approaches, however, would have obvious advantages. Historically, such attempts have been limited by image quality and translational motion. In addition, the complexity and time-consuming nature of some methods limit their acceptance. Calculation of the ejection fraction at rest and during stress, for example, is fraught with technical challenges and rarely performed in routine practice. A more practical approach involves the estimation of left ventricular volume changes during stress. The normal response to stress includes a decrease in both end-systolic and end-diastolic volume that can be visually appreciated using side-
by-side inspection of images. Failure of the ventricular size to decrease is an abnormal response. An increase in volume with stress, especially the end-systolic volume, often indicates severe and extensive (i.e., multivessel) disease. An example of this phenomenon is provided in Figure 16.12. In this case, apical dilation was due to a severe stenosis of the proximal left anterior descending coronary artery. Figure 16.13 is an example of an abnormal volume response in a patient with extensive coronary disease. Note the increase in left ventricular systolic dimension, especially in the four-chamber view. The right ventricle also dilates, in this case, due to proximal right coronary artery ischemia. Although systolic dilation is an ominous sign during treadmill exercise, a mild increase in chamber volume may be seen in normal subjects during supine bicycle exercise. With this form of stress, elevation of the legs increases venous return throughout exercise so that left ventricular dilation at peak exercise may be a nonspecific finding. Once exercise stops, the cavity usually will rapidly decrease in size.
Tardokinesis, a temporal delay in endocardial excursion, is seen in the basal inferior wall (arrow) in the two-chamber view.
When image quality is suboptimal, wall motion analysis can be augmented through the use of contrast agents that improve endocardial border definition and increase both the confidence and the accuracy of diagnosis. In general, when two or more left ventricular segments are not seen on the resting study, use of contrast should be considered. The contrast can be delivered either as intermittent boluses of a diluted solution or as a continuous infusion. Using low mechanical index (less than 0.5) imaging, border delineation is improved and both wall thickening and endocardial excursion are better evaluated. Figure 16.14 demonstrates endocardial definition with the use of contrast, allowing an area of anteroseptal ischemia to be accurately identified. In a randomized, crossover single-center study (Plana et al., 2008), the use of contrast during dobutamine stress echocardiography increased the percentage of interpretable segments, both at baseline and with stress. This led to an increase in overall accuracy and a higher level of confidence in interpretation (Fig. 16.15).
FIGURE 16.12. This treadmill exercise echocardiogram demonstrates an abnormal left ventricular volume response to stress. The resting study is normal. Postexercise, there is evidence of anteroseptal, apical, and lateral ischemia, resulting in dilation of the left ventricle. Video 16-12
This treadmill exercise echocardiogram demonstrates an abnormal left ventricular volume response. These frames were taken at end-systole. Resting images are on the left and postexercise on the right. The postexercise images demonstrate a larger end-systolic volume compared with baseline, suggesting chamber enlargement in response to stress.

Several schemes for interpreting and reporting stress echocardiographic results are in clinical use. One approach divides the left ventricle into 16 segments (Fig. 16.16, left) and then grades each segment on a scale from 1 to 4 in which 1 is considered normal, 2 indicates hypokinesis, 3 indicates akinesis, and 4 corresponds to dyskinesis. Wall motion is analyzed at baseline, and a wall motion score index is generated according to the formula:

$$\text{Wall motion score index} = \frac{\text{Segment scores}}{\text{No. of segments scored}} \quad \text{[Eq. 16.1]}$$
FIGURE 16.14. This is an example of a technically difficult treadmill exercise echocardiogram. Endocardial definition on the noncontrast-enhanced study was poor. With the addition of contrast, endocardial definition improved. In this long-axis view, taken before (A) and immediately after (B) exercise, distal septal ischemia is present. [0]

Video 16-14A
FIGURE 16.15. The use of contrast to improve endocardial border definition has an impact on the sensitivity and specificity of the test. The left panels demonstrate the effect of contrast on sensitivity and specificity in those studies grouped by confidence of interpretation of the unenhanced images. A trend toward improved specificity is demonstrated. On the right, in those studies in which the interpreter was confident based on the unenhanced image, contrast added no additional benefit with respect to accuracy. (Reprinted with permission from Plana JC, Mikati IA, Dokainish H, et al. A randomized cross-over study for evaluation of the effect of image optimization with contrast on the diagnostic accuracy of dobutamine echocardiography in coronary artery disease. J Am Coll Cardiol Imaging 2008;1(2):145–152. Copyright © 2008 American College of Cardiology Foundation.)
Analysis of stress echocardiograms should include regional wall motion assessment. This entails dividing the left ventricle into regions that can be analyzed either from parasternal or from apical views. On the left, the standard 16-segment model is demonstrated. On the right, a slightly different approach to segmentation involves the 17-segment model in which the apical cap is analyzed separately in the four-chamber and two-chamber views.

A 17-segment model, which includes an apical cap, is another option and has the advantage of being more compatible with most nuclear imaging schemes (Fig. 16.16, right). Both of these schemes have been endorsed by the American Society of Echocardiography. A similar approach is then taken for analysis of wall motion during stress. In this case, the development of hyperkinesis is assumed to be normal and assigned a score of 1. Thus, a normal study would be associated with a wall motion score index of 1 at both baseline and stress. Any score greater than 1 would indicate abnormal wall motion. An increase in score would indicate either an increase in the extent and/or the severity of a wall motion abnormality. An example of wall motion scoring of a stress echocardiogram is provided in Figure 16.17. This approach has several advantages. It provides a systematic approach to wall motion analysis and encourages a thorough and standardized approach. Furthermore, it acknowledges the subjectivity of wall motion analysis but provides a quantitative reporting scheme that allows studies to be compared. The prognostic value of wall motion score index has been demonstrated in several studies.
Categorization of Wall Motion

Hypokinesis is the most subtle form of abnormal wall motion. It is defined as the preservation of some degree of thickening and inward motion of the endocardium during systole but less than normal. It has been defined arbitrarily as less than 5 mm of endocardial excursion. Distinguishing between normal wall motion and hypokinesis can be challenging, particularly in the setting of advanced age or β-blocker therapy. Hypokinesis is most likely to be truly abnormal if it is limited to a region or territory that corresponds to the distribution of one coronary artery and is associated with normal (or hyperdynamic) wall motion elsewhere. Akinesis is defined as the absence of systolic myocardial thickening and endocardial excursion. Bear in mind that translational motion of the heart during systole can create the illusion of akinesis. However, wall thickening is less translation dependent and should be relied on in such cases. Dyskinesis is the most extreme form of a wall motion abnormality and is defined as systolic thinning and outward motion or bulging of the myocardium during systole. A left ventricular segment that is thin and/or highly echogenic suggests the presence of scar. Other less common wall motion responses have also been recognized. For example, early relaxation is used to describe a segment that appears to contract in early systole and then relaxes or dilates earlier than the other walls. It is a common cause of false-positive results because it is most likely a normal variant and not associated with ischemia. Again, trimming the cine loop to include only the first half of systole is a useful way to identify early relaxation and differentiate it from truly abnormal wall motion.

Wall Motion Response to Stress

By comparing wall motion at baseline and during stress, valuable diagnostic information is available (Table 16.6). Wall motion that increases or augments during stress is generally considered normal. The development of a wall motion abnormality during stress in an area normal at rest is most suggestive of ischemia. Segments that are abnormal at rest and remain unchanged with stress are generally best interpreted as showing evidence of infarction without additional ischemia. Hypokinetic areas that worsen during stress are usually labeled ischemic. These may represent a combination of previous nontransmural infarction and induced ischemia. Segments that are akinetic or
dyskinetic at baseline, even if wall motion worsens during stress, are best interpreted as indicating infarction, and the ability to detect additional ischemia in such segments is limited. Occasionally, wall motion appears normal at rest and is unchanged with stress, that is, neither hyper- nor hypokinetic. Some readers consider this abnormal and report it as an ischemic response. Although this may be the case, it is also the cause of many false-positive findings. Bear in mind that lack of hyperkinesis has multiple etiologies, including low workload, delayed postexercise imaging, β blockade, and cardiomyopathy. Elderly patients, especially women, may be unable to manifest a frankly hyperkinetic response. Therefore, to minimize false-positive results, consider these other possibilities before interpreting lack of hyperkinesis as an ischemic end point. A marked increase in blood pressure during exercise can also prevent the development of hyperkinesis or even result in global hypokinesis. An example of such a response is provided in Figure 16.18. Despite an adequate level of exercise and an appropriate heart rate response, the peak-exercise views are unchanged or, in some areas, mildly hypokinetic. This was due to a marked increase in blood pressure during exercise.

FIGURE 16.17. An example of a stress echocardiographic report is provided, including a regional wall motion scoring summary. LVSI, left ventricle score index; %FM, percentage of normally functioning segments; LAD, left anterior descending; LCX, left circumflex; MI, myocardial infarction; RCA, right coronary artery. See text for details.
Finally, segments abnormal at baseline that improve with stress are uncommon and represent a special category. During exercise testing, these most likely indicate either a normal response or a localized abnormality in which the improvement is due to tethering from the surrounding normal myocardium. With dobutamine, however, improvement may indicate viability and the potential for recovery after revascularization. This topic is covered later in this chapter.

**FIGURE 16.18.** This exercise echocardiogram was performed in a patient who developed marked hypertension in response to exercise. The significant increase in blood pressure resulted in mild global hypokinesis. Failure to develop hyperdynamic wall motion is an abnormal response but in this case was due to afterload mismatch.
Strain Imaging

To provide a more quantitative approach, strain imaging has been applied to stress echocardiography. This approach relies on speckle tracking to quantify myocardial deformation in response to applied stress. Strain has been proposed as an objective, quantifiable marker of ischemia during stress testing. Experimental studies have shown that strain is affected early in the course of ischemia and therefore could be a more sensitive marker of disease. The potential to identify and even quantify subtle manifestations of ischemia is an attractive feature of strain imaging. The theoretic advantages of strain imaging include a relative independence of translational motion and
tethering, its inherently quantitative nature, the ability to distinguish active from passive motion, and the potential to examine wall motion throughout the cardiac cycle. Using modern equipment, strain can now be derived automatically and simultaneously from all areas within the heart. The utility of strain imaging to assess resting left ventricular function is discussed elsewhere, but most studies have relied on global longitudinal strain (GLS) as the best and most reproducible marker of dysfunction (Fig. 16.19). How this concept will be applied to stress echocardiography is more problematic and there are several questions that must be answered. First, the ability to obtain reproducible strain data at higher heart rates is difficult. Second, the normal response of strain to exercise and pharmacologic stress has yet to be clearly defined. And finally, what will define an abnormal response and how will that contribute to the data already available? Identifying a regional equivalent of GLS will be challenging. Perhaps a change in GLS from rest to stress will be global indicator of ischemia, similar to an increase in end-systolic volume. To date, most studies involving strain imaging in conjunction with stress echocardiography have utilized dobutamine as the stressor. The results have been modest, with most studies reporting reasonable sensitivity but lower specificity. Further improvements in technology and additional clinical studies will be required before strain echo during stress testing will be ready for routine clinical use.

FIGURE 16.19. An example of global longitudinal strain imaging during dobutamine stress. There is abnormal strain at baseline in the basal anterolateral region (A). At peak stress (B), a much larger area of reduced strain is evident, involving a significant portion of the anterior, lateral, and posterior walls. On angiography, the patient had distal left main coronary narrowing and total occlusion of the proximal left circumflex artery.
A practical application of stress echocardiography is to predict the presence of disease in specific coronary arteries or branches. The relationship between left ventricular segments or territories and the corresponding artery distribution is covered in Chapters 5 and 15. A similar approach is applied to stress echocardiography. By recording the left ventricle in multiple views, an evaluation of the territories of each of the three main coronary arteries is possible. This allows a prediction of both the location and the extent of disease to be made on the basis of wall motion. In general, stress echocardiography is more sensitive in patients with multivessel disease compared with single-vessel disease and more accurate for specifically identifying disease in the left anterior descending artery or right coronary artery compared with the left circumflex artery. Because of the variability in coronary artery distribution, accurate differentiation between lesions of the right coronary artery and left circumflex artery is not always possible.

Figure 16.20 is an example of localized apical ischemia induced during dobutamine echocardiography. Wall motion is normal at the 20 μg/kg/min stage (heart rate, 72 bpm), but apical dyskinesis develops at the next stage, associated with a much higher heart rate. Figure 16.21 shows inferior ischemia in a patient with no prior history of heart disease. At baseline, wall motion is normal. With stress, the inferior wall becomes hypokinetic, although the most helpful indicator of ischemia is the absence of inferior wall
thickening. In Figure 16.22, multivessel ischemia is demonstrated using dobutamine stress. This patient had a prior history of inferior myocardial infarction. Note the delayed normalization of left ventricular function during recovery. In Figure 16.23, ischemia involving the septum and inferior wall during bicycle stress echocardiography is apparent. Figure 16.24 is an example of extensive anteroapical and lateral ischemia during treadmill exercise. Postexercise left ventricular dilation occurs as a result of the extensive ischemia. At catheterization, the patient had disease of left anterior descending and circumflex coronary arteries.

FIGURE 16.20. An example of an abnormal dobutamine stress echocardiogram. The four-chamber view is shown and demonstrates apical ischemia. The abnormality is apparent only at peak stress (lower right quad).
CORRELATION WITH SYMPTOMS AND ELECTROCARDIOGRAPHIC CHANGES

It should be apparent that the analysis of the stress echocardiogram is only one component of the comprehensive stress test and that the other parameters, including the development of symptoms and/or ECG changes, cannot be ignored. In virtually every study that has examined the question, wall motion has been shown to be more sensitive and specific than either symptoms or ST-segment changes for the detection of coronary artery disease. In most instances, there is concordance among the various parameters that define ischemia. When a patient experiences typical chest pain in association with ECG and wall motion abnormalities, the diagnosis is straightforward. When results are discordant, however, certain assumptions must be made. Because wall motion is such a sensitive and specific marker of ischemia and because of the limitations in interpreting symptoms and ECG changes, the final report generally relies most heavily on the echocardiographic findings. In fact, one of the most common indications for stress echocardiography is to assess symptoms in patients who have had or would likely have an abnormal or nondiagnostic stress ECG. This would include patients with an abnormal ECG or left ventricular hypertrophy. In addition, women, because of the higher rate of a false-positive stress ECG, are often considered candidates for stress echocardiography. Figure 16.25 is an example of a patient with extensive coronary disease and a markedly
abnormal stress echocardiogram, but a negative stress ECG.

FIGURE 16.21. This is an example of inferior ischemia in a patient with no prior history of heart disease. The resting study is normal. The patient exercised to a high workload on the treadmill. Postexercise, there is an inferior wall motion abnormality that can be seen in the short-axis and two-chamber views. On coronary angiography, there was a significant lesion on the mid right coronary artery.

Video 16-21A
FIGURE 16.22. From a patient with a history of prior inferior myocardial infarction, multivessel ischemia is demonstrated with this dobutamine stress echocardiogram involving the septum, apex, inferior, and anterior walls. Video 16-22a
Video 16-22b

Video 16-22c
FIGURE 16.23. This bicycle exercise echocardiogram demonstrates multivessel ischemia involving the septum and inferior wall. [Video 16-23a](coming soon)
FIGURE 16.24. This treadmill exercise echocardiogram demonstrates extensive ischemia at a low workload. The resting study is normal while the postexercise images reveal anteroapical and lateral wall motion abnormalities. There is also an increase in left ventricular end-systolic volume with stress. This occurred at a relatively low heart rate and is consistent with coronary disease involving the left anterior descending and the left circumflex arteries.
FIGURE 16.25. This patient exhibits extensive ischemia involving the territories of the left anterior descending and right coronary arteries. Despite the markedly abnormal wall motion response, the ECG was nondiagnostic. [0]
Wall motion changes in the absence of symptoms are usually an indication of painless ischemia, a common finding. There is some evidence that ischemia in the absence of chest pain and/or ST depression is less extensive and/or severe. More problematic is the situation of ischemic ECG changes in the absence of wall motion abnormalities. When this occurs in populations with a high likelihood of a false-positive stress ECG (e.g., women), a normal stress echocardiogram is strong evidence against coronary disease. However, in subsets of patients in whom the ECG is expected to be more reliable or when the changes are accompanied by typical symptoms, the possibility of a false-negative echocardiographic result must be entertained. In one study using bicycle exercise (Ryan et al., 1993), precise concordance between the ECG and the echocardiogram occurred in approximately half of all cases, and the echocardiogram correctly classified patients in most instances of disagreement (Fig. 16.26). However, a positive ECG with a normal echocardiogram developed in 4% of cases. At catheterization, six of these patients had angiographic coronary artery disease, and the remaining seven did not. In a more recent study (Zacharias et al., 2017), 385 consecutive patients from a single center with suspected coronary disease were randomized to either a nonimaging treadmill examination or exercise echocardiography. Stress echocardiography yielded fewer inconclusive results, fewer false-positive findings, and was more cost effective than the nonimaging test. An example of a false-negative stress echocardiogram is provided in Figure 16.27. In this patient with atypical symptoms, ST-segment
depression developed during treadmill exercise, in association with chest pain, but in the absence of abnormal wall motion. At cardiac catheterization, a high-grade right coronary artery stenosis was present (Fig. 16.27). In this case, poor exercise capacity and a submaximal heart rate response may have contributed to the erroneous echocardiographic result.

Thus, the two objective indicators of ischemia during stress testing provide concordant information most of the time. When they disagree, echocardiography is more sensitive and specific and should be relied on in most instances. However, ignoring a markedly positive stress ECG, especially when accompanied by typical symptoms, is not advisable. A careful analysis of all echocardiographic images and all the available data should be undertaken.
The addition of imaging to routine stress testing has consistently led to an improvement in both sensitivity and specificity for the detection of coronary disease. Several studies have examined the accuracy of exercise echocardiography to detect coronary artery disease. Using angiography as the standard for comparison, the overall sensitivity has ranged from 71% to 94%. Similar studies have been performed using dobutamine stress echocardiography, and a comparable range of sensitivity values has been reported. The limitations of such comparisons are noteworthy. For example, differences in patient populations will explain much of this range. If a series includes a high percentage of patients with a condition, such as left ventricular hypertrophy, known to adversely affect accuracy, a lower sensitivity will be reported. Some of the variability of sensitivity values can be explained on the basis of the level of coronary artery stenosis considered significant in the different studies. The percentage of stenosis used to define a significant lesion varies from 50% to 75%, and quantitative angiographic techniques were used infrequently. It is likely that some 50% of lesions will not result in the development of ischemia during stress testing, thereby creating the potential for a false-negative result.

Another factor that affects reported accuracy is the inclusion of patients with resting wall motion abnormalities in many series. A resting wall motion abnormality is highly predictive of the presence of coronary disease, and in such patients, it is the extent rather than the presence of coronary artery disease that is important. Including patients with resting wall motion abnormalities will tend to increase the sensitivity of the stress test because patients will be correctly identified as having disease whether or not inducible ischemia occurs. In patients with normal wall motion at rest, the reported sensitivity of exercise echocardiography is somewhat lower. Another important factor is the subjective approach to wall motion interpretation, which has been used in most reported series. If very subtle abnormalities (such as lack of hyperkinesis) are interpreted as abnormal, sensitivity will tend to be higher but at the expense of lower specificity. If only the most obvious wall motion abnormalities are interpreted as positive, mild disease will be missed, and sensitivity will decrease and specificity will increase. It is not surprising then that studies that report the highest sensitivity will likewise demonstrate very modest specificity and vice versa.

In addition to the degree of coronary artery narrowing, other factors that
affect the sensitivity of the test include the presence of multivessel disease, the level of stress achieved, and the image quality. Sensitivity is consistently higher among patients with multivessel coronary disease compared with those with single-vessel disease. The location of disease may also affect accuracy. Stenoses in the left anterior descending and right coronary artery are detected more reliably than lesions in the left circumflex artery. Another potential cause of false-negative results during dobutamine stress echocardiography is the presence of left ventricular hypertrophy. Studies have shown that patients with increased wall thickness, in the setting of normal left ventricular mass (i.e., small left ventricular chamber size), have a disproportionately high frequency of false-negative results. This combination of thick walls and small left ventricular cavity size, termed concentric remodeling, is a common finding in elderly patients with hypertension. In one large series (Smart et al., 2000), this group of patients accounted for a majority of the false-negative results. The authors postulated that a blunted increase in end-systolic wall stress at peak dobutamine infusion may account for the reduced sensitivity in this subgroup. From a practical standpoint, physicians who interpret dobutamine stress echocardiographic studies should be aware of this phenomenon. Patients with concentric remodeling, especially those with hyperdynamic wall motion and/or a reduced blood pressure response during dobutamine infusion, may not manifest wall motion abnormalities in the presence of angiographic coronary artery disease. Figure 16.28 is an example of a false-negative dobutamine stress echocardiogram in a patient with mild left ventricular hypertrophy. Note the hyperdynamic response to stress. Another example of a false-negative result, in this case involving treadmill exercise, is provided in Figure 16.27. Poor exercise capacity, β-blocker therapy, and a submaximal heart rate response likely contributed in this case.
FIGURE 16.27. A false-negative exercise echocardiogram is shown (A). The left ventricle becomes hyperdynamic postexercise, although the patient had poor exercise capacity and a submaximal heart rate response. Coronary angiography revealed (B) a subtotaled right coronary artery.
FIGURE 16.28. An example of a false-negative dobutamine stress echocardiogram is presented from a patient with mild left ventricular hypertrophy. Despite the presence of coronary artery disease, a wall motion abnormality did not develop.
Addressing the issue of specificity in studies comparing stress echocardiography with angiography is limited by referral bias. When angiography is used as the gold standard, the reported specificity of exercise echocardiography ranges from 64% to 100%, although in most series, values of 80% to 90% are found. Because of referral bias, the number of patients with “normal” stress echocardiograms in such series is often quite low. An alternative approach uses the concept of normalcy rate. This approach examines the likelihood that the stress echocardiogram will be interpreted as normal in a group of patients with a very low pretest likelihood of disease. Applied to stress echocardiography, normalcy rates of 92% to 100% have been reported. As is discussed later, a normal wall motion response during
stress echocardiography, even in the presence of known coronary artery disease, confers a favorable prognosis in most cases. Among the most common causes of false-positive results is left bundle branch block. Figure 16.29 is an example of left bundle branch block in a patient undergoing treadmill stress echocardiography. Note the abnormal septal motion, both at rest and with stress. In this case, a nonischemic cardiomyopathy was present, making wall motion interpretation especially challenging. This confusing picture can sometimes be clarified by trimming the loops to avoid the first few frames of systole and by focusing on systolic thickening rather than endocardial motion.

Another form of bias in published studies that likely affects both sensitivity and specificity is test verification bias. This phenomenon results in a distortion of true accuracy because published series include selected patients with a high percentage of angiographic referrals, that is, the decision to perform angiography depends on the results of the test being studied. This leads to a misleading increase in sensitivity and decrease in specificity compared with how the test would likely perform in an unselected population. Test verification bias has been demonstrated in exercise echocardiography (Roger et al., 1997). When adjusted for, true sensitivity is lower than reported, whereas specificity is higher. Because of differences in the prevalence of coronary disease, the decrease in sensitivity is greater in women than in men. This phenomenon has been shown to plague virtually all forms of stress testing. Recognizing that it occurs and understanding its impact are key to the optimal use of stress echocardiography in clinical practice.
FIGURE 16.29. An exercise echocardiogram from a patient with LBBB is shown. The patient had resting left ventricular dysfunction, including abnormal septal motion. This was the result of nonischemic cardiomyopathy.

Video 16-29

To fully understand test performance, another factor that must be considered is the issue of pretest probability of disease. Bayes’ theorem tells us that tests such as stress echocardiography are most useful when applied to
a patient with an intermediate probability of coronary artery disease. Conversely, test results obtained in patients with either high or low pretest probability are less helpful. The reasons for this are illustrated in Figure 16.30. If we begin with the assumption that stress echocardiography has a sensitivity of 80% and a specificity of 90% (whether these are the true values is irrelevant to the discussion), consider the outcome of performing the test in 100 patients all of whom have a 10% pretest likelihood of having coronary artery disease. Among the 10 patients with disease, assuming a sensitivity of 80%, there will be 8 true-positive results and 2 false-negative results. Among the 90 patients without disease, assuming a specificity of 90%, there will be 81 true-negative and 9 false-positive results. Thus, among the 83 negative test results, there are 81 true-negatives and only 2 false-negatives. But of the 17 positive tests, 8 will be true-positives and 9 false-positives. Therefore, a negative test will be helpful and likely to be correct. However, since all patients had a very low likelihood of disease to begin with, the finding of a negative stress echocardiographic result is of limited value. A positive result, on the other hand, is just as likely to be a false-positive as a true-positive finding. Said differently, given a sensitivity of 80%, in this low-risk population, the chance of having CAD given a positive stress echocardiographic result is less than 50%.

If the test is applied to a cohort with a high pretest likelihood of disease, the opposite occurs. A negative result has a high probability of being a false-negative, potentially a very serious problem for the patient. It is only when the test is performed in intermediate-risk individuals that a reasonable balance between false-positive and false-negative results is obtained and the utility of the test is greatest.

**Role of Myocardial Perfusion Imaging**

In addition to improving endocardial border detection (which was discussed previously), contrast agents can be used to detect changes in myocardial perfusion that occur in response to stress. In theory, a perfusion defect must precede the development of a wall motion abnormality, so a method to assess myocardial perfusion should increase the sensitivity of the test to detect ischemia. Animal studies have confirmed this temporal relationship between perfusion and function. As ischemia develops, a perfusion defect will likely
develop prior to a wall motion abnormality. This is elegantly depicted in Figure 16.31, which illustrates the rate at which perfusion and wall motion become abnormal during incremental dobutamine infusion, in the presence of a flow-limiting stenosis. In addition, for a given stenosis, the spatial extent of the perfusion defect may exceed that of the wall motion abnormality, especially in the setting of single-vessel disease. For all these reasons, the ability to assess regional perfusion during stress echocardiography is desirable.

![FIGURE 16.30. The importance of pretest probability of disease on test performance is demonstrated. See text for details.](image)

After intravenous injection, the distribution of the contrast agent parallels blood flow and can be visualized (the contrast effect) as it traverses the microvasculature of the tissue, generating a time-intensity curve. Thus, perfusion can be assessed as a relative change (rest vs. stress), a regional
difference (e.g., lateral wall vs. septum), or more quantitatively based on changes in the rate of flow or blood volume. An echocardiographic test that combines wall motion assessment with the simultaneous ability to evaluate changes in perfusion in response to stress would have considerable utility.

As it applies to stress echocardiography, in most cases, the perfusion information serves as a supplement to wall motion for the diagnosis of coronary artery disease. Both exercise and pharmacologic stress modalities can be used for this purpose. In a recent prospective clinical study (Shah et al., 2015) involving 220 patients undergoing either exercise or pharmacologic stress echocardiography along with perfusion imaging, the prognostic value of change in wall motion and/or hypoperfusion were compared. Both wall motion response and a stress-induced perfusion defect added significantly to clinical risk factors and baseline left ventricular function for identifying risk of future events. The study demonstrated that assessment of myocardial perfusion and wall motion can be accomplished simultaneously in clinical practice and both contribute independently to risk stratification. In Figure 16.32, an investigational agent is used during vasodilator stress. After bubble destruction, real-time power Doppler imaging is performed. The study illustrates delayed refilling of the apical and lateral myocardium (compared with other areas) at peak stress in a patient with three-vessel coronary artery disease. Ideally, both perfusion and wall motion are assessed.
FIGURE 16.31. The temporal relationship between perfusion and wall thickening during experimental ischemia. In the presence of a flow-limiting stenosis, incremental doses of dobutamine lead to an abnormal reduction of both wall thickening (open squares) and perfusion (closed squares) compared to baseline (BL). Notice that perfusion becomes abnormal at the first stage of dobutamine while the changes in wall thickening are more subtle and do not become statistically different from baseline until the middose levels. These data suggest that perfusion should occur earlier during the course of induced ischemia. (Illustration provided courtesy of H. Leong-Poi, MD, Keenan Research Centre, St. Michael’s Hospital, University of Toronto, Ontario, Canada.)
FIGURE 16.32. This vasodilator stress echocardiogram demonstrates apical and lateral perfusion abnormalities. Apical four-chamber (4C) (top) and two-chamber (2C) (bottom) views are shown at rest (left) and peak stress (right). Using intermittent triggered imaging, myocardial perfusion at baseline is uniformly normal. With stress, there is hypoperfusion of the apical and distal anterior and lateral walls (arrows). (Courtesy of J. Jollis, MD.)
The use of these newer contrast agents for the specific purpose of perfusion imaging is not approved by the US Food and Drug Administration. Despite proof of concept and numerous clinical studies demonstrating utility, it remains unclear when or if perfusion stress echocardiography will become a routine approach to stress testing.

**Comparison With Nuclear Techniques**

An alternative approach to assessing accuracy involves the comparison of stress echocardiography and nuclear perfusion techniques. Several studies have addressed this important issue and have generally demonstrated a high degree of correlation between the different modalities. In one series (Quinones et al., 1992) in which 289 patients were subjected to simultaneous treadmill exercise echocardiography and tomographic thallium scintigraphy, the concordance between the tests was 87%. Overall accuracy is generally similar, although nuclear techniques may be more sensitive, whereas echocardiography is generally more specific when compared with angiography. Echocardiographic and nuclear imaging has also been compared using dobutamine stress, and similar levels of accuracy have been found.

A meta-analysis (Fleischmann et al., 1998) of the clinical reports that have compared echocardiographic and nuclear imaging during exercise has been reported. Analysis of the pooled data revealed almost identical sensitivity values but higher specificity for exercise echocardiography. Thus, summary
receiver operator curves revealed that echocardiography better discriminated between patients with and without disease. The relative cost-effectiveness of the different strategies to test for coronary disease has also been examined and compared (Kuntz et al., 1999; Garber et al., 1999). Because of the inherently lower costs and similar overall accuracy, stress echocardiography performs well in such analyses. The results of these models underscore the importance of relative accuracy and operator dependency. For most types of patients and levels of disease severity, exercise echocardiography is an attractive cost-effective alternative to both nonimaging treadmill testing and nuclear techniques.

It should be recognized that both tests are operator dependent and often rely on subjective interpretation of results. Thus, the relative superiority of one technique versus the other is largely a matter of expertise. The advantages of stress echocardiography include the versatility of the technique with respect to the availability of additional diagnostic information, the lower cost of the test, and the opportunity to avoid radiation exposure. In addition, stress echocardiography is more convenient for the patient because the need to return for late imaging is avoided.

**CLINICAL APPLICATION OF STRESS ECHOCARDIOGRAPHY**

The accuracy and versatility of stress echocardiography support its use in a variety of settings. It has both diagnostic and prognostic utility. Echocardiographic imaging should be seen as a supplement to routine stress testing that increases both the sensitivity and the specificity of the test for diagnosing ischemia. In addition, the opportunity to assess left ventricular function and wall motion at rest provides further value. In 2008, Appropriate Use Criteria were published for stress echocardiography. These were updated in 2011 and a total of 89 specific indications were included, covering the majority clinically encountered scenarios. These indications addressed the appropriateness of the technique based on a variety of factors, such as pretest probability of disease, TIMI score, troponin levels, the resting ECG, and ability to exercise. Considering the number of possible combinations of these clinical factors, the Appropriate Use Criteria are quite complex and difficult
to summarize. They do, however, provide some general guidelines for the practicing clinician. First, nonimaging exercise testing is a reasonable alternative for many patients, assuming the patient is capable of achieving an adequate level of exercise. Second, the decision to include imaging should be based on a combination of available factors that determine the incremental value of the imaging data. Third, stress echocardiography is an appropriate test in many different settings, including the diagnosis of CAD in intermediate-risk patients, evaluation of the patient with acute chest pain, the assessment of newly diagnosed systolic dysfunction, and the risk stratification of certain patients following myocardial infarction and/or revascularization. All individuals involved in the practice of stress echocardiography are strongly encouraged to become familiar with these criteria as they provide a logical framework for clinical decision making.

**Prognostic Value of Stress Echocardiography**

Several features of the resting echocardiogram are known to provide prognostic information. Among these, wall motion, left ventricular function and mass are well-established determinants of the risk of future cardiovascular events. The treadmill test alone (without imaging) also offers powerful prognostic information. It is not surprising then that the combination of exercise parameters and echocardiographic data should provide incremental information on risk status. Specifically, the development of a wall motion abnormality as a marker of inducible ischemia has been shown in several studies to be a powerful predictor of high-risk status. Although most studies have focused on inducible ischemia as the primary predictor, exercise duration, workload achieved, blood pressure response, and ECG changes are simultaneously available and should be incorporated into the overall determination of prognosis. The echocardiogram itself offers a range of information, including resting left ventricular function and mass. Although the presence or absence of a new wall motion abnormality is important, additional data from the stress echocardiogram should also be evaluated. These include the extent and severity of the wall motion abnormality, the volume response of the left ventricle (assessed at end-systole), the number of coronary arteries that are involved, and changes in right ventricular function. Only after all these available parameters have been
evaluated is a complete determination of risk possible.

The prognostic value of stress echocardiography has been evaluated in several settings (Table 16.7). Among patients with normal wall motion before and immediately after exercise, the likelihood of a coronary event over the ensuing 1 to 3 years is very low. McCully et al. (1998) examined 1,325 patients, of whom 35% had an intermediate (26% to 69%) and 10% had a high (≥70%) pretest probability of disease. All patients were characterized as having a normal exercise echocardiogram. Event-free survival at 1, 2, and 3 years was 99%, 98%, and 97%, respectively. Predictors of events, by multivariate analysis, were age, low exercise workload, angina, and left ventricular hypertrophy. The predictive value of wall motion assessment in the setting of a normal stress ECG has also been examined (Bouzas-Mosquera et al., 2009). In this series of 4,004 consecutive patients with a normal exercise ECG and no chest pain during treadmill testing, the development of a wall motion abnormality was relatively common (16.7%) and was highly predictive of both death and major cardiac events.

In contrast to a normal result, an abnormal exercise echocardiogram generally identifies patients at increased risk of cardiac events. Figure 16.33 is an example of extensive ischemia of the distal septum and apex, with associated apical dilation following treadmill exercise. This occurred at a low workload in a patient exertional dyspnea. On coronary angiography, a 95% proximal left anterior descending artery lesion was found. The echocardiographic findings that have been correlated with risk include a new wall motion abnormality, rest and exercise wall motion score index, and end-systolic volume response. In most series, echocardiographic evidence of ischemia was the most potent marker of high-risk status and has consistently been a better discriminator than other variables, such as exercise-induced ST-segment depression. It is also apparent that stress echocardiography provides more than simply a binary result, that is, normal or abnormal. In one large series, the postexercise wall motion score index was linearly related to event rate, suggesting that both the extent and the severity of disease determine risk. In Figure 16.34, dobutamine stress echocardiography demonstrates a limited area of inferior hypokinesis at peak stress. This occurred in a patient with single-vessel disease involving the proximal right coronary artery. A subtle lateral wall motion abnormality is shown in Figure 16.35. In this example, the patient exercised to a high workload and a peak heart rate of
148 per minute. Wall motion was normal at baseline, with lateral hypokinesis postexercise, seen in the four-chamber view. Limited ischemia occurring at a high workload generally carries a favorable prognosis and this patient was managed with aggressive medical therapy.

The prognostic value of stress echocardiography has also been compared with nuclear techniques. In most such series, echocardiography has provided similar or superior discriminatory power. Thus, high-risk status correlates best with the presence of inducible ischemia. A meta-analysis compared the negative predictive value of stress echocardiography versus nuclear perfusion imaging (Metz et al., 2007). The annualized event rate in the setting of a normal stress test was 0.45% per year for nuclear imaging and 0.54% per year for echocardiography. This was similar for men and women. In one of the largest series published to date, the prognostic value of both exercise and dobutamine echocardiography was compared in men and women (Fig. 16.36). This study showed that risk could be stratified on the basis of the extent of ischemia. It further demonstrated clearly that patients referred for exercise testing and pharmacologic testing are fundamentally different. That is, patients undergoing dobutamine stress, presumably because of their inability to exercise, have a relatively worse prognosis, regardless of the results of the test. The inability to perform an exercise test is itself an ominous prognostic sign.

| First Author, yr | Type of Stress | Inclusion/Exclusion | # of Pts | Duration of F/U | Event-Free Survival (%) | Event Rate (%)
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<tr>
<td>Heupler, 1997</td>
<td>TME</td>
<td>Women only</td>
<td>508</td>
<td>41 ± 10 mo</td>
<td>96%</td>
<td>55% 4%</td>
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<td>Krivokapich, 1999</td>
<td>Dob</td>
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<td>558</td>
<td>1 yr</td>
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<td>Yao, 2003</td>
<td>TME,</td>
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<td>1,500</td>
<td>2.7 ± 1.0</td>
<td></td>
<td>0.9%</td>
</tr>
<tr>
<td>Study</td>
<td>Type</td>
<td>Duration</td>
<td>Follow-up</td>
<td>Events 3 yrs</td>
<td>Events 2 yrs</td>
<td>Events 1 yr</td>
</tr>
<tr>
<td>------------------</td>
<td>-------</td>
<td>----------</td>
<td>-----------</td>
<td>--------------</td>
<td>--------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Krivokapich, 1993</td>
<td>TME</td>
<td>360</td>
<td>1 yr</td>
<td>3%</td>
<td>9%</td>
<td>9%</td>
</tr>
<tr>
<td>Chuah, 1998</td>
<td>Dob</td>
<td>860</td>
<td>3 yrs</td>
<td>98%</td>
<td>97%</td>
<td>96%</td>
</tr>
<tr>
<td>McCully, 1998</td>
<td>TME</td>
<td>Normal stress echos only</td>
<td>1,325</td>
<td>Median 23 mo</td>
<td>99%</td>
<td>98%</td>
</tr>
<tr>
<td>McCully, 2002</td>
<td>TME</td>
<td>Abn stress echo, but with good exer capacity</td>
<td>1,874</td>
<td>3.1 ± 1.6 yrs</td>
<td>1.6%</td>
<td>w/nl LVESV response</td>
</tr>
<tr>
<td>Smart, 1999</td>
<td>Dob</td>
<td>Rest EF &lt;40%</td>
<td>350</td>
<td>&gt;18 mo</td>
<td></td>
<td>Hard</td>
</tr>
<tr>
<td>Poldermans, 1999</td>
<td>Dob</td>
<td></td>
<td>1,734</td>
<td>1 yr</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortigiani, 1998</td>
<td>Dip, Dob</td>
<td>Women with chest pain</td>
<td>456</td>
<td>32 ± 19 mo</td>
<td>99.2% (hard events, 3 yrs)</td>
<td>69.5% (hard events, 3 yrs)</td>
</tr>
<tr>
<td>Sicari, 2003</td>
<td>Dip, Dob</td>
<td>Multicenter</td>
<td>7,333</td>
<td>2.6 yrs</td>
<td>92%</td>
<td>71%</td>
</tr>
<tr>
<td>Bholasigh, 2003</td>
<td>Dob</td>
<td>CPU pts, negative troponin T</td>
<td>377</td>
<td>6 mo</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Author</td>
<td>Modality</td>
<td>Follow-up</td>
<td>Study Population</td>
<td>Mean Age</td>
<td>3-yr Survival</td>
<td>Ischemia Only</td>
</tr>
<tr>
<td>---------------</td>
<td>----------</td>
<td>----------------</td>
<td>------------------</td>
<td>----------</td>
<td>---------------</td>
<td>---------------</td>
</tr>
<tr>
<td>Steinberg, 1997</td>
<td>Dob</td>
<td>Long-term (&gt;5 yrs) follow-up</td>
<td>120</td>
<td>5 yrs</td>
<td>5% (hard events, yrs)</td>
<td></td>
</tr>
<tr>
<td>Marwick, 1997</td>
<td>TME</td>
<td>463</td>
<td>44 ± 11 mo</td>
<td>90% (all events, 4.5 yrs)</td>
<td>61% ischemia only 29% ischemia + scar</td>
<td></td>
</tr>
<tr>
<td>Biagini, 2005</td>
<td>Dob</td>
<td>7-yr follow-up</td>
<td>2,276 females, 1,105 males</td>
<td>7 ± 3.4 yrs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chaowalit, 2006</td>
<td>Dob</td>
<td>Diabetics</td>
<td>2,349</td>
<td>5.4 ± 2.2 yrs</td>
<td>81% 3-yr survival</td>
<td>70% 3-yr survival</td>
</tr>
</tbody>
</table>

Dip, dipyridamole; Dob, dobutamine; CPU, chest pain unit; CHF, congestive heart failure; F/U, follow-up; LVESV, left ventricular end-systolic volume; MI, myocardial infarction; Pts, patients; TME, treadmill; WMA, wall motion abnormality; WMSI, wall motion score index.

**FIGURE 16.33.** Extensive ischemia of the distal septum and apex, with apical dilation is seen on this treadmill exercise echocardiogram. This occurred at a low workload.
FIGURE 16.34. A subtle exercise-induced wall motion abnormality involving the basal inferior wall is seen on this dobutamine stress test. Although this is a common area for a false-positive result, in this patient single-vessel disease of the right coronary artery was present. [Video 16-34a](coming soon)
FIGURE 16.35. Limited ischemia of the lateral wall is demonstrated on this treadmill exercise echocardiogram. This is best seen in the four-chamber view. In this format, a resting image is presented with three postexercise loops.
Whether or not functional testing with either echocardiographic or nuclear techniques is the optimal approach to risk stratification in patients with stable symptoms has recently been questioned. The PROMISE trial (Prospective Multicenter Imaging Study for the Evaluation of Chest Pain) compared functional testing with CT angiography in over 9,000 patients with stable chest pain and intermediate pretest likelihood of disease. Patients who underwent CT angiography were more likely to have abnormal findings and experience subsequent events, compared to patients undergoing functional testing such as stress echocardiography. The superiority of CT angiography was attributed to its ability to detect nonobstructive CAD which would not be detected with a stress imaging test, but which may confer an increased risk of
future events. However, when clinical risk (the Framingham risk score) was added to the results of functional testing, the superiority of anatomic testing was eliminated. This important trial underscores the importance of assessing global risk, which includes clinical factors as well as test results.

It seems likely that functional testing with either echocardiography or nuclear scintigraphy will continue to be used for risk assessment in many patients. Other echocardiographic findings, including left ventricular volume and ejection fraction, also contribute prognostic information, as does treadmill variables such as workload, blood pressure, ECG, and symptoms. In multivariate models, however, nonechocardiographic parameters such as age, symptoms, and diabetes frequently contribute independent prognostic data.

**Stress Echocardiography in Patients With Acute Chest Pain**

In the United States each year, acute chest pain accounts for over six million visits to the Emergency Department. A variety of testing options for these patients are now available and Appropriate Use Criteria have recently been developed specifically for cardiovascular testing in the Emergency Department. This is an area where CT angiography has been shown to be quite useful. Several studies have demonstrated their ability to accurately risk stratify patients in an efficient and cost-effective manner. Functional testing, with either nuclear or echocardiographic methods, is also widely utilized. In the recent guidelines, stress echocardiography is considered an appropriate test for many patients with suspected acute coronary syndrome, such as those with negative biomarkers and absence of ST-segment elevation on ECG. As in other settings, a negative result on stress echocardiography confers a favorable prognosis with a low probability of an event. Figure 16.37 demonstrates right ventricular ischemia in a patient who had presented to the Emergency Department with anginal chest pain but a normal ECG. On treadmill echocardiography, he became profoundly dyspneic at a low workload. Left ventricular wall motion was unchanged, but the right ventricular free wall became akinetic postexercise. Coronary angiography revealed a proximal right coronary artery lesion.

**Stress Echocardiography After Myocardial Infarction**
Stress testing after myocardial infarction is used both to identify high- and low-risk subsets and to predict the location and extent of coronary disease. When applied to this population, it must be recognized that most patients will have a resting wall motion abnormality. The goal of the test is to identify ischemia at a distance and, in doing so, to predict both the likelihood of multivessel disease and the presence of inducible ischemia. In this setting, a normal response would be the development of hyperdynamic wall motion in all regions remote from the infarct. Therefore, the most important positive finding is the detection of a new wall motion abnormality remote from the site of previous infarction. Appropriate Use Criteria for the role of stress echocardiography following an acute coronary syndrome are provided in Table 16.8.

**FIGURE 16.36.** Risk-adjusted 5-year survival in women (top) and men (bottom) who underwent exercise (left) and dobutamine (right) stress echocardiography. Survival curves are provided for those patients with no evidence of ischemia, single-vessel ischemia, and multivessel ischemia. Within each group, the
presence and extent of ischemia stratified the patients. Note that survival is worse for those patients who underwent dobutamine versus exercise testing and that outcome was poorest for men undergoing dobutamine stress echocardiography in whom multivessel ischemia developed. (From Shaw LJ, Vasey C, Sawada S, Rimmerman C, Marwick TH. Impact of gender on risk stratification by exercise and dobutamine stress echocardiography: long-term mortality in 4234 women and 6898 men. *Eur Heart J* 2005;26:447–456, with permission.)

Exercise echocardiography can be used to risk stratify patients after an acute coronary event (Figs. 16.38 to 16.42). The ability to identify multivessel disease and the extent of the jeopardized myocardium, combined with a functional assessment of exercise capacity in a patient recovering from myocardial infarction, accounts for the prognostic value of the test. In Figure 16.38, from a patient who underwent stent placement following anterior myocardial infarction, very limited apical ischemia is demonstrated, best seen in the apical long-axis view. Dobutamine echocardiography can also be used for this purpose (Fig. 16.39). In this example, a patient with a remote history of inferior myocardial infarction undergoes stress testing. A shallow inferobasal aneurysm is present, but the remaining areas become hyperdynamic with dobutamine, conferring low-risk status. Figure 16.40 illustrates worsening of an inferoposterior wall motion abnormality in response to stress. Although no new areas of abnormal wall motion develop, worsening of a resting abnormality may indicate peri-infarct ischemia. Another example of worsening wall motion during exercise is provided in Figure 16.41. In this example, apical hypokinesis becomes akinesis postexercise and some dilation of the apex occurs.

Evidence of ischemia not only predicts high-risk status but also correlates with the likelihood of multivessel coronary disease. In one series (Carlos et al., 1997), dobutamine echocardiographic evidence of multivessel involvement was a better predictor of future events than angiographic evidence of multivessel disease. Thus, absence of evidence of inducible ischemia by stress echocardiography identifies patients recovering from infarction with a favorable prognosis in whom further testing may be unnecessary. Inducible ischemia, on the other hand, is a powerful indicator of high risk and suggests the need for further testing, specifically angiography. In Figure 16.42, from a patient with remote inferior myocardial infarction and stent placement, a new inducible distal anterior wall motion is demonstrated following treadmill exercise. Three-vessel coronary artery disease was
FIGURE 16.37. An unusual example of ischemia during exercise affecting the right ventricle is provided. This patient had presented to the Emergency Department with exertional dyspnea. Left ventricular wall motion is preserved and coronary angiography demonstrated a proximal right coronary artery lesion.
Stress Echocardiography After Revascularization

Stress testing after revascularization is used to evaluate the initial success of the procedure, to look for recurrence of disease, and to assess symptoms in patients with known coronary disease. The limitations of symptoms and the stress ECG in this setting underscore the importance of imaging. Exercise echocardiography has been used before and after angioplasty to localize disease and to document objective improvement after the procedure. Appropriate Use Criteria for the role of stress echocardiography following revascularization are provided in Table 16.9.

<table>
<thead>
<tr>
<th>Table 16.8</th>
<th>APPROPRIATE USE CRITERIA FOR STRESS ECHOCARDIOGRAPHY FOLLOWING ACUTE CORONARY SYNDROME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indication</td>
<td>Score (1–9)</td>
</tr>
<tr>
<td>STEMI</td>
<td></td>
</tr>
<tr>
<td>163</td>
<td>PCI with complete revascularization; no recurrent Sx</td>
</tr>
<tr>
<td>164</td>
<td>Stable and no Sx post-MI; no prior angiography</td>
</tr>
<tr>
<td>165</td>
<td>Hemodynamically unstable; shock; or mechanical complications</td>
</tr>
<tr>
<td>UA/NSTEMI</td>
<td></td>
</tr>
<tr>
<td>166</td>
<td>Stable and no Sx post-MI; no prior angiography</td>
</tr>
<tr>
<td>ACS—asymptomatic postrevascularization</td>
<td></td>
</tr>
</tbody>
</table>
Prior to discharge in a pt who has been adequately revascularized

Cardiac rehabilitation

Prior to initiation of cardiac rehabilitation (as a stand-alone indication)


FIGURE 16.38. This is an example of very limited ischemia in a patient with prior myocardial infarction. The patient had suffered an anterior myocardial infarction which was treated with angioplasty and stent placement. One month later, a treadmill exercise echocardiogram was performed. The resting study was normal, with no evidence of the prior infarction. Postexercise, a small area of apical dyskinesis develops but is visualized only on the apical long-axis view.
Video 16-38a

Video 16-38b

coming soon

coming soon
Mertes and colleagues (1993) used bicycle stress echocardiography to evaluate patients 6 months after a percutaneous coronary intervention. They reported a sensitivity of 83% and a specificity of 85% for the detection of significant coronary stenoses. Similar results have been reported using stress echocardiography after coronary artery bypass surgery. In this setting, stress echocardiography has been successfully used to detect the presence of stenotic grafts, nonrevascularized coronary arteries, and diseased native vessels distal to the surgical anastomosis. A study by Elhendy and colleagues (2006) suggests that the addition of contrast to dobutamine stress may increase the sensitivity of the test for detection of occluded vein grafts. In this series of 64 patients, contrast echocardiography had a 90% per-patient sensitivity and a 74% per-region sensitivity for detecting diseased grafts.

A practical application in this setting is to provide objective evidence of ischemia in a subset of patients with a high likelihood of atypical symptoms. Figure 16.43 is an example of stress echocardiography before and after revascularization. In this example, a patient undergoes dobutamine stress echocardiography and multivessel ischemia is detected at a low heart rate. Four months later, after surgical revascularization, another dobutamine study is performed. A much higher heart rate and improved wall motion response are demonstrated.
FIGURE 16.39. This dobutamine stress echocardiogram was performed in a patient with a previous inferior myocardial infarction. An inferobasal aneurysm is demonstrated in the two-chamber view. With dobutamine, there is a normal hyperdynamic response in all other areas. No evidence of ischemia was detected.
FIGURE 16.40. This is an example of worsening of a pre-existing wall motion abnormality in a patient with prior inferior myocardial infarction. The resting wall motion abnormality involves the inferior, posterior, and lateral walls. With exercise, there is worsening of wall motion in the infarct zone, best appreciated in the apical long-axis view. [Video 16-40a](#)
coming soon

Video 16-40b

coming soon

Video 16-40c
FIGURE 16.41. From a patient with a remote history of an anterior myocardial infarction, this treadmill exercise echocardiogram demonstrates apical hypokinesis at rest with more severe and extensive akinesis postexercise. Stress-induced apical dilation is best seen in the two-chamber view (bottom panels).
FIGURE 16.42. This patient had a history of inferior myocardial infarction and PCI of the right coronary artery. The exercise echocardiogram reveals normal inferior wall motion, but the development of mild anterior hypokinesis poststress. Three-vessel coronary disease was present. Video 16-42a
Table 16.9  APPROPRIATE USE CRITERIA FOR STRESS ECHOCARDIOGRAPHY FOLLOWING REVASCULARIZATION

<table>
<thead>
<tr>
<th>Indication</th>
<th>Score (1–9)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptomatic postrevascularization</strong></td>
<td></td>
</tr>
<tr>
<td>169  Ischemic equivalent</td>
<td>A (8)</td>
</tr>
<tr>
<td><strong>Asymptomatic postrevascularization</strong></td>
<td></td>
</tr>
<tr>
<td>170  Incomplete revascularization; additional revascularization feasible</td>
<td>A (7)</td>
</tr>
<tr>
<td>171  &lt;5 yrs after CABG</td>
<td>I (2)</td>
</tr>
<tr>
<td>172  ≥5 yrs after CABG</td>
<td>U (6)</td>
</tr>
<tr>
<td>173  &lt;2 yrs after PCI</td>
<td>I (2)</td>
</tr>
<tr>
<td>174  ≥2 yrs after PCI</td>
<td>U (5)</td>
</tr>
<tr>
<td><strong>Cardiac rehabilitation</strong></td>
<td></td>
</tr>
<tr>
<td>175  Prior to initiation of cardiac rehabilitation (as a stand-alone indication)</td>
<td>I (3)</td>
</tr>
</tbody>
</table>


**Preoperative Risk Assessment**

To assess preoperative risk prior to noncardiac surgery, stress echocardiography can be used to demonstrate ischemia and assess risk. Appropriate Use Criteria have included specific guidelines for the use of
stress echocardiography in the setting of preoperative risk evaluation (Table 16.10). In the past decade a lesser role for preoperative stress testing has been advocated. Most patients who are clinically stable with preserved functional capacity, undergoing low- or intermediate-risk noncardiac surgery do not benefit from further risk assessment. In fact, in the 2011 revision of the Appropriate Use Criteria for echocardiography, stress testing was deemed appropriate only for patients undergoing vascular surgery, with more than one clinical risk factor and poor (or unknown) functional capacity. For other scenarios involving intermediate-risk surgery, the appropriateness of stress echocardiography was considered uncertain.

Much of the data on the utility of stress echocardiography for preoperative assessment were developed in relatively high-risk patients with vascular disease. In this high-risk subset, dobutamine stress echocardiography has consistently demonstrated value and the presence or absence of an inducible wall motion abnormality has been the most potent determinant of relative risk. The absence of an inducible wall motion abnormality confers a very favorable prognosis, with a negative predictive value of 93% to 100%. In this setting, predictive value refers to the test’s ability to identify patients who subsequently experience perioperative events. In part, this very high-negative predictive value is confounded by the inclusion of patients with a low pretest likelihood of coronary disease in whom the added value of stress testing is questionable.

The presence of an induced wall motion abnormality substantially increases the relative risk to the individual patient. The positive predictive value of an inducible wall motion abnormality has ranged from 7% to 33% when hard events are used as the end point. An intermediate-risk subgroup includes those patients with a resting wall motion abnormality but no evidence of ischemia. A resting wall motion abnormality, most likely indicating previous myocardial infarction, has also been associated with a much lower risk compared with those with induced ischemia. Most of these patients can safely undergo elective surgery, with an overall perioperative risk similar to that of the “normal” group.
FIGURE 16.43. Two dobutamine stress echocardiograms are provided from a patient with diabetes and peripheral vascular disease. On the first study (left), extensive wall motion abnormalities are induced during dobutamine infusion, consistent with multivessel ischemia (videos A–C). The patient then underwent surgical revascularization. Four months later, a second dobutamine stress echocardiogram was performed (right, videos D–F). Note the striking improvement in the left ventricular response to stress. A higher heart rate is achieved, and only a moderate-sized apical wall motion abnormality is apparent.

Video 16-43a
Compared to nuclear stress testing, stress echocardiography appears to provide similar or even superior preoperative risk assessment. In a meta-analysis involving 68 studies and over 10,000 patients, thallium imaging and stress echocardiography were compared for risk stratification prior to elective noncardiac surgery (Beattie et al., 2006). For both tests, a moderate or large abnormality was predictive of perioperative events. However, stress echocardiography had greater negative predictive power than nuclear imaging.
Stress Echocardiography in Women

It is now well recognized that important differences exist between men and women in both the evaluation and treatment of ischemic heart disease. This topic was reviewed in detail in a 2014 publication by the American Heart Association (Mieres et al., 2014). This important publication addressed the role of noninvasive testing for women with suspected ischemic heart disease. It emphasized the utility of nonimaging exercise testing for symptomatic women with good exercise capacity and a normal resting ECG. For those with limited exercise ability, baseline ST-segment abnormalities, or a prior indeterminate stress ECG, stress echocardiography was considered a Class I recommendation (level of evidence B). Although diagnostic accuracy is similar to other stress modalities, the document pointed out the advantage of stress echocardiography in avoiding radiation exposure, particularly among younger women.

<table>
<thead>
<tr>
<th>Table 16.10</th>
<th>APPROPRIATE USE CRITERIA FOR STRESS ECHOCARDIOGRAPHY FOR PREOPERATIVE EVALUATION PRIOR TO NONCARDIAC SURGERY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indication</td>
<td>Score (1–9)</td>
</tr>
<tr>
<td><strong>Low-risk surgery</strong></td>
<td></td>
</tr>
<tr>
<td>154 Preoperative evaluation for risk assessment</td>
<td>I (1)</td>
</tr>
<tr>
<td><strong>Intermediate-risk surgery</strong></td>
<td></td>
</tr>
<tr>
<td>155 Moderate to good functional capacity (≥ 4 METS)</td>
<td>I (3)</td>
</tr>
<tr>
<td>156 No clinical risk factors</td>
<td>I (2)</td>
</tr>
<tr>
<td>157 ≥1 clinical risk factors; poor or unknown functional capacity</td>
<td>U (6)</td>
</tr>
<tr>
<td>158 No Sx &lt;1 yr post normal catheterization, noninvasive test, or prior revascularization</td>
<td>I (1)</td>
</tr>
<tr>
<td><strong>Vascular surgery</strong></td>
<td></td>
</tr>
<tr>
<td>159 Moderate to good functional capacity (≥ 4 METS)</td>
<td>I (3)</td>
</tr>
<tr>
<td>160 No clinical risk factors</td>
<td>I (2)</td>
</tr>
<tr>
<td>161 ≥1 clinical risk factors; poor or unknown functional capacity</td>
<td>A (7)</td>
</tr>
<tr>
<td>162 No Sx &lt;1 yr post normal catheterization, noninvasive test, or prior revascularization</td>
<td>I (2)</td>
</tr>
</tbody>
</table>
There is some evidence that stress testing is applied less frequently in women than in men. The relatively lower prevalence of disease and the higher rates of a false-positive ECG response complicate stress testing in women. Several series have examined the role of both exercise and dobutamine stress echocardiography in this large patient subset. The majority of these studies have demonstrated that wall motion analysis increases both the sensitivity and the specificity of the test. Most series report a sensitivity of 80% to 90% and a specificity of 85% to 90%. In addition to its accuracy, studies have shown stress echocardiography to be a cost-effective method to evaluate chest pain in women. Other investigators have explored the possibility that stress echocardiography is less accurate in women than in men. It now appears clear that no significant gender difference exists, with respect to both the diagnostic and the prognostic value of the test. In one large multicenter registry, the prognostic value of stress echocardiography was compared in 4,234 women and 6,898 men (Shaw et al., 2005). Echocardiography was similarly predictive of events in men and women. Risk-adjusted 5-year survival rates were 99.4%, 97.6%, and 95% for women who underwent exercise testing, for 0, single-vessel, and multivessel ischemia, respectively. For women who underwent dobutamine testing, 5-year survival was 95%, 89%, and 86% for those with 0, single-vessel, and multivessel ischemia, respectively (see Fig. 16.36). Figure 16.44 demonstrates the development of ischemia of the distal septum, anterior wall, and apex from a woman with a previously normal nonimaging treadmill test.

**Assessment of Myocardial Viability**

The capacity of dysfunctional myocardium to recover spontaneously or improve after revascularization has been recognized for many years. The term viable is commonly used to refer to myocardium that has the potential for functional recovery, that is, either stunned or hibernating. Distinguishing viable from nonviable myocardium in patients with resting left ventricular dysfunction has been extensively examined using a variety of imaging
techniques including echocardiography. To begin the analysis, the resting echocardiogram has some utility for predicting viability; the more severe the wall motion abnormality is at rest, the less likely it is to be viable. Dyskinetic regions, for example, are less likely than hypokinetic segments to recover. Thin, scarred segments are also likely to be nonviable. However, the resting echocardiogram is neither sensitive nor specific for this purpose. The use of dobutamine echocardiography is based on the observation that viable myocardium will augment in response to β-adrenergic stimulation, whereas nonviable myocardium will not. In practice, dobutamine is infused at incremental rates while wall motion and endocardial thickening are carefully monitored. The biphasic response, augmentation at low dose followed by deterioration at higher doses, is most predictive of the capacity for functional recovery after revascularization. Figure 16.45 is an example of viability and ischemia in a patient with reduced left ventricular function. Sustained improvement and “no change” are patterns that correlate with nonviability, that is, lack of improvement after revascularization.

FIGURE 16.44. Treadmill exercise echocardiography in this female patient demonstrates marked ischemia of the distal septum, apex, and distal anterior
wall. She had recently had a normal nonimaging exercise study. Video 16-44b

coming soon

Video 16-44a

coming soon

Video 16-44b

coming soon
FIGURE 16.45. This dobutamine stress echocardiogram demonstrates global hypokinesis, especially involving the anterior and lateral walls at baseline. At low
dose (upper right), there is some augmentation of wall motion. With increasing doses of dobutamine, extensive ischemia involving the septum, apex, lateral, and inferoposterior walls is demonstrated. It is important to note that the dobutamine infusion was continued longer than necessary. The study should have been terminated following the 30 μg/kg/min stage (lower left), before atropine was administered. On coronary angiography, there was extensive and severe three-vessel coronary disease. [Video 16-45a]

Video 16-45a

coming soon

[Video 16-45b]

Video 16-45b
With an improvement in resting left ventricular function after revascularization as the end point, dobutamine echocardiography has been tested in two clinical scenarios. Early studies focused on patients soon after myocardial infarction, in whom stunning may have been the predominant pathologic process. Later, the test was extended to include patients with chronic coronary disease and ischemic cardiomyopathy. In most series, sensitivity (for predicting functional recovery) has ranged from 80% to 85% with slightly higher specificity (85% to 90%). The amount of myocardium identified as viable correlates fairly well with the degree of improvement in global function after revascularization and with long-term outcome. When compared with nuclear techniques, dobutamine echocardiography provides
generally concordant results. However, nuclear techniques will identify significantly more segments (and patients) as viable. In most series, sensitivity favors nuclear methods, whereas dobutamine echocardiography is consistently more specific. Thus, all the methods appear to provide a similar positive predictive value. That is, evidence of viability by any of the techniques is predictive of the potential for functional recovery after revascularization. However, the negative predictive value varies widely among the different modalities, and in many series, dobutamine echocardiography is favored.

The prognostic value of this application has also been examined. Although these studies are observational and randomized trials are not yet available, they demonstrate the important link between evidence of viability and management. The presence of viability identifies patients in whom revascularization is associated with a significant survival advantage compared with medical management. Absence of viability is associated with no significant outcome advantage, whether medical or surgical therapy is implemented. These results were confirmed in a meta-analysis that included more than 3,000 patients studied with either echocardiographic or nuclear methods (Allman et al., 2002). Among patients with viability, surgical revascularization improved prognosis compared with medical therapy. In patients without viability, outcome was similar regardless of treatment strategy (Fig. 16.46). This is in contrast to the results of a multicenter registry in which medically treated patients with viability had a better prognosis than patients without viability (Picano et al., 1998). However, this study focused on patients early after acute myocardial infarction, with moderate to severe left ventricular dysfunction, all of whom were treated medically. In this subset, sustained improvement conferred a survival advantage, whereas ischemia identified a high-risk cohort.

Despite a multitude of clinical studies, viability assessment remains a complex and controversial area. The STICH Randomized Trial (Surgical Treatment for Ischemic Heart Failure) compared the outcome of patients with ischemic cardiomyopathy treated medically versus surgically. A nonrandomized subset of STICH attempted to assess the role of viability testing using either SPECT or dobutamine echocardiography. Over 80% of the patients included in the substudy had evidence of significant viability, distributed between the two treatment arms (optimal medical therapy vs.
bypass surgery). Although the presence of viability seemed to confer a survival benefit, the overall results, once adjusted for baseline variables, failed to demonstrate a clear role for viability testing. A link between the presence or absence of viable myocardium and either treatment or outcome could not be proven. Although the proper interpretation of the STICH substudy results continues to be a source of debate, viability assessment in select patients is reasonable. For example, in a high-risk symptomatic patient with coronary anatomy amenable for revascularization, the presence and extent of viable myocardium may aid substantially in management.

**STRESS ECHOCARDIOGRAPHY IN NONISCHEMIC HEART DISEASE**

Stress echocardiography has an expanding role in the evaluation of patients with other forms of heart disease (Table 16.11). A detailed review of this topic was recently published jointly by the European Association of Cardiovascular Imaging and the American Society of Echocardiography (Lancellotti et al., 2017). During routine stress testing, in patients with known or suspected coronary disease, important valvular abnormalities are occasionally identified with Doppler. In one series involving 1,272 consecutive patients (Gaur et al., 2003), significant mitral regurgitation was detected in 5% of patients, aortic regurgitation in 13%, and aortic or mitral stenosis in approximately 1% each. Even in patients who had a previous Doppler study as part of a routine echocardiogram, an important new Doppler finding was recorded in 9%. This suggests that a limited Doppler study should be a part of most stress echocardiographic examinations.
Exercise echocardiography can also be used specifically for the assessment of valvular heart disease. Ideally, supine bicycle exercise should be used for this application. This allows imaging to be performed during (rather than after) exercise and also facilitates acquisition of multiple parameters, for example, valve gradients and pulmonary artery pressure. Several forms of nonsevere valve disease may benefit from stress echocardiographic evaluation. For example, in patients with mitral stenosis of “borderline” severity, the response to exercise can be helpful, particularly to correlate symptoms with objective evidence of disease. Some patients with relatively mild disease will have a significant increase in mean gradient during exercise. An exercise or postexercise mean gradient of >15 mm Hg is considered evidence of severe stenosis. This may be accompanied by an inappropriate increase in systolic pulmonary artery pressure (>60 mm Hg) that also can be documented with the Doppler technique. Stress echocardiography has also been used in patients with mitral stenosis to select candidates for balloon mitral valvuloplasty and to document the improved hemodynamics after the procedure (Fig. 16.47).

<table>
<thead>
<tr>
<th>Table 16.11</th>
<th>ROLE OF STRESS ECHOCARDIOGRAPHY IN OTHER FORMS OF CARDIOVASCULAR DISEASE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indication</td>
<td>Examples of Use</td>
</tr>
<tr>
<td>Valvular heart disease</td>
<td></td>
</tr>
</tbody>
</table>
| Aortic valve | Mild/mod AR with Sx  
Severe AS with no Sx  
Low-flow low-gradient AS |
|--------------|---------------------------------------------------------------|
| Mitral valve | Mild/mod MS with Sx  
Severe MR with no Sx |
| Prosthetic valves | AVR with suspected patient–prosthesis mismatch |
| Hypertrophic cardiomyopathy | Assessment of MR and/or LVOT gradient |
| Pulmonary hypertension | Exertional Sx with mild pulmonary HTN at rest |
| Diastolic dysfunction | Dyspnea on exertion, with normal systolic function |
| Congenital heart disease | Aortic coarctation gradient |

Detecting dynamic mitral regurgitation using exercise Doppler techniques is also possible. Unexpected worsening of mitral regurgitation severity can be recorded during stress with color Doppler imaging. Exercise-induced worsening of mitral regurgitation has been reported in the absence of ischemia or left ventricular dilation. In patients with valvular aortic stenosis, Doppler can be used to quantify the change in gradient during exercise in asymptomatic patients. Again, the test may be useful in clinical decision making in patients with exertional symptoms whose stenosis appears borderline on the resting study.

Stress echocardiography, usually using dobutamine, has particular value in patients with left ventricular dysfunction and a moderate aortic valve gradient. In such cases, the resting study often fails to differentiate between moderate and severe aortic stenosis based on gradient alone. Dobutamine, by increasing transvalvular flow, can be used to distinguish moderate stenosis in the setting of poor left ventricular function from critical aortic stenosis. This topic is covered more fully in Chapter 10.

Exercise echocardiography has also been used to study prosthetic valve function. Pressure gradients across normally functioning prostheses often increase substantially during exercise. Stress echocardiographic techniques have proven valuable in understanding and quantifying the hemodynamic differences among the various types of prosthetic valves. Exercise hemodynamics may also provide evidence of patient–prosthesis mismatch. Other applications of stress echocardiography include the detection of exercise-induced changes in pulmonary artery pressure in patients with chronic lung disease, the evaluation of the dynamic outflow tract gradient in
patients with hypertrophic obstructive cardiomyopathy, and the assessment of doxorubicin cardiomyopathy.

**Diastolic Stress Echocardiography**

Diastolic stress refers to the use of exercise Doppler imaging to detect reduced diastolic function reserve and associated increases in left ventricular filling pressure. Initial studies in this area focused on the potential for detecting diastolic abnormalities that might represent an early marker of ischemia. More recently, and more useful, has been the application of diastolic stress testing to patients with unexplained dyspnea, as evidence of heart failure with preserved ejection fraction (HFpEF).
Mean gradient 8 mm Hg
HR 64/min

HR 68/min
RVSP 30 mm Hg

Mean gradient 18 mm Hg
HR 95/min

HR 105/min
Mean gradient 24 mm Hg

HR 95/min
RVSP 45-50 mmHg
Diastolic stress testing is optimally performed with supine bicycle exercise, which allows data to be acquired at peak exercise. Alternatively, treadmill exercise can be used with imaging in the immediate postexercise period. Because fusion of the mitral E and A waves is a problem, obtaining Doppler data at heart rates of 100 to 110 per minute is recommended, so postexercise treadmill imaging may be a viable option. The test can either be performed as part of a routine stress echocardiogram, combined with wall motion assessment, or as a stand-alone test.

The most important parameters to record are rest and exercise mitral inflow, septal and lateral annular velocities, and the peak velocity of the tricuspid regurgitation jet. In normal subjects, E/e′ is <10 at rest and remains so during exercise. A tricuspid regurgitation velocity <2.8 cm/s generally excludes the possibility of significant pulmonary hypertension. However, a modest increase in pulmonary artery pressure may occur during exercise in normal subjects (especially athletes) so a cutoff during exercise of 3.1 m/s has been advocated in some studies. Abnormal diastolic function during exercise is indicated by an E/e′ >14 and/or a tricuspid regurgitation velocity of >3.1 m/s. Although preliminary studies in this area are promising, challenges remain. Additional work in this area is needed, but the potential of increased application of diastolic indicators to stress echocardiography is high.

Suggested Readings

Accuracy


**TECHNIQUE AND METHODOLOGY**


**Prognosis and Risk Stratification**


McCully RB, Roger VL, Mahoney DW, et al. Outcome after abnormal exercise echocardiography for


**Preoperative Risk Assessment**


**Viability**


**Miscellaneous**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
CLINICAL AND ECHOCARDIOGRAPHIC OVERVIEW

Cardiomyopathy represents a diverse group of diseases intrinsic to the myocardium. By strict definition, they are a primary disorder of the heart muscle and are not related to valve disease, hypertension, or coronary artery disease. From a practical standpoint, severe dysfunction due to diffuse coronary disease and chronic ischemia is considered a form of cardiomyopathy (ischemic cardiomyopathy). Traditionally, cardiomyopathies are divided into dilated (or congestive) and nondilated or restrictive forms. Some cardiomyopathies may present as either a dilated or restrictive form. An additional subset includes true hypertrophic cardiomyopathy, which can either be nonobstructive or obstructive. This chapter deals with dilated cardiomyopathy. Restrictive, hypertrophic, and other cardiomyopathies will be addressed in Chapter 18.

Imaging modalities other than echocardiography can play a valuable role in the diagnosis and management of patients with dilated myopathies. Cardiac magnetic resonance imaging (CMR) provides the most accurate determination of left ventricular volumes and left ventricular wall thickness. While left ventricular volumes from CMR and echocardiography generally correlate well in high-quality studies, the absolute left ventricular volumes obtained with echocardiography is frequently systematically lower than those with CMR. This is largely due to inadvertent inclusion of trabeculae and papillary muscles when tracing the endocardial border on an echocardiogram which are more effectively excluded on CMR. CMR with gadolinium enhancement can provide valuable clues as to the presence of myocardial fibrosis which, depending on its pattern and location, may provide diagnosis
of a specific form of cardiomyopathy. CMR with delayed gadolinium enhancement also provides an accurate method for the diagnosis of acute myocarditis when used in the appropriate clinical situation. Cardiac CT similarly can provide high-resolution ventricular volumes but generally provides less incremental information over echocardiography than is provided by CMR. Finally, positron emission tomography with F18 deoxyglucose provides evidence of metabolic activity and, when combined with an evaluation of myocardial perfusion with rubidium, provides valuable information with respect to the diagnosis of certain inflammatory conditions such as cardiac sarcoidosis.

**DILATED CARDIOMYOPATHY**

There are multiple etiologies for dilated cardiomyopathy (Table 17.1). Clinically, all types of cardiomyopathy share a constellation of symptoms that can be present to varying degrees, including congestive heart failure, low-output state, fatigue, dyspnea, arrhythmias, and sudden cardiac death. Echocardiography serves as a definitive tool for establishing the presence and severity of cardiomyopathy. It may provide information regarding the specific etiology and can be used to accurately track the secondary and physiologic abnormalities associated with the cardiomyopathy. The American College of Cardiology/American Heart Association guidelines for management of congestive heart failure consider echocardiography a class I diagnostic test, implying that it is generally indicated and useful in all patients with congestive heart failure and suspected cardiomyopathy. Its use is considered appropriate in a broad range of situations in patients with known or suspected cardiomyopathy (Table 17.2). Echocardiographic imaging can provide valuable prognostic information and serves as a guide to the success of therapy.

<table>
<thead>
<tr>
<th>Table 17.1 CLASSIFICATION OF CARDIOMYOPATHY AND DISEASES RESULTING IN ACUTE OR CHRONIC LEFT VENTRICULAR DYSFUNCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dilated cardiomyopathy</td>
</tr>
<tr>
<td>Indication</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>Idiopathic cardiomyopathy</td>
</tr>
<tr>
<td>Familial cardiomyopathy</td>
</tr>
<tr>
<td>Noncompacted myocardium</td>
</tr>
<tr>
<td>Peripartum cardiomyopathy</td>
</tr>
<tr>
<td>Hemochromatosis</td>
</tr>
<tr>
<td>Infectious</td>
</tr>
<tr>
<td>Postviral myocarditis</td>
</tr>
<tr>
<td>Human immunodeficiency virus related</td>
</tr>
<tr>
<td><em>Legionella</em> infection</td>
</tr>
<tr>
<td>Sepsis (gram negative)</td>
</tr>
<tr>
<td>Toxic cardiomyopathy</td>
</tr>
<tr>
<td>Adriamycin</td>
</tr>
<tr>
<td>Alcohol</td>
</tr>
<tr>
<td>Carbon monoxide poisoning</td>
</tr>
<tr>
<td>Other chemotheraphy</td>
</tr>
<tr>
<td>High-output cardiomyopathy</td>
</tr>
<tr>
<td>Tachycardia-mediated cardiomyopathy</td>
</tr>
<tr>
<td>Thyrotoxicosis</td>
</tr>
<tr>
<td>Nutritional (beriberi, thiamine deficiency)</td>
</tr>
<tr>
<td>Peripheral left-to-right shunt lesions</td>
</tr>
<tr>
<td>Anemia</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Asymmetric septal hypertrophry (idiopathic hypertrophic cardiomyopathy)</td>
</tr>
<tr>
<td>Obstructive vs. nonobstructive</td>
</tr>
<tr>
<td>Concentric hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Isolated apical hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Atypical hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Restrictive cardiomyopathy</td>
</tr>
<tr>
<td>Idiopathic</td>
</tr>
<tr>
<td>Infiltrative</td>
</tr>
<tr>
<td>Amyloidosis</td>
</tr>
<tr>
<td>Glycogen storage diseases</td>
</tr>
<tr>
<td>Hemochromatosis</td>
</tr>
<tr>
<td>Postradiation therapy</td>
</tr>
<tr>
<td>Endocardial fibroelastosis</td>
</tr>
<tr>
<td>Other</td>
</tr>
<tr>
<td>Friedreich ataxia</td>
</tr>
<tr>
<td>Muscular dystrophies</td>
</tr>
</tbody>
</table>

**Table 17.2**  
**APPROPRIATENESS CRITERIA FOR THE APPLICATION OF ECHOCARDIOGRAPHY IN TTE IN KNOWN OR SUSPECTED DILATED CARDIOMYOPATHY**
<table>
<thead>
<tr>
<th>Score (1–9)</th>
<th>4. Frequent VPCs or exercise-induced VPCs</th>
<th>A (8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5. Sustained or nonsustained atrial fibrillation, SVT, or VT</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>7. Clinical symptoms or signs consistent with a cardiac diagnosis known to cause lightheadedness/presyncope/syncope (including but not limited to aortic stenosis, hypertrophic cardiomyopathy, or HF)</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>58. Suspected cardiovascular source of embolus</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>67. Initial evaluation of suspected hypertensive heart disease</td>
<td>A (8)</td>
<td></td>
</tr>
<tr>
<td>68. Routine evaluation of systemic hypertension without signs of symptoms of hypertensive heart disease</td>
<td>rA (3)</td>
<td></td>
</tr>
<tr>
<td>70. Initial evaluation of known or suspected HF (systolic or diastolic) based on symptoms, signs, or abnormal test results</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>74. Routine surveillance (&lt;1 yr) of HF (systolic or diastolic) when there is no change in clinical status or cardiac examination</td>
<td>rA (2)</td>
<td></td>
</tr>
<tr>
<td>75. Routine surveillance (≥1 yr) of HF (systolic or diastolic) when there is no change in clinical status or cardiac examination</td>
<td>U (6)</td>
<td></td>
</tr>
<tr>
<td>76. Initial evaluation or reevaluation after revascularization and/or optimal medical therapy to determine candidacy for device therapy and/or to determine optimal choice of device</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>77. Initial evaluation from CRT device optimization after implantation</td>
<td>U (6)</td>
<td></td>
</tr>
<tr>
<td>81. To determine candidacy for ventricular assist device</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>82. Optimization of ventricular assist device settings</td>
<td>A (7)</td>
<td></td>
</tr>
<tr>
<td>83. Reevaluation for signs/symptoms suggestive of ventricular assist device-related complications</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>84. Monitoring for rejection in a cardiac transplant recipient</td>
<td>A (7)</td>
<td></td>
</tr>
<tr>
<td>85. Cardiac structure and function evaluation in a potential heart donor</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>86. Initial evaluation of known or suspected cardiomyopathy (e.g., restrictive, infiltrative, dilated, hypertrophic, or genetic cardiomyopathy)</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>87. Reevaluation of known cardiomyopathy with a change in clinical status or cardiac examination to guide therapy</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>88. Routine surveillance (&lt;1 yr) of known cardiomyopathy without a change in clinical status or cardiac examination</td>
<td>I (2)</td>
<td></td>
</tr>
<tr>
<td>89. Routine surveillance (≥1 yr) of known cardiomyopathy without a change in clinical status or cardiac examination</td>
<td>U (5)</td>
<td></td>
</tr>
<tr>
<td>90. Screening evaluation for structure and function in first-degree relatives of a patient with an inherited cardiomyopathy</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>91. Baseline and serial reevaluation in a patient undergoing therapy with cardiotoxic agents</td>
<td>A (9)</td>
<td></td>
</tr>
<tr>
<td>112. Evaluation to facilitate clinical decision making with regard to anticoagulation, cardioversion, and/or radiofrequency ablation</td>
<td>A (9)</td>
<td></td>
</tr>
</tbody>
</table>
Although the primary diagnostic features of dilated cardiomyopathy are left ventricular dilation and systolic dysfunction, secondary features are common and contribute substantially to symptoms and prognosis. These include diastolic dysfunction with chronic elevation of left atrial pressure, secondary mitral and tricuspid regurgitation, secondary pulmonary hypertension, and concurrent right ventricular dysfunction. The primary and secondary abnormalities seen in dilated cardiomyopathy are listed in Table 17.3. The most common clinical presentation of dilated cardiomyopathy is congestive heart failure with shortness of breath and exercise intolerance. Depending on severity and duration, patients with dilated cardiomyopathy may be asymptomatic, or present with New York Heart Association class I to IV symptoms.

The echocardiographic features of dilated cardiomyopathy parallel the primary and secondary findings noted in Table 17.3. Left ventricular dilation is ubiquitous and a requisite component for establishing the diagnosis of dilated cardiomyopathy. The degree of dilation can be mild or substantial with left ventricular internal dimensions of 9 cm or more occasionally encountered. The distribution of systolic dysfunction within the left ventricular walls is dependent on whether the cardiomyopathy has an ischemic etiology. If an ischemic etiology is present, there usually is greater regional variation in systolic dysfunction than if the process is nonischemic. It should be emphasized, however, that in documented nonischemic cardiomyopathy, there is regional variation in systolic dysfunction, typically with the proximal inferoposterior and posterior lateral walls having relatively preserved function. As a consequence of dilation and systolic dysfunction, the left ventricle takes on a more spherical geometry. Normally, the long-axis dimension of the left ventricle exceeds the minor axis dimension (diameter) with a ratio of 1.6:1 or greater. With progressive dilation, the minor axis increases disproportionally, and the ratio of long to minor axis decreases. Typically, a ratio (sphericity index) of less than 1.5:1 implies pathologic
remodeling. The increasing spherical geometry results in apical and lateral displacement of the papillary muscles. This effectively reduces the length of the mitral apparatus and results in functional mitral regurgitation.

<table>
<thead>
<tr>
<th>Table 17.3</th>
<th>SECONDARY ABNORMALITIES IN CARDIOMYOPATHY</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left ventricular dilation</strong></td>
<td>Increasing sphericity of left ventricular geometry</td>
</tr>
<tr>
<td></td>
<td>Apical and lateral displacement of papillary muscles</td>
</tr>
<tr>
<td></td>
<td>Functional mitral regurgitation</td>
</tr>
<tr>
<td></td>
<td>Left ventricular thrombus</td>
</tr>
<tr>
<td><strong>Left atrial dilation</strong></td>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td></td>
<td>Left atrial thrombosis/stasis of blood</td>
</tr>
<tr>
<td><strong>Pulmonary hypertension</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Tricuspid regurgitation</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Right ventricular dilation/dysfunction</strong></td>
<td></td>
</tr>
</tbody>
</table>

Figures 17.1 through 17.6 depict several features of dilated cardiomyopathy. Notice in Figures 17.1 and 17.2 the relatively mild left ventricular dilation and preservation of normal ventricular geometry. When comparing diastolic and systolic frames, ventricular systolic dysfunction is clearly present, but the ejection fraction is reduced to 35%. Figures 17.3 and 17.4 are more extreme examples of long-standing dilated cardiomyopathy in which the left ventricle has taken on a more spherical geometry. Note the relationship of the maximal lateral dimension to ventricular length, which is increased compared with the geometry seen in normal individuals and increased compared with the milder dilated cardiomyopathy presented in Figures 17.1 and 17.5 depicts secondary mitral regurgitation due to apical and lateral displacement of the papillary muscles, resulting in abnormal apically displaced coaptation of the mitral valve leaflets.
FIGURE 17.1. Parasternal views recorded in a patient with a dilated cardiomyopathy. **A:** In the parasternal long-axis view, note the dilation of the left ventricle (65 mm) and left atrium (50 mm). **B:** In the short-axis view, note the normal circular geometry of the left ventricle and the uniform wall thickness. In real time, all walls are uniformly hypokinetic.

**Video 17-1a**

coming soon

**Video 17-1b**

coming soon
FIGURE 17.2. Apical four-chamber view recorded in the same patient as in Figure 17.1. In this example, normal left ventricular geometry has been preserved, with a long-axis dimension significantly greater than the short-axis dimension, as noted in the schematic in the upper left.
FIGURE 17.3. Parasternal long-axis view recorded in a patient with a long-standing, idiopathic dilated cardiomyopathy revealing marked dilation of the left ventricle but relatively preserved left atrial and right ventricular size. In the real-time image, note the severe global hypokinesis and spherical geometry of the ventricle.
Video 17-3

Figure 17.6 depicts a classic ischemic cardiomyopathy. Note the thin, scarred inferior and inferoposterior walls and generalized hypokinesis of the remaining walls. This image is consistent with an established extensive inferior myocardial infarction with milder degrees of secondary left ventricular dysfunction in the remaining segments, resulting in global systolic dysfunction and reduced global ventricular function.

There are several M-mode findings to be noted in patients with systolic dysfunction. The first is the E-point septal separation (EPSS) defined as the distance (in millimeters) from the anterior septum to the maximal early opening point (E-point) of the mitral valve (Fig. 17.7). Because the internal dimension of the left ventricle is proportional to diastolic left ventricular volume and the maximal diastolic excursion of the mitral valve is proportional to mitral stroke volume, the ratio of the two dimensions will be proportional to the ejection fraction. As such, limited mitral valve opening (manifested by a greater distance between the E-point and the septum) is an indirect indicator of reduced ejection fraction. The normal EPSS is 6 mm, with progressively larger EPSS representing lower ejection fraction. Evaluation of aortic valve motion also provides clues to left ventricular performance. Normally, the aortic valve has crisp opening and closing points and as such opens as a “box” when imaged with M-mode echocardiography. Reduced forward flow results in a more gradual closure during systole so that there is rounding of aortic valve closure due to reduced forward flow (Fig. 17.8).
FIGURE 17.4. Apical four-chamber view recorded in a patient with a dilated cardiomyopathy and spherical ventricular geometry in which the long- and short-axis dimensions are essentially equal. This has resulted in lateral displacement of the papillary muscles and retraction of the mitral apparatus toward the apex. Video 17-4
FIGURE 17.5. Apical four-chamber view recorded in a patient with a nonischemic dilated cardiomyopathy. Note the biatrial enlargement as well as the left
ventricular enlargement and global hypokinesis. In the color flow image, note the functional mitral regurgitation. In the upper panel, note the coaptation of the mitral valve well above the plane of the annulus (dotted line), which is also schematized. Both the tenting area and height, which are related to severity of functional mitral regurgitation, are as noted.
FIGURE 17.6. Parasternal long-axis view recorded in a patient with an ischemic cardiomyopathy. **A:** Recorded in end diastole. Note the dilated left ventricle and the relative preservation of ventricular septal thickness (*upper arrows*) as compared with the thinned posterior wall (PW) (*lower arrows*). **B:** End-systolic frame. Note the hypokinesis of the anterior septum and akinesis of the posterior wall.

Video 17-6
FIGURE 17.7. M-mode echocardiograms recorded in two patients with cardiomyopathy and systolic dysfunction. In each case, note the increased E-point septal separation (EPSS) indicative of reduced ejection fraction. The EPSS is (A) 1.2 cm and (B) 3.0 cm. This suggests that the ejection fraction for the patient represented in B is substantially worse than that in A. The inset in A
demonstrates a classic b-bump in mitral valve closure. Note that the smooth continuation between the A point and the closure point (c) is interrupted by transient reopening of the mitral valve denoted by the B-bump, which is indicative of elevated left ventricular diastolic pressure.

FIGURE 17.8. M-mode echocardiogram recorded through the aortic valve in a patient with a dilated cardiomyopathy and reduced stroke volume. Note the gradual curved closure of the aortic valve at end systole (arrow). This is due to progressively diminishing forward flow as a consequence of severe systolic dysfunction. The small inset in the upper left schematizes the normal opening and closing pattern of the aortic valve.
FIGURE 17.9. M-mode echocardiograms of the lateral mitral annulus recorded from the left ventricular apex. The top panel was recorded in a patient with normal ventricular function and annular excursion toward the apex is 15 mm. The middle panel was recorded in a patient with an ejection fraction of 42% and a dilated cardiomyopathy. Note the annular excursion of 10 mm. The bottom panel was recorded in a patient with an ejection fraction of 21% and reveals annular excursion of 6 mm.
An indirect and nonvolumetric measure of left ventricular systolic function is measurement of the descent of the base of the heart. With normal ventricular contraction, there is motion of the annulus of the heart toward the apex of ≥10 mm. In this technique, an M-mode cursor or a Doppler sample volume is placed in the lateral annulus or the proximal ventricular septum. The total excursion of the annulus toward the apex can then be measured (Fig. 17.9). For patients with global ventricular dysfunction, there is a direct relationship between annular excursion and left ventricular ejection fraction, such that the lower the systolic excursion, the lower the ejection fraction. More recently, analogous information has been derived from Doppler tissue imaging of the mitral annulus (Fig. 17.10). Caution is advised when using these indirect measures of ventricular function, as the range of normal is substantial. For any given patient, they may have value to track serial changes in function over time. In general, these indirect measures of ventricular function are rarely utilized and have been largely supplanted by direct measurement of left ventricular volumes and ejection fraction by two-dimensional echocardiography.
Adult Echo

X5-1
149Hz
13cm

2D
25%
C 45
P Low
3en
TDI
53%
3.2MHz

PW
45%
SF 5.0mm
3.2MHz
10.9cm

s' = 10 cm/sec
LVEF = 60%

PW
35%
SF 5.0mm
3.2MHz
11.7cm

s' = 6 cm/sec
LVEF = 46%

TDI
2.9 MHz
Gain 70
2/4/0

PW
3.3 MHz
Gain D
13.1 cm
Angle 0°
Filter 25Hz
75 mm/s

s' = 3 cm/sec
LVEF = 22%
FIGURE 17.10. Illustration of the s’ wave of the lateral mitral annulus from Doppler tissue tracking over a range of left ventricular systolic function. The top panel was recorded in a patient with normal left ventricular systolic function and an s’ of 10 cm/s. The middle panel was recorded in a patient with an ejection fraction of 46% related to a nonischemic cardiomyopathy with an s’ of 6 cm/s. The bottom panel was recorded in a patient with severe left ventricular systolic dysfunction and an ejection fraction of 22%. Note the markedly reduced s’ of 3 cm/s. It should be stressed that the range of normal for s’ is quite broad and that there may be overlap between varying degrees of left ventricular systolic function. As such this value should not be used as a stand-alone assessment, but placed in context of other findings and/or serial changes.

FIGURE 17.11. Illustration of the Simpson method, or method of discs for calculating ventricular volumes. Apical four-chamber view recorded at end diastole from which the endocardial border has been traced is presented. In this example, the two annular margins and apex have been manually identified (small square boxes) after which the endocardial border has been automatically tracked and manually adjusted as needed. From this a left ventricular diastolic volume in the four-chamber view is calculated at 135 mL. This process is repeated at end systole for the four-chamber view and for both diastole and systole for the apical two-chamber view from which a biplane ventricular volume can be calculated. From these volumes stroke volume and ejection fraction are then calculated as noted in the panel at the upper left.

Once the diagnosis of cardiomyopathy has been established, it is clinically
useful to quantify the degree of systolic dysfunction. Previously used two-dimensional echocardiographic measures of left ventricular function included linear- and area-based measurements of left ventricular size from which the derived parameters of fractional shortening and fractional area change were calculated. In modern practice, quantitation of ventricular function is done by assessment of ventricular volumes from two- or three-dimensional echocardiography. From these data sets, left ventricular volume can be determined in systole and diastole, from which stroke volume and ejection fraction are calculated (Fig. 17.11).

Three-dimensional echocardiography has the ability to quantify left ventricular volumes not only at mid diastole and end systole but throughout the cardiac cycle (Fig. 17.12). Regional volume changes can also be determined from this three-dimensional volume. Multiple studies have demonstrated the superiority of three-dimensional echocardiography over two-dimensional volume quantitation with respect to both absolute accuracy and reproducibility. Three-dimensional echocardiography remains limited by reliance on automatic edge detection algorithms, which may result in erroneous data in a poor-quality data set where the entire endocardial border is not easily identified.

Evaluation of left ventricular deformation with strain rate and torsion evaluation provides valuable information regarding left ventricular systolic and, to a lesser degree, diastolic function in cardiomyopathy. The basic methods for this were discussed in Chapter 5 dealing with evaluation of left ventricular systolic function. Multiple parameters have been evaluated for their ability to accurately provide information regarding systolic function in a clinically relevant manner. Because of its relatively standardized performance, global longitudinal strain (GLS) may be the single most reliable parameter available for evaluation of left ventricular detailed mechanics (Fig. 17.13). It has seen widespread application in dilated cardiomyopathy for following the status of patients receiving cardiotoxic chemotherapy and other uses. Strain rate imaging and ventricular torsion have seen far less acceptance because of their technical complexity and often intrinsic noise.
FIGURE 17.12. Three-dimensional volumetric image of a patient with a nonischemic dilated cardiomyopathy and markedly reduced left ventricular systolic function. The panel at the lower right is a three-dimensional reconstruction of the left ventricular cavity. The two upper panels are the apical four- and two-chamber views from which the annular points and apex have been identified, after which the chamber boundaries are automatically tracked. From this left ventricular volume is calculated continuously throughout the cardiac cycle as seen in the graph at the lower left. Diastolic and systolic volumes as well as stroke volume and ejection fraction are then calculated as noted.
FIGURE 17.13. Illustration of tissue tracking determination of global longitudinal strain and subsequent calculation of ventricular volumes and ejection fraction. The image at the upper left is an apical four-chamber view recorded in mid systole. Strain of the seven visualized segments is as noted. The graph at the bottom depicts the strain in each of the seven separate segments. Note that strain is reduced in each of the seven segments but reaches its maximum value at a similar time point. At the lower right is a bull's-eye image from the same study which incorporates strain recorded in the apical four-chamber, apical three-chamber, and apical two-chamber views from which global longitudinal strain of the left ventricle is calculated as –10.6%. Diastolic and systolic volumes as well as ejection fraction are as noted in the upper right.
Doppler parameters, which can be employed to evaluate systolic and diastolic dysfunction in cardiomyopathy, are listed in Table 17.4 and are discussed in Chapters 5 and 6. Stroke volume can be determined by recording the time velocity integral (TVI) in the left ventricular outflow tract which, when multiplied by the cross-sectional area of the left ventricular outflow tract, provides actual volume of flow. For any individual patient, one can assume the outflow tract area remains a constant, and, therefore, comparison of the TVI alone provides a reliable means for comparing the left ventricular stroke volume at different time points. Figure 17.14 shows examples of left ventricular outflow tract TVI in patients with dilated cardiomyopathy and varying degrees of systolic dysfunction. Once the per-beat stroke volume has been determined, cardiac output can be calculated as the product of the heart rate and forward stroke volume.

<table>
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<th>Table 17.4</th>
<th>ROLE OF DOPPLER ECHOCARDIOGRAPHY IN CARDIOMYOPATHY</th>
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<tr>
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<td>Doppler-based left ventricular outflow tract time velocity integral (TVI)</td>
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Assessment of diastolic properties of the right ventricle
Doppler flow in the hepatic veins
Superior vena caval Doppler flow

FIGURE 17.14. Left ventricular outflow tract time velocity integral (TVI) recorded in three patients with cardiomyopathy and reduced forward stroke volume. In the upper panel, note the marked decrease in TVI of 6 cm with less reduction in the
middle panel. The bottom panel was recorded in a patient with severe left systolic
dysfunction and reveals beat-to-beat variability in both the peak velocity and TVI
which is a Doppler correlate of *pulsus alternans*, a clinical finding noted in
advanced systolic dysfunction.

**FIGURE 17.15.** Doppler through the left ventricular outflow tract in a patient with
a dilated cardiomyopathy and severely reduced left ventricular systolic function. In
the upper panel note the relatively normal time velocity integral of the first QRS
complex, followed by absent flow (X) with a premature ventricular contraction. The
post-PVC beat has an augmented TVI (*upper-pointing arrow*) and the following
sinus beat a markedly reduced TVI. The bottom panel was recorded in the same
patient using continuous-wave Doppler through the left ventricular outflow tract. Note that only every other QRS complex is associated with forward flow in the left ventricular outflow tract. Alternate complexes (X) are not associated with any evidence of forward flow. This is the Doppler equivalent of clinical pulsus alternans.

An additional observation which can be made from the TVI of the left ventricular outflow tract in dilated cardiomyopathy is detection of pulsus alternans. This typically occurs following a premature ventricular contraction, after which the post-compensatory sinus beat is augmented. Following this alternate beats have a greater stroke volume and therefore greater blood pressure. Pulsus alternans is one of the classic physical examination signs of an advanced cardiomyopathy with reduced left ventricular systolic function and has a definite correlate in the Doppler profile of the LVOT (Fig. 17.15).

A final means for assessing left ventricular systolic function is calculation of the left ventricular dP/dt (see Chapter 5 for detailed methodology). Figure 17.16 illustrates the range of left ventricular dP/dt encountered in patients with dilated cardiomyopathy. This noninvasively determined dP/dt correlates well with values determined by cardiac catheterization, and dP/dt <600 mm Hg/s has been associated with a worsened prognosis.

Assessment of diastolic function in dilated cardiomyopathy provides valuable clues to the pathology underlying the development of symptoms. This topic has been extensively discussed in Chapter 6. It is important to integrate multiple observations of diastolic function to reliably determine the status of left atrial filling pressures and overall diastolic function. The echocardiographic and Doppler parameters that can be used to evaluate diastolic dysfunction in dilated cardiomyopathy are listed in Table 17.4. There are several parameters that should be obtained in all patients with cardiomyopathy, including mitral valve inflow patterns and Doppler tissue imaging for annular velocity.

By combining the mitral valve inflow pattern with information from Doppler tissue imaging of the annulus, an index of mitral valve (E) to annular E velocity (e’) can be obtained (Fig. 17.17). There is a rough correlation between the index (E/e’) and left atrial filling pressure. The majority of individuals with E/e’ ≥15 have elevated pulmonary papillary wedge pressures and individuals with E/e’ ≤8 generally have low left atrial filling pressures.
E/e' values between these values are associated with a broad range of filling pressures. This measure appears independent of heart rate and, because it relies only on early filling velocities, is also valid in patients with atrial fibrillation. Recent data have suggested that this relationship may be substantially less robust in clinical practice than initially reported, especially in patients with severe left ventricular dysfunction or acutely decompenated heart failure.
**FIGURE 17.16.** Examples of left ventricular $dP/dt$ calculated from continuous-wave Doppler of mitral regurgitation in three patients with dilated cardiomyopathy and varying degrees of left ventricular systolic dysfunction. **A:** Left ventricular $dP/dt$ is relatively preserved at 967 mm Hg/s. **B, C:** Moderate and marked reduction in left ventricular $dP/dt$ is noted.

**FIGURE 17.17.** Composite imaging recorded in a patient with a nonischemic dilated cardiomyopathy. The central image is an apical four-chamber view from which the left atrial volume has been calculated and is noted to be markedly enlarged at 66.9 mL/m². The panel at the upper right is the mitral inflow pattern revealing an $E/A$ ratio of 1.9 with a short deceleration time. The panel at the lower right is the lateral annular velocities which are pathologically diminished with $e'$ of 5 cm/s. The average $E/e'$ ratio was 22.5 suggesting elevated left atrial pressure.
The myocardial performance index is a unitless number reflecting global left ventricular systolic and diastolic performance. It is defined as the ratio of the total isovolumic times (isovolumetric contraction and relaxation) to ejection time (Fig. 17.18). It is calculated from Doppler tracings of the left ventricular outflow tract and mitral valve inflow. Normally, this value is 0.40 or less, with increasing values representing progressively worse left ventricular performance. It has been shown to provide independent prognostic information in patients with heart failure due to dilated cardiomyopathy.
SECONDARY FINDINGS IN DILATED CARDIOMYOPATHY

Secondary features of dilated cardiomyopathy that can be detected with echocardiography are listed in Table 17.3. Some secondary findings, such as left atrial dilation and right heart involvement, are nearly ubiquitous and are an essential part of establishing the diagnosis. Others, such as secondary mitral regurgitation, thrombus formation, and secondary pulmonary hypertension occur to a variable degree and are dependent on both the severity and duration of cardiomyopathy.
Some degree of left atrial dilation is ubiquitous in dilated cardiomyopathy and is dependent on the duration of cardiomyopathy. The left atrium can dilate to substantial dimensions, and left atrial dimensions of more than 6 cm are occasionally encountered. Left atrial area or volume can be measured from the apical view. In the setting of left ventricular dysfunction, left atrial dilation, whether quantified as a linear dimension, area, or volume is a marker of more severe and long-standing ventricular dysfunction. Left atrial dilation is largely due to elevated diastolic pressures in the left ventricle and often concurrent mitral regurgitation. It may also be due to a myopathic process in the atrial wall. All these result in a heightened likelihood of developing atrial fibrillation or flutter. Recent data suggest a strong, independent relationship between left atrial area or volume and prognosis in patients with cardiomyopathy, as well as numerous other conditions.

As a consequence of left atrial dilation, especially if seen in the presence of poor atrial mechanical function or atrial fibrillation, left atrial spontaneous contrast is not uncommonly encountered with transesophageal imaging. Occasionally, spontaneous contrast may be seen in the left ventricle as well (Fig. 17.19). Left ventricular thrombus can form in patients with dilated cardiomyopathy and reduced left ventricular systolic function. The appearance of thrombus can be highly variable and ranges from mural, where it adheres to the overall wall geometry, to pedunculated and mobile. Figures 17.20 through 17.24 depict the range of thrombus burden seen in cardiomyopathy. Notice in Figure 17.20 the pedunculated sessile thrombus at the apex of the left ventricle and in Figure 17.21 the more pedunculated and complex mobile thrombus along the inferior wall. In addition to standard two-dimensional echocardiography, three-dimensional echocardiography can be utilized to further define the location and characteristics of thrombus (Fig. 17.20). On occasion, thrombi assume a cystic-type appearance. This may be related either to liquefaction of an otherwise solid thrombus or the characteristics of ultrasound where only the leading and trailing edges of a thrombus are detected (Fig. 17.23). Left ventricular contrast agents can be utilized to further define thrombus (Fig. 17.23B), or in equivocal cases confirm or refute the potential diagnosis. Thrombi may become chronically adherent to a wall and even calcify (Fig. 17.24). In general, detection of thrombus in the presence of dilated cardiomyopathy confers the same thromboembolic risk as it does in patients with acute myocardial infarction.
FIGURE 17.19. Apical four-chamber view recorded in a patient with a dilated cardiomyopathy and severe left ventricular systolic dysfunction. This image was recorded in early diastole. Note the spontaneous echo contrast filling the left ventricular cavity, better appreciated in the real-time image. In this diastolic frame, there is forward flow from the left atrium into the left ventricle creating a negative contrast effect just distal to the mitral valve tips (arrows).
FIGURE 17.20. Apical views recorded in a patient with a dilated cardiomyopathy and a pedunculated apical thrombus. In the routine two-dimensional image, note the filling defect in the apex (arrows), which is also seen in the four-chamber view.
extracted from the three-dimensional data set. The small inset is a short-axis view of the ventricular apex, extracted from a three-dimensional set, also identifying the nearly spherical thrombus. 

Video 17-20a

coming soon

Video 17-20b
FIGURE 17.21. Off-axis apical imaging in a patient with an ischemic cardiomyopathy and a pedunculated, mobile left ventricular thrombus (arrows) along the distal inferoapical wall. The inset at the lower left is real-time three-dimensional imaging in the same patient again demonstrating a spherical, pedunculated thrombus at the inferoapical wall. [Video 17-21]
FIGURE 17.22. Apical four-chamber view recorded in a patient with an ischemic cardiomyopathy and an established apical infarct. In the central panel note the sessile thrombus at the apex of the left ventricle (arrow). The inset at the lower right is a subcostal four-chamber view in which a pedunculated thrombus can be seen at the apex of the right ventricle (arrow). Video 17-22
Because of increasing spherical geometry of the left ventricle, normal coaptation of the mitral leaflets becomes interrupted as the papillary muscles are displaced apically and laterally. This results in a shortened length of coaptation of the mitral valve leaflets, which ordinarily coapt along a several-millimeter length of their edge (the zona coapta). With displacement of the papillary muscles, functional mitral regurgitation occurs as the leaflets coapt only at their tips or occasionally fails to make contact at all during systole. Figures 17.5 and 17.25 to 17.29 depict functional mitral regurgitation in patients with dilated cardiomyopathy. Quantitation of mitral regurgitation is undertaken in a manner identical to that described in Chapter 11 for other etiologies of mitral regurgitation. The severity of functional mitral regurgitation is most closely related to mitral annular diameter and the tenting area of the mitral leaflets (Fig. 17.5).
FIGURE 17.23. Apical four-chamber view recorded in a patient with a nonischemic dilated cardiomyopathy with components of noncompaction. In A, note the oval cystic-appearing mass at the left ventricular apex which appears attached to the septum by a broad stalk (arrows). This is consistent with a mobile thrombus in the presence of a dilated cardiomyopathy. B was recorded after injection of a contrast agent for left ventricular opacification and nicely defines the
boundary of the clot within the apex of the left ventricle (double-headed arrows).
FIGURE 17.24. Apical four-chamber view recorded in a patient with a long-standing dilated cardiomyopathy and a large complex chronic thrombus along the mid-lateral wall (arrows). Note the marked heterogeneity of the echotexture of the thrombus with components suggesting calcification. The inset at the upper left is a cardiac CT scan from the same patient, clearly demonstrating the calcific components of the thrombus. (Video 17-24)

Video 17-24

In some patients with marked ventricular remodeling and concurrent
diastolic dysfunction, diastolic mitral regurgitation may develop. This is the result of a marked increase in diastolic pressure and reversal of the left atrial to left ventricular pressure gradient in diastole. This phenomenon is heart rate dependent and is most often seen in patients with concurrent heart block or pronounced bradycardia. Although identifiable with color flow imaging, the timing of this phenomenon is best appreciated from the spectral Doppler display.

**FIGURE 17.25.** Parasternal long-axis view recorded in a patient with a nonischemic cardiomyopathy and moderate to severe functional mitral regurgitation. In the central illustration, recorded in early systole, note the dilated left atrium and left ventricle and the apical displacement of the mitral leaflet tips. At the upper left is an expanded view of the mitral valve in which coaptation failure (arrow) is demonstrated. The panel at the lower right was recorded with color-flow Doppler and demonstrates moderate to severe functional mitral regurgitation.
Video 17-25

coming soon

Video 17-25 CFD

coming soon
FIGURE 17.26. Parasternal short-axis view recorded in same patient mentioned in Figure 17.25. The central illustration was recorded at mid systole at the level of the mitral valve tips. Note the areas of failure of coaptation noted by the small “x.” At the upper left is a short-axis color Doppler image recorded at the same level from which an elongated oval regurgitation jet is depicted in its short axis. At the lower right is a real-time three-dimensional image recorded from the left atrial perspective in this patient in which the elongated slit-like regurgitant orifice (×) can be seen in this mid systolic image. Video 17-26 3D
Because of either concurrent involvement of the right ventricle or secondary pulmonary hypertension and subsequent tricuspid annular dilation, tricuspid regurgitation is frequently noted in advanced cardiomyopathy (Fig. 17.30). The tricuspid regurgitation jet can be used to determine right ventricular systolic pressure as for any other cause of pulmonary hypertension.

**ETIOLOGY OF DILATED CARDIOMYOPATHY**

It is often not possible to determine the etiology of a dilated cardiomyopathy. Table 17.1 lists a number of dilated cardiomyopathies, some of which can be
specifically identified using echocardiographic techniques. A clinically relevant distinction to be made is between an ischemic and nonischemic cardiomyopathy. Distinguishing features of an ischemic cardiomyopathy include a relatively greater degree of regional heterogeneity of systolic function often with areas of frank scar or aneurysm formation. When either a substantial area of scar, conforming to a well-defined coronary territory, or left ventricular aneurysm is noted, the likelihood of an ischemic etiology is high. Figure 17.6 was recorded in a patient with a classic ischemic cardiomyopathy. There was global left ventricular dysfunction with frank akinesia with a shallow aneurysm in the posterior wall, allowing an echocardiographic diagnosis of ischemic cardiac disease to be made. Often patients will present with a dilated, globally hypokinetic ventricle but no obvious evidence of myocardial infarction. In these instances, there may be no echocardiographic features that allow an ischemic etiology to be established. Even in the presence of a nonischemic cardiomyopathy, there will be regional variation in left ventricular systolic dysfunction, typically with the proximal inferoposterior and posterior lateral walls having relatively preserved function when compared with the other regions. Because of heterogeneity in regional wall stress, the degree of dysfunction can also vary when apical and basal segments are compared. Stress echocardiography, generally with dobutamine, has shown promise for separating ischemic from nonischemic cardiomyopathy. (See Chapter 15 for further discussion of ischemic cardiomyopathy.)
One form of dilated cardiomyopathy that can be diagnosed with near certainty using echocardiography is noncompaction of the myocardium. Developmentally, the ventricular myocardium begins as a series of sinusoids that compact into organized myocardial fibers. Occasionally during development, compaction fails to occur and the ventricular myocardium
persists in the embryonic noncompacted state, which does not provide the requisite level of contractile efficiency to protect ventricular geometry. Typically, these individuals present in either childhood or the second or third decade of life, often with arrhythmias, left ventricular dilation, and global systolic dysfunction. Embolic events are also more common. Figures 17.31 to 17.36 were recorded in patients with noncompacted myocardium. In each instance, note the honeycombed appearance of the myocardium, which may be generalized or focal. This appearance initially could be confused with multiple ventricular thrombi, but its diffuse nature is a distinguishing characteristic that allows the diagnosis of noncompaction to be made. The amount of myocardium involved with noncompaction is highly variable. Occasional patients are identified who have limited areas of noncompaction with only mild compromise of ventricular function. Limited regions of noncompaction may occasionally be encountered in hypertrophic and other cardiomyopathy as well.

**FIGURE 17.28.** Apical four-chamber view recorded in a patient with a dilated cardiomyopathy and eccentric functional mitral regurgitation. Note the relatively normal hemispherical PISA (arrows) and the eccentric, laterally directed severe mitral regurgitation jet within the left atrium. The inset at the upper left is a detailed
view of the mitral valve in systole. Note the failure of coaptation with apical tethering of the lateral leaflet (arrow) compared to the medial leaflet of the mitral valve. This results in a laterally directed mitral regurgitation jet.

CMR has proven of substantial value in identifying areas of myocardial noncompaction and, because of its intrinsic three-dimensional acquisition and high resolution, provides an excellent method for quantifying the amounts of noncompacted myocardium (Fig. 17.36). Several larger-scale studies of cardiac MR in cardiomyopathy have demonstrated that many patients with a dilated cardiomyopathy may have limited regions of noncompaction which may be either an incidental finding or part of the pathophysiology of an idiopathic dilated cardiomyopathy.
FIGURE 17.29. M-mode echocardiogram recorded in a patient with a nonischemic-dilated cardiomyopathy and reduced left ventricular systolic function. In the upper panel, note the increased E-point septal separation consistent with reduced ejection fraction and the suggestion that the anterior and posterior leaflets do not fully appose in systole. The lower panel is a color Doppler M-mode from the same echocardiogram demonstrating a systolic jet within the boundary of the mitral valve leaflets in systole.
FIGURE 17.30. Apical four-chamber view recorded in a patient with a dilated cardiomyopathy. Due to the combination of right ventricular dysfunction and pulmonary hypertension, the tricuspid annulus is dilated, and there is evidence of functional tricuspid regurgitation. In this example, the right ventricular systolic pressure can be calculated as 74 mm Hg from the continuous-wave Doppler image, which revealed a right ventricular right atrial gradient of 64 mm Hg.

Video 17-30
FIGURE 17.31. Apical long-axis view recorded in a patient with systolic
dysfunction related to ventricular noncompaction. Note the combination of protruding trabecular myocardial echoes and interstitial spaces, which fill in with color Doppler flow imaging.
FIGURE 17.32. Apical four-chamber view recorded in a patient with marked left ventricular noncompaction and subsequent severe systolic dysfunction. The arrows on the right denote the area of the true lateral border of the left ventricle. Note the marked area of noncompaction along the lateral wall with multiple complex components and invaginations in the left ventricle (smaller arrows). The inset at the upper left is a real-time three-dimensional echocardiogram recorded in the same patient in which the sponge-like mass of noncompacted myocardium is clearly visible (arrows).
Many dilated cardiomyopathies may be the sequelae of acute myocarditis that may not have been clinically recognized. If echocardiographic imaging is performed early in the course of this disease, one will classically note relatively preserved wall thickness and chamber size with global systolic dysfunction. If there is no spontaneous recovery of the myocarditis, progressive dilation and wall thinning with increasing left ventricular dysfunction will typically occur. More often, however, patients present after the chambers have dilated and thinned and therefore are indistinguishable from cardiomyopathy from other etiologies. CMR performed in the acute phase can document systolic dysfunction and delayed gadolinium will show a pattern of diffuse uptake consistent with myocardial inflammation and edema. This topic is discussed subsequently.
FIGURE 17.33. Three-dimensional echocardiographic image recorded in a patient with dilated cardiomyopathy related to ventricular noncompaction. Note the complex, honeycombed appearance of the endocardial surface which is the result of the multiple noncompacted sinusoids.
FIGURE 17.34. Apical four-chamber view recorded in a patient with a markedly dilated apex and global systolic dysfunction. A: Note the dilation and rounding of the ventricular apex, which is filled with vague echoes. B: Recorded after injection of intravenous contrast for left ventricular opacification and clearly demonstrates multiple small sinusoidal spaces (arrows) consistent with ventricular noncompaction.
Poorly controlled hypertension results in hypertensive cardiovascular disease and, when long standing, the appearance of a dilated cardiomyopathy. In this instance, left ventricular hypertrophy typically persists in the presence of chamber dilation and global dysfunction (Fig. 17.37). The combination of hypertrophy with moderate dilation and global dysfunction is fairly typical of end-stage hypertensive cardiovascular disease with subsequent left ventricular dysfunction but could also be mimicked by a variety of infiltrative diseases. Significant diastolic function is invariably seen as well.

Long-standing renal disease, typically in a patient on dialysis, can also result in a fairly characteristic cardiomyopathy. The concurrent metabolic abnormalities and hypertension result in annular calcification with marked left ventricular hypertrophy. Left ventricular systolic dysfunction and congestive heart failure are present due to a combination of metabolic effects and the effects of long-standing hypertrophy. On occasion, such individuals have shown improvement in ventricular function after either renal transplantation or more aggressive dialysis regimens. This topic is discussed in Chapter 22 which deals with echocardiography in systemic diseases.
FIGURE 17.35. Apical four-chamber view recorded in a patient with ventricular noncompaction. The central image is an apical four-chamber view in which marked noncompaction of the ventricular apex can be noted. Also note the suggestion of hypertrabeculation of the right ventricular lateral wall (arrows) presumably related to a similar phenomenon. The inset at the upper right is a short-axis cardiac MRI recorded in the same patient. Note the marked amount of noncompaction of the left ventricle. Also note the hypertrabeculation and/or noncompaction noted in the right ventricular free wall (arrows).
Cardiac sarcoidosis is probably underrecognized as a source of cardiomyopathy. From an echocardiographic perspective these patients may present with atypically located regional wall motion abnormalities and/or global left ventricular systolic dysfunction. CMR with gadolinium enhancement may reveal findings relatively specific to cardiac sarcoidosis, including epicardial enhancement and atypically located areas of enhancement in the proximal septum. This issue is discussed in Chapter 22 dealing with systemic diseases.

A subset of patients with advanced arrhythmogenic right ventricular dysplasia (AVRD) will have concurrent left ventricular systolic dysfunction (Fig. 17.38). The diagnosis can be established when there is disproportionate right ventricular enlargement and dysfunction with characteristic hypertrabeculation and other findings. The appearance of the left ventricle is similar to that in any other form of nonischemic dilated cardiomyopathy.

**DETERMINATION OF PROGNOSIS IN DILATED CARDIOMYOPATHY**

Several echocardiographic and Doppler findings can be related to prognosis in dilated cardiomyopathy. These are listed in Table 17.5. In addition to long established parameters of left ventricular size, volume, and ejection fraction, more recently developed parameters of left ventricular mechanics, such as systolic strain, have been shown to have prognostic value. Patients with lower GLS have a worse prognosis than patients with relatively preserved GLS (Fig. 17.13).

Doppler parameters provide substantial prognostic information. The Doppler finding carrying the most important prognostic information is a restrictive filling pattern or grade 3/4 diastolic dysfunction (Fig. 17.17). This is characterized as a high E/A ratio, typically greater than 2.5, in association with a short deceleration time (<130 to 150 ms) and reduced annular e’. This pattern indicates an advanced degree of diastolic dysfunction. This pattern also implies marked elevation of end-diastolic and left atrial pressures and as such is often seen in individuals with marked left atrial dilation and secondary pulmonary hypertension. The adverse prognosis associated with a restrictive filling pattern has been demonstrated in numerous studies.
Diastolic dysfunction is additive to reduced systolic function with respect to prognosis, and patients with advanced diastolic dysfunction and severe systolic dysfunction typically have a 2-year survival of less than 50%. A parameter unique to echocardiography is the myocardial performance index, which combines both systolic and diastolic performance (Fig. 17.18). Myocardial performance index of more than 0.40 has been linked to adverse prognosis in a broad range of disease states, including dilated cardiomyopathy.

**FIGURE 17.36.** Transthoracic echocardiogram recorded in a patient with a nonischemic cardiomyopathy related to ventricular noncompaction. **A** is an apical four-chamber view. The small downward-pointing arrows denote the location of the true ventricular apex. The inward-pointing arrows denote the margins of the noncompacted myocardium. Incidental note is made of a defibrillator lead in the right ventricle (long arrow). The inset at the lower right is a cardiac MRI recorded from the same patient. The small double-headed arrow denotes the thickness of the normal compacted myocardium and the longer double-headed arrow the extent of ventricular noncompaction. Note the similarity between the ratio of compacted to noncompacted myocardium in the cardiac MRI and echocardiogram. **B** is a short-axis view recorded in the same patient, again showing marked areas of noncompaction (long double-headed arrow) with a smaller proportion of compacted myocardium (short double-headed arrow). The inset at the lower left is a cardiac MRI recorded in the same patient in a short-axis view in which the marked extent of ventricular noncompaction (double-headed arrow) can also be appreciated.
The presence of mitral and tricuspid regurgitation also affects prognosis. As a general rule, more severe mitral regurgitation is the sequelae of greater left ventricular dilation and changes in geometry, and, as such, the impact of mitral regurgitation independent of the underlying process is difficult to establish. Several studies have demonstrated, however, that increasing degrees of mitral and tricuspid regurgitation correlate with a worse prognosis. Severe mitral regurgitation in the presence of systolic dysfunction in patients with congestive heart failure carries a prognosis substantially worse than that of individuals with lesser degrees of mitral regurgitation. In addition, the left ventricular $dP/dt$, calculated from mitral regurgitation spectral velocity, also has been shown to carry prognostic information with the likelihood of events
being inversely proportional to positive and negative $dP/dt$.

**FIGURE 17.37.** Parasternal long-axis view recorded in a patient with long-standing, poorly treated hypertension who has developed systolic dysfunction. Note the left ventricular hypertrophy with only mild chamber dilation and the global hypokinesis in the real-time image. The mitral valve inflow pattern reveals a short deceleration time of 110 ms and there are reduced annular velocities, all consistent with grade 3 diastolic dysfunction. 

Video 17-37
FIGURE 17.38. Transthoracic echocardiogram recorded in a patient with severe left ventricular systolic dysfunction noted as a concurrent feature of marked
arrhythmogenic right ventricular dysplasia (ARVD). A is an apical four-chamber view from which marked dilation of the right ventricle is noted. Also note the marked hypertrophic elation in the right ventricle which is a feature of this entity. Left ventricular systolic function is globally diminished which can be appreciated in the real-time image. B: Parasternal short-axis view recorded in the same patient. Note the marked dilation and excess trabeculation of the right ventricle and in the real-time image the global hypokinesis of the left ventricle.

Table 17.5
ECHOCARDIOGRAPHIC AND DOPPLER PREDICTORS OF ADVERSE PROGNOSIS IN CARDIOMYOPATHY
**THE ROLE OF ECHOCARDIOGRAPHY IN BASIC AND ADVANCED THERAPY**

Although decisions regarding specific forms of medical and nonmedical therapy should be made on clinical grounds and incorporating all available data, the echocardiogram can play a valuable role in stratifying patients into different therapeutic subtypes. Detection of a dilated cardiomyopathy with systolic dysfunction identifies a patient for whom combined therapy with angiotensin-blocking drugs, β-blockers, and spironolactone has been shown to provide symptomatic and prognostic benefit. Similarly, avoidance of this type of therapy in individuals with other types of cardiomyopathy (e.g., hypertrophic) may be appropriate. Restrictive physiology identifies an end-stage subpopulation for whom very aggressive management is indicated and when combined with other parameters may identify a subset of patients likely to be volume overloaded for whom aggressive diuretic therapy may be beneficial. It should be emphasized, however, that decisions regarding appropriate specific therapies should be made using a combination of clinical, echocardiographic, and other information and not based on echocardiographic observations alone. Another clinical decision that is based on determination of left ventricular ejection fraction is implantation of an automatic implantable defibrillator. Multiple clinical trials have demonstrated the threshold level of left ventricular ejection fraction below which prophylactic implantation of an implantable defibrillator is cost effective and efficacious for patient survival.
Biventricular Pacing for Cardiomyopathy

One approach to treatment of patients with dilated cardiomyopathy and concurrent left bundle branch block is biventricular resynchronization therapy. A percentage of patients with a left bundle branch block and no other identifiable etiology (such as coronary disease, prior myocarditis, or exposure to chemotoxic agents) progressively develop global left ventricular systolic dysfunction (Fig. 17.39). Other than the typical left bundle branch block mediated septal abnormality, these cardiomyopathies are not distinguishable on echocardiography from a cardiomyopathy of any other etiology. It is hypothesized that the mechanical dyssynchrony related to the left bundle branch block results in progressively inefficient global left ventricular mechanics, which over time results in a dilated cardiomyopathy. Resynchronization of left ventricular contraction with a biventricular pacemaker, in which the right ventricle and left ventricle (typically from a coronary sinus lead) are synchronously paced, results in improved symptomatic status, survival benefit, and improvement in left ventricular volumes and parameters of systolic function, such as left ventricular ejection fraction (Fig. 17.40). When decisions for proceeding with CRT are based on the presence of a left bundle branch block, reduced left ventricular ejection fraction, and symptom status, approximately 70% of patients will achieve a benefit from CRT with respect to one or more parameters of survival, symptomatic status, left ventricular remodeling, and systolic function. It should be emphasized that virtually all clinical trials have demonstrated that patients may not improve in all of these different areas.
FIGURE 17.39. Apical four-chamber view recorded in a patient with a documented nonischemic cardiomyopathy related to left bundle branch block. The central image is an end-systolic view of the left ventricle in which left ventricular dilation and reduced systolic function are apparent. The inset at the upper left was recorded with color Doppler and reveals moderate functional mitral regurgitation.

Video 17-39 CFD
FIGURE 17.40. Apical four-chamber view recorded in the same patient presented in Figure 17.39. This echocardiogram was recorded after resynchronization with a biventricular pacemaker. The four-chamber view was recorded at end systole. Note the reduced left ventricular cavity size and normal left ventricular systolic function compared to the preresynchronization image. The inset at the upper left was recorded with color Doppler imaging and reveals very mild residual mitral regurgitation.
Video 17-40 CFD

coming soon

Video 17-40

coming soon
FIGURE 17.41. Parasternal short-axis view and M-mode echocardiogram recorded in a patient with left ventricular systolic dysfunction related to left bundle branch block. A: Note the full-thickness myocardium and normal circular geometry of the ventricle, which has markedly impaired function in the real-time image. B: On the M-mode echocardiogram, note the septal to posterior wall delay (SPWΔ) of 390 ms consistent with marked dyssynchrony between the ventricular septum and posterior walls.
FIGURE 17.42. Doppler tissue imaging for velocity in the proximal septum and lateral walls in a patient with left bundle branch block being considered for resynchronization therapy. Peak systolic velocity is as noted by the diagonal arrows for both the ventricular septum (IVS) and lateral walls. The time difference (Δ) between IVS and lateral wall peak velocity is prolonged at 120 ms. AVC, aortic valve closure; AVO, aortic valve opening.

Because the etiology of left bundle branch block–mediated cardiomyopathy is presumed to be mechanical dyssynchrony, multiple
studies have attempted to identify echocardiographic parameters of dyssynchrony which would be predictive of a favorable CRT response. One of the earlier echocardiographic parameters evaluated was the M-mode derived temporal difference between septal and posterior wall maximum excursion (SPWΔ, Fig. 17.41). Either Doppler-derived or tissue tracking parameters of delayed lateral wall contraction have also been evaluated (Figs. 17.42 and 17.43). While each of these parameters has shown predictive value for a favorable response in relatively small single-center studies, when applied prospectively in larger populations, no one parameter or combination of parameters has proven to be substantially more accurate than the prediction afforded by the presence of a left bundle branch block with a QRS greater than 150 ms in the presence of reduced systolic function and symptoms. At this time, the major electrophysiology societies do not recommend using echocardiographic methods as the primary decision making tool for recommend CRT.
representation of volume change in each of the 17 analyzed segments and the table outlines multiple parameters of dyssynchrony based on time-to-minimum volume in each of the subsegments.
There are several echocardiographic findings which may provide some incremental information with respect to managing patients with left bundle branch block and congestive failure. Relatively small studies have suggested that the patients with severely remodeled left ventricles with marked left ventricular dilation are less likely to be favorable responders than those with relatively smaller left ventricles. Clinically, patients with a shorter duration of left ventricular dysfunction and symptoms are also more likely to have a favorable response. A category of CRT patients known as “hyperresponders” has been defined as those with normalization of left ventricular systolic function following CRT. These patients have been identified as those with a left bundle branch block as the only potential source for left ventricular systolic dysfunction, relatively short duration of symptoms, and relatively smaller left ventricles. Echocardiography can obviously play a role in helping identify this subset of patients.

After placement of CRT device, echocardiography is an appropriate tool for evaluating the response, including quantitative assessment of changes in left ventricular diastolic and systolic volumes and ejection fraction and the secondary effect that remodeling has on functional mitral regurgitation.

Echocardiography can also be used for adjusting the atrioventricular (AV) delay in the CRT device. The goal of optimizing AV delays to ensure that conduction through the native conduction system is minimized and that the left ventricle is exclusively activated by the biventricular device, as well as to ensure that optimal diastolic filling time is allowed. The ideal AV delay allows for clear discrimination between an E and an A wave. If the AV interval is too short, atrial contribution to left ventricular filling is compromised, and if the AV interval is excessively long, native conduction still allows dyssynchronous contraction. A complete “titration” of AV delay can be done while monitoring a variety of parameters of left ventricular performance, including mitral inflow, left ventricular ejection fraction, TVI.
of the left ventricular outflow tract, and severity of mitral regurgitation (Figs. 17.44 and 17.45). Left ventricular dP/dt derived from the continuous-wave Doppler can also be used as a marker of left ventricular efficiency and global function. The dyssynchronous contraction of the left ventricular walls results in inefficient global function, as contraction of the lateral wall may not begin until after contraction of the septal wall. This results in a narrow time window in which all ventricular walls are simultaneously contracting and a pathologically gradual generation of pressure development. Successful resynchronization results in a greater time of mutual wall contraction and therefore improves global efficiency as manifest by a more rapid increase in pressure generation within the left ventricle manifest as an increase in the dP/dt during CRT (Fig. 17.46).

CARDIAC TRANSPLANTATION

Cardiac transplantation is a final option for patients with medically refractory end-stage cardiovascular disease. Although the operative approach to transplantation is relatively straightforward, the evaluation and management of patients after cardiac transplant remain challenging.

After cardiac transplantation, echocardiography plays a number of roles. It is important for the echocardiographer to recognize the anticipated appearance of a heart after cardiac transplantation. In the past, most cardiac transplants were accomplished with atrial wall to atrial wall anastomoses. This results in the postoperative atria being composed of the portions of both the donor and recipient atria and pulmonary veins. This anastomotic approach avoids the potential problem of pulmonary vein stenosis. It results in the appearance of prominent suture lines along the atrial wall and atrial septum, which should not be confused with thrombus or other pathologic mass (Fig. 17.47). Current transplant technique includes a bicaval anastomosis of the right atrium and obvious suture lines may not be present. Often, in an effort to avoid subsequent functional tricuspid regurgitation, a tricuspid annuloplasty may also be performed (Fig. 17.48). This technique also results in an initially smaller right atrial size. A dilated left atrium is noted in the majority of patients. Left atrial enlargement is often most pronounced when viewed from an apical four-chamber view. Other common sequelae of
cardiac transplantation are variable degrees of right ventricular dysfunction. Right ventricular dilation and/or dysfunction after cardiac transplantation is multifactorial and often related to relatively poor preservation of the right ventricle during the harvesting and transplantation, as well as the impact of pre-existing pulmonary hypertension, which is often seen in end-stage heart disease. Because of right ventricular dilation and trauma from repeated right ventricular biopsy, tricuspid regurgitation is common (Fig. 17.49).

An additional complication of repeated right ventricular biopsy is development of a coronary fistula from an intramyocardial vessel into the right ventricular cavity. These are typically of little hemodynamic or clinical significance but should be accurately identified so as to not confuse the continuous coronary to cavity fistula flow for a ventricular septal defect. Figure 17.50 was recorded in a patient with a continuous flow murmur at the apex subsequently identified as a coronary to right ventricular apical fistula.

![FIGURE 17.45. Impact of varying atroventricular (AV) delay during biventricular pacing on left ventricular outflow tract time velocity integral (TVI). Five examples of the left ventricular outflow tract spectral Doppler imaging are presented during intrinsic rhythm and during biventricular pacing at AV delay ranging from 140 to 200 ms. A graphic demonstration of the AV delay versus Doppler tissue imaging is shown in the upper left. Note that the maximal forward flow occurs during biventricular pacing with AV delay of 160 ms in this patient.](image)
FIGURE 17.46. Spectral displays of the mitral regurgitation jet recorded before (A) and immediately after institution of biventricular pacing for resynchronization (B). Note the markedly reduced $\frac{dP}{dt}$ of 425 mm Hg/s in A and a dramatic increase to 857 mm Hg/s immediately following institution of biventricular pacing, indicative of improved overall efficiency of left ventricle pump function.

On rare occasions one may note thrombus forming in the left atrium in a patient who has previously undergone cardiac transplantation (Fig. 17.51). The thrombus occasionally is confined to the recipient portions of the left atrium. This location is presumably related to differentially worse left atrial
wall mechanics in the diseased recipient left atrium compared to the relatively healthy donor left atrium.

After cardiac transplantation, patients are followed for the development of cardiac rejection. Numerous attempts have been made to use echocardiographic parameters to monitor patients for cardiac rejection. Unfortunately, no echocardiographic parameter has been demonstrated to provide sufficient sensitivity and specificity when compared with the standard of cardiac biopsy. Patients with acute severe rejection may have the appearance of left ventricular wall thickening (pseudohypertrophy) and systolic dysfunction. Unfortunately, this appearance is seen only in patients with severe cardiac rejection, when the diagnosis is otherwise not in doubt (Fig. 17.52).

**FIGURE 17.47.** Apical four-chamber view recorded in a patient following cardiac transplantation 10 years prior to this examination. Note the marked dilation of the left atrium and the abnormal contour of the atrial septum. The bright focal echo on the atrial septum (arrow) is the result of the atrial to atrial anastomosis and is due to the atrial suture line.
FIGURE 17.48. Apical four-chamber view recorded in a patient following cardiac transplantation. In this case a bicaval anastomosis was used for the right atrium and a simultaneous tricuspid annuloplasty was performed. Note the relatively normal size of the right atrium and the narrowed tricuspid inflow tract (arrows). Also note the enlarged and elongated left atrium which is the result of an atrial wall to atrial wall anastomosis.
FIGURE 17.49. Apical four-chamber view recorded in a patient 3 years following cardiac transplantation. In the central image note the marked enlargement of both the right and left atria. There is an eccentric tricuspid regurgitation jet of moderate severity. The inset at the upper right is an expanded view of the tricuspid valve in systole in which disruption of the septal leaflet with partial flail (arrow) can be appreciated as the etiology of the tricuspid regurgitation.
Video 17-49 CFD

coming soon

Video 17-49

coming soon
FIGURE 17.50. Apical four-chamber view recorded in a patient 5 years following cardiac transplantation who had undergone multiple percutaneous right ventricular biopsies. Note the disorganized color-flow signal at the apex of the right ventricle (arrows). The inset at the upper left is continuous-wave Doppler through the region of interest, confirming relatively high-velocity continuous flow at the apex consistent with a coronary to right ventricular cavity fistula.

Video 17-50
FIGURE 17.51. Apical four-chamber view recorded 7 years following cardiac transplantation. An incidental finding was a laminar thrombus along the more superior wall of the enlarged left atrium (arrows). This area would be anticipated to be the residual recipient left atrium as opposed to the somewhat smaller donor left atrium. This thrombus resolved with anticoagulant therapy.
FIGURE 17.52. Parasternal long-axis views recorded in a patient following
cardiac transplantation. **A:** Baseline parasternal long-axis view recorded at a time in which the patient had no clinical or other evidence of cardiac rejection. Note the normal tissue signature of the ventricular septum and posterior wall. **B:** Recorded when the patient presented with acute cardiac rejection. Note the normal size of the left ventricle but the increased tissue signature in both the ventricular septum (arrows) and posterior wall, indicative of presumed myocardial edema related to rejection. Also note that both the baseline and rejection echocardiograms are recorded on an identical platform with essentially identical machine settings and transducers. The inset at the upper right is the plot of global longitudinal strain revealing pathologically reduced global longitudinal strain of −9.3% but only mildly reduced left ventricular ejection fraction of 50.2%.

Video 17-52a

Video 17-52b
FIGURE 17.53. Apical four-chamber view recorded at the time of transvenous right ventricular biopsy performed for monitoring of cardiac rejection. Note the position of the bioptome along the apical portion of the right side of the ventricular septum (arrow). Also note the premature ventricular contraction (PVC), which has been provoked by the procedure.

Video 17-53
For chronic, long-term monitoring, other echocardiographic features that have been evaluated have included serial evaluation of left ventricular systolic function, which may decrease with acute severe rejection or after long-standing rejection of lesser severity. Unfortunately, reduction in left ventricular systolic function is an end-stage phenomenon and therefore cannot be relied on for early monitoring of rejection. Patients who have undergone cardiac transplantation have an accelerated rate of coronary atherosclerosis, even if both the donor and recipient are relatively young. This has been referred to as transplant vasculopathy. In these individuals, premature coronary artery disease develops, the sequela of which is acute myocardial infarction. Because the transplanted heart is denervated, these infarcts may be clinically silent. As such, development of congestive heart failure in a patient after cardiac transplantation should result in an echocardiographic search for occult myocardial infarction. Dobutamine stress echocardiography has been employed in many centers to screen for posttransplant coronary artery disease.

Doppler echocardiography has been used in various formats for detection of cardiac rejection. Diastolic function of the transplanted heart, even in the absence of rejection, is often abnormal, and as such, no given Doppler parameter has shown significant discriminatory ability for separation of rejection from nonrejection. More recently, Doppler tissue imaging has been used to evaluate mitral annular or myocardial motion in transplant recipients. At this time, no single or combination of echocardiographic or Doppler parameters should be considered as a reliable indicator of the presence or absence of milder forms of cardiac rejection. As such, endomyocardial biopsy will continue to be necessary.

Percutaneous myocardial biopsy may be performed with ultrasound rather than fluoroscopic guidance. This is typically done by imaging from an apical four-chamber view at which time the bioptome can be seen to enter the right atrium and right ventricle (Fig. 17.53). Echocardiography is used to identify the appropriate site for biopsy (apical septum rather than free wall) and to screen for complications such as the iatrogenic right ventricular perforation and pericardial effusion.
Modern therapy of end-stage cardiovascular disease involves a wide range of medical and mechanical options. One of the newer forms of therapy is a left ventricular assist device (LVAD), which can be used as a temporary bridge to cardiac transplant or used as “destination therapy” in patients for whom transplant is not an option.

Echocardiography plays several roles in patients for whom LVAD therapy is being contemplated or has already been undertaken. First, echocardiography is instrumental in identifying patients who are candidates for LVAD therapy based on decreased left ventricular systolic function and absence of relative contraindication. Other specific features which have relevance for implantation of a ventricular assist device include the presence of apical thrombus, which will necessitate alteration in the surgical procedure for implantation of an apical cannula and the presence of pre-existing aortic insufficiency, which, if moderate or greater, adversely affects efficiency of the LVAD. Other features that have relevance to decision making include the degree of right ventricular dysfunction and the presence of pulmonary hypertension, both of which may reduce the benefit of an LVAD.

An additional factor which impacts the success of LVAD therapy is left ventricular size. By definition, all patients being considered for an LVAD have a dilated left ventricle and severe left ventricular systolic dysfunction. It has become apparent that those with “smaller” left ventricular internal dimensions, typically defined as less than 6 cm, have worse outcomes following LVAD implantation than those with “larger” left ventricular internal dimensions. One presumed mechanism of this is that, with successful LVAD therapy, there is reduction in size of the left ventricular internal dimension which then has an adverse secondary effect on right ventricular geometry as the septum moves away from the right ventricle. This results in widening of the tricuspid annulus and increasing degrees of tricuspid regurgitation, after which right ventricular forward flow may become the limiting factor to delivering blood to the left ventricle for augmentation by the LVAD.

After implantation, the echocardiographer should be cognizant of the anticipated appearance of the visualized portions of the device as well as the appearance of the assisted left ventricle. Typically, the ventricle will remain dilated (but not to the prior degree) and appear to have some contractility. Because the left ventricle is fully unloaded by the assist device, the apparent
mechanical contractility of the ventricle may be misleading with respect to actual intrinsic cardiac function. Typically, the ventricle will contract in conjunction with the electrocardiogram and the mitral valve open and close synchronously. The aortic valve, because of the absence of forward left ventricular flow may remain in a persistently closed position (Fig. 17.54).

A large-bore cannula will be identified at the apex of the left ventricle (Fig. 17.55). It is often difficult to ascertain the presence or absence of the thrombus associated with the cannula because of shadowing. It is important to identify the flow characteristics into the inlet cannula. Because of highly variable position and angulation of the cannula at the apex, multiple windows will be required to identify the optimal imaging plane for interrogation of inflow velocities. With color flow Doppler, one generally sees smooth phasic inflow and, when interrogated with pulsed-wave Doppler, phasic flow into the cannula. The velocity of this flow is highly variable and dependent on pump speed and angle of interrogation. At relatively low pump speeds, the LVAD device is producing lower degrees of cardiac output and lesser degrees of left ventricular unloading in diastole. This results simultaneously in a decrease in the velocity of flow into the inlet cannula in diastole and, if a degree of left ventricular residual contractility is present, an increase in the percentage of aortic valve opening, or other evidence of left ventricular contribution to forward flow. At higher pump speeds, there will be an increase in the inflow Doppler velocity and a decrease in the apparent contribution of left ventricular contractility to forward flow. The outlet cannula is placed in the ascending aorta and delivers nearly continuous flow into the aorta. Because of the partial preload dependency of the device flow has phasic characteristics (Fig. 17.56). Interrogation of the outflow cannula and Doppler interrogation are often best accomplished from the right parasternal window.
FIGURE 17.54. Parasternal long-axis view recorded in a patient after implantation of a left ventricular assist device (LVAD). Note the dilated left ventricle and the cannula in the left ventricular apex (arrow). In the real-time image, note the motion of the left ventricular walls, which is markedly abnormal due to postoperative motion as well as intrinsic dysfunction. Also note the opening and closing of the mitral valve and the persistently closed aortic valve. M-mode echocardiography confirms absence of aortic valve opening with any cardiac cycle. PI, pleural effusion.
If there is a sufficient residual left ventricular contractility, there may be a contribution to forward flow by the diseased left ventricle. This may be manifest as varying degrees of aortic valve opening with systole, including patterns of partial aortic valve opening with each systolic cycle or intermittent aortic valve opening with less than 100% of the cardiac cycles. This is best quantified by M-mode echocardiography as the “opening fraction” (Fig. 17.57). A second measure of residual left ventricular contractility can be derived from the continuous-wave Doppler of an aortic insufficiency jet. As a consequence of continuous flow into the ascending aorta, there is frequent dilation of the proximal aorta resulting in aortic insufficiency of varying degrees. In the majority of instances, this is less than or equal to mild aortic insufficiency but on occasion can reach more significant levels which may be deleterious to LVAD efficiency. Because flow into the ascending aorta is continuous from the LVAD, the aortic insufficiency jet is similar to a continuous jet, often partially interrupted by residual left ventricular contractility which may be insufficient to open the aortic valve or provide forward flow but does result in interruption of the otherwise continuous aortic insufficiency jet (Fig. 17.58). The degree to which the aortic insufficiency jet is interrupted is directly proportional to the amount of pressure generation within the diseased left ventricle.
FIGURE 17.55. Apical view recorded in a patient with a left ventricular assist device. Note the large bore cannula at the left ventricular apex (arrows) and the laminar flow converging toward the inlet cannula. In the lower panel, note the smooth, homogeneous phasic flow into the inlet cannula timed with ventricular systole.
FIGURE 17.56. Transesophageal echocardiogram visualizing the ascending aorta in a patient with a left ventricular assist device. The image was recorded at the level of the inlet cannula (arrows) and a phasic color flow signal is noted in the aorta. The accompanying continuous-wave Doppler reveals a smooth, phasic outflow of the cannula with a peak velocity of approximately 1 m/s.
FIGURE 17.57. M-mode echocardiogram of the aortic valve in a patient with a normally functioning LVAD. Note the varying degrees of opening of the aortic valve. The longer arrows denote cycles in which the aortic valve has near-normal opening, but a rounded closure consistent with low-forward flow. The short arrow depicts a cycle in which there is only partial opening of the aortic valve. For the two cardiac cycles denoted by “x” there is no appreciable opening of the aortic cusps.
FIGURE 17.58. Continuous-wave Doppler recording of aortic insufficiency in a patient with a normally functioning LVAD. Note the continuous nature of the aortic insufficiency. Also note the systolic interruption of the aortic insufficiency jet (*long arrow*) which is the result of residual pressure generation by the left ventricle, sufficient to overcome aortic diastolic pressure. The magnitude of this interruption of aortic insufficiency in systole is an indicator of the degree of residual left ventricular pressure generation.

In view of the above, it should be recognized that there are no “normal” values for the Doppler inflow or outflow velocities but rather they must be placed in context of pump speed and residual contractility and angle of interrogation.

On occasion, an assessment of residual left ventricular contractility is desirable. This assessment can be done by reducing the pump speed of the LVAD (Figs. 17.59 and 17.60). Once device support has been diminished, the degree of residual ventricular contractility can be assessed, typically by observation of aortic valve opening. An aortic valve opening ratio, defined as the percentage of electrocardiographic beats with forward flow sufficient to open the aortic valve, can be followed with varying levels of pump support and is one of the markers of recovery of function. Other parameters that can be followed include forward flow in the left ventricular outflow track (Fig. 17.61) and left ventricular volume. Because of the underlying wall motion
abnormalities, related to the underlying disease and postoperative state as well as variable unloading by the assist device, determination of ejection fraction has been less useful.

FIGURE 17.59. Parasternal long-axis view recorded in a patient with a left
ventricle assist device (LVAD). This image was recorded with full device support. Note the motion of the ventricular walls but the persistently closed aortic valve in the two-dimensional image as well as in the accompanying M-mode echocardiogram. [Video 17-59]
FIGURE 17.60. Parasternal long-axis view recorded in the same patient depicted in Figure 17.59 with the left ventricular assist device (LVAD) deactivated to assess for recovery of function. Note the retained contractility of the left ventricle and the persistent opening of the aortic valve with each systolic beat visualized in the real-time image and in the M-mode echocardiogram.
Several complications may occur in patients with LVADs, many of which can be assessed with echocardiography. A well-described complication is thrombosis of the inlet cannula which results in decreased forward flow and increased energy consumption by the pump (Fig. 17.62). Because of difficult imaging planes and shadowing from the cannula, direct visualization of a thrombus (which may lie within nonvisualized portions of the cannula) may be problematic. Similarly, evaluation of the Doppler inflow velocities must be taken in context of pump speed and previously established baseline values. The combination of reduced (from a previously established baseline) inflow velocities, especially if previously phasic and now “fragmented,” is good indirect evidence of inlet cannula thrombosis (Fig. 17.63). A final maneuver to screen for pump or internal cannula thrombosis is to evaluate inlet cannula flow with the device turned off. As these devices are not valved, if powered off retrograde flow will occur from the higher pressure ascending aorta into the left ventricle (Fig. 17.64). Detection of such retrograde flow is circumstantial evidence against internal thrombus within the device.

An immediate complication of LVAD placement can be kinking of the outflow cannula. This is generally recognized as reduced pump flow with increased energy expenditure and may be manifest as low-velocity disorganized flow in the ascending aorta (Fig. 17.65). Chest CT can easily identify this phenomenon which will require surgical correction. Because forward flow out of the left ventricle is markedly diminished the aortic valve may be persistently closed. This serves as a nidus for thrombus formation.
within the coronary sinuses (Fig. 17.66). Thrombus in this location has recognized embolic potential.
FIGURE 17.61. Spectral Doppler of the left ventricular outflow tract time velocity integral (TVI) recorded in a patient with a left ventricular assist device. A: Recorded shortly after implantation of the left ventricular assist device. Note the markedly reduced TVI of 3.3 cm consistent with minimal contribution of forward flow. B: Recorded 1 month after partial recovery of function, and with full device support reveals an increased TVI of 11.9 cm consistent with a significant contribution of left ventricular contractility to forward flow. C: The right ventricular outflow tract TVI recorded at the same time, revealing a TVI of 15.4 cm consistent with a greater degree of forward flow in the right ventricular outflow tract compared with the left ventricular outflow tract where flow is augmented by the assist device.

FIGURE 17.62. Echocardiogram recorded in a patient with clinical evidence of inlet valve thrombosis based on pump performance and significant hemolysis. The central image is a three-dimensional echocardiogram of the apex of the left ventricle focusing on the inlet cannula (downward-pointing arrow). The leftward-pointing arrow depicts a superimposed mass on the tip of the inlet cannula representing thrombus. The inset at the lower right is a standard two-dimensional echocardiogram recorded in the same patient revealing similar findings.
Video 17-62

coming soon

Video 17-62b

coming soon
FIGURE 17.63. Apical four-chamber view with spectral Doppler in a patient with thrombosed inlet cannula. Note there is no conclusive anatomical evidence of a thrombus burden in or around the cannula. Note the spectral Doppler profile which is low velocity and fragmented (lower left) compared to the baseline Doppler flow profile in the lower right which shows a smooth nonfragmented pulsatile inflow. (Video 17-63)
As previously mentioned, the continuous high flow into the proximal aorta results in variable degrees of dilation of the proximal aorta which may subsequently cause functional aortic regurgitation (Fig. 17.67). This is generally minimal or mild and of no hemodynamic or clinical relevance. On occasion, the degree of aortic insufficiency can reach physiologically deleterious levels and require replacement or oversewing of the aortic valve. When moderate and severe aortic insufficiency occurs, a portion of the flow from the LVAD becomes trapped in a closed loop, as blood is ejected into the ascending aorta as well as back into the left ventricle. This complication will require surgical correction. Because of the atypical continuous nature of the aortic insufficiency standard Doppler parameters of aortic insufficiency severity will systematically underestimate the actual severity.

**FIGURE 17.64.** Parasternal long-axis view recorded in a patient with a left ventricular assist device and suspected device thrombosis. This image was recorded with the device turned off. Because the device is not valved it allows bidirectional flow through the device and if inactivated the higher pressure aortic flow in diastole results allows retrograde flow into the left ventricle. The central image is recorded with color Doppler during diastole. Flowout of the cannula is noted and is confirmed by the spectral Doppler on the upper left and the color Doppler flow image on the lower left. This is indirect evidence that there is no
device thrombosis which would be expected to impede passive retrograde flow.

**FIGURE 17.65.** Transesophageal echocardiogram of the aorta recorded in a patient with a continuous rotary pump left ventricular assist device for which there
was evidence of decreasing forward flow acutely. Notice the fainter and more disorganized flow out of the cannula into the aorta (arrow) and the diminished velocity of flow (<50 cm/s) on the spectral tracing compared with the normal flow profile in Figure 17.56. In this case, the reduction in flow into the aorta was related to kinking of the outlet cannula. 

Video 17-65

A final complication is overly effective pumping of blood from the left ventricle resulting in a “suction event.” In this instance, the left ventricular internal dimension is inappropriately shrunken which increases the tricuspid annulus dimension resulting in severe, functional tricuspid regurgitation. In this situation, right ventricular function becomes the limiting factor in delivering blood to the LVAD and effective cardiac output drops. In severe instances, the left ventricle may become completely collapsed down around the inlet cannula. A suction event is corrected by reducing pump flow and allowing the left ventricle to dilate and refill more appropriately (Figs. 17.68 to 17.70).
FIGURE 17.66. Transesophageal echocardiogram recorded in a longitudinal view of the aorta in a patient with a left ventricular assist device. In the central image note the thrombus in the noncoronary sinus (arrows). The inset at the upper left is a short-axis view of the aorta also demonstrating thrombus filling the noncoronary sinus.

Video 17-66 Sx
FIGURE 17.67. Parasternal long-axis echocardiogram recorded in a patient 6 months status-post implantation of a rotary continuous flow left ventricular assist device. Note the persistently closed aortic valve related to complete support by the device, and in the color flow Doppler, the continuous jet of aortic insufficiency. The origin of the jet is presumed to be dilation of the proximal aorta with malcoaptation of aortic cusps in this case resulting in chronic mild to moderate aortic insufficiency. The spectral Doppler was recorded from the apex of the left ventricle and reveals phasic interruption of the continuous aortic insufficiency jet (arrow), which is a manifestation of residual pressure generation by the left
ventricle.

Video 17-67 CFD

Video 17-67
FIGURE 17.68. Parasternal long-axis and apical four-chamber views recorded in...
a patient shortly after implantation of a rotary flow left ventricle assist device. At the time of this echo, output was reduced and there was evidence of malperfusion. These images were recorded at a high device speed (9,600 rpm) and reveal a completely collapsed left ventricle and a dilated hypokinetic right ventricle. In this instance, the device operating at maximum speed has decompressed the left ventricle to an extent that it has collapsed on itself, further impeding inflow to the device and thereby compromising performance.
FIGURE 17.69. Apical long-axis view recorded in the same patient depicted in Figure 17.68 after decreasing device speed to 8,500 rpm. Notice with less mandatory removal of blood from the left ventricle, the left ventricle has now expanded and device inflow is no longer compromised.

Video 17-69

When evaluating patients with LVADs, it should be recognized that
multiple devices are currently available with more being introduced. Each shares some common attributes in that there is an apically placed inlet cannula and an outlet cannula in the ascending aorta, and although the pump is operating at a continuous manner, there is a degree of pulsatility to the flow related to variable left ventricular contribution. The pump output is determined by pump speed (in RPMs) with different pumps operating over a different range of speeds. As such, there is no equivalence of pump speed and cardiac output across different manufacturer’s devices. At least one newer generation continuous flow pump contains an electromagnetically driven rotary pump incorporated into the inlet cannula. As such, the pump is placed at the left ventricular apex. While the device in this location does not interfere with two-dimensional imaging, it does create significant electrical interference with Doppler signals, the degree of which can be highly variable but in general precludes determination of flow into the inlet cannula because of distortion of the Doppler signals (Fig. 17.71). On occasion, depending on the pump orientation and imaging window, the artifact from the pump can distort Doppler signals in areas remote from the actual pump (Fig. 17.72) which often is obvious as a distorted color Doppler signal but on occasion could result in confusion with a pathologic intracardiac jet such as mitral regurgitation.

FIGURE 17.70. A is an apical four-chamber view recorded in a patient with an ischemic cardiomyopathy and a left ventricular assist device. This image was recorded at a time of clinical deterioration and evidence of malperfusion. Note the marked dilation of the right ventricle and right atrium and the small underfilled left ventricle. The inset at the upper left is a color Doppler demonstrating the presence of severe functional tricuspid regurgitation related to the marked annular dilation of the right ventricle. B was recorded after reduction in LVAD pump speed and reveals an appropriately filled left ventricle and reduction in the size of the right ventricle and right atrium. Note the large apical aneurysm. The inset at the
upper left is a color Doppler demonstrating a significant reduction in the severity of tricuspid regurgitation.
FIGURE 17.71. Transesophageal echocardiogram recorded in a patient with a left ventricular assist device. The pump mechanism of this device is incorporated into the inlet cannula and placed at the apex of the left ventricle and results in substantial destructive interference of Doppler signals. In this instance, a color Doppler interrogation is being performed from a transesophageal echocardiogram. Note the artifactual continuous Doppler signal within the left atrium (horizontal arrows) which could be mistaken for mitral regurgitation.

Video 17-71

HEART FAILURE WITH PRESERVED EJECTION
A substantial number of patients presenting with congestive heart failure have normal or near-normal systolic function and a syndrome of congestive heart failure with preserved ejection fraction (HFpEF). These patients have virtually identical presentations and outcomes compared to patients with dilated cardiomyopathy and reduced ejection fraction. Routine clinical parameters, such as available from the chest x-ray, ECG, and physical examination, are not sufficient to identify patients with this subset of congestive heart failure which may comprise up to 40% of those presenting with clinical congestive heart failure. Echocardiography is instrumental in characterizing patients presenting with congestive heart failure as either having reduced or preserved systolic function. Appropriate characterization of patients with or without significant systolic dysfunction has obvious implications with respect to clinical management.

**FIGURE 17.72.** Parasternal long-axis view recorded in a patient with a dilated cardiomyopathy and a left ventricular assist device resulting in destructive interference of the color Doppler signal. Note the complete filling of the left atrium with color Doppler signal in a pattern physiologically inconsistent with true mitral regurgitation.
Acute myocarditis is typically a viral or postviral process. It results in the acute onset of left ventricular systolic dysfunction, which can range from mild and clinically undetectable to fulminant and fatal over a short course. Although myocarditis often is the sequelae of viral infection, not all patients will have evidence of an antecedent acute febrile, and presumably viral, illness. Clinically, patients with acute viral myocarditis present with tachycardia, hypotension, and shortness of breath. Atrial fibrillation and ventricular arrhythmias are not uncommon. The clinical course of myocarditis is highly variable with variable resolution occurring in a matter of weeks in some patients. A minority of patients will have an acute fulminant and rapidly fatal course. The majority will have a less fulminant course and experience some degree of recovery but often are left with a degree of left ventricular dysfunction.
FIGURE 17.73. Transthoracic echocardiogram recorded in a patient with acute myocarditis and fulminant ventricular failure. A: Parasternal long-axis view. Incidental note is made of a pleural effusion posterior to the left ventricle. The left ventricle has normal geometry and size but can be appreciated to be globally hypokinetic in the real-time image. B: Parasternal short-axis view recorded in same patient. Again note the pleural effusion (Pl). Left ventricular geometry is relatively normal but severe global hypokinesis can be appreciated in the real-time image. There are multiple thrombi present in the right ventricle. Video 17-73a
FIGURE 17.74. Apical four-chamber view recorded in a young patient presenting with acute myocarditis. The central illustration is an apical four-chamber view revealing unremarkable left ventricular size and geometry. The ventricle is noted to have low normal left ventricular systolic function in the real-time image. At the lower right is global longitudinal strain recorded in this patient. Note the calculated ejection fraction is at the lower limits of normal at 52.4% and global longitudinal strain mildly reduced at –16.5%.
Two-dimensional echocardiography should be an early and universally used tool in suspected myocarditis. Acutely, the echocardiographic findings of myocarditis are near-normal ventricular dimensions with a global decrease in systolic function. As with cardiomyopathy, there may be regional variation in the degree to which function is diminished. Subsequent to the initial insult, ventricular dilation may result in varying degrees of mitral or tricuspid regurgitation. In addition, inflammation of the visceral pericardium may result in pericardial effusion, which is typically small. Figures 17.73 and 17.74 were recorded in patients with acute myocarditis. Once the diagnosis has been clinically established, echocardiography should be used for serial follow-up because there will be varying degrees of improvement in left ventricular function. The degree to which recovery of function occurs plays a role in decision making with respect to the type and duration of therapy such as afterload reduction, diuretics, and other modalities. In addition to anatomical imaging, assessment of GLS has occasionally revealed abnormalities in subclinical disease or in patients with relatively preserved ejection fraction (Fig. 17.74).

On occasion, the pattern of involvement in acute myocarditis suggests a specific etiology. Lymphocytic and giant cell myocarditis may present with predominantly anterior wall and right ventricular involvement. Either of these two diagnoses should be considered when myocarditis is associated with a focal distribution of wall motion abnormalities but will require myocardial biopsy for confirmation.
FIGURE 17.75. Cardiac magnetic resonance imaging recorded in a patient presenting with acute myocarditis. This study was obtained in the same patient presented in Figure 17.74. The central image was recorded following gadolinium and demonstrates patchy areas of late gadolinium enhancement both in the mid-myocardium and subepicardial layers of the lateral wall. The inset at the upper left is a T2-weighted image showing increased signal intensity suggestive of myocardial edema.

CMR with gadolinium enhancement plays a valuable role in the diagnosis of acute myocarditis. Because myocarditis is an acute inflammatory process, gadolinium is retained in the interstitium during the acute phase and on delayed imaging appears in a pattern inconsistent with the sequelae of coronary disease. In general, diffuse enhancement is seen which may not be entirely global but does not conform to the pattern one would expect with coronary disease (Fig. 17.75). Other sophisticated CMR techniques such as T2-weighted imaging may allow detection of myocardial edema which is often noted in the acute phase of myocarditis.

Evaluating patients with myocarditis for recovery is done by following left ventricular size and function, including left ventricular volumes and ejection fraction. Other parameters that can be followed include Doppler tissue velocities, which typically are blunted in acute myocarditis but will increase
toward normal with recovery of the underlying process (Fig. 17.76).

**FIGURE 17.76.** Doppler tissue annular velocities recorded in a patient presenting with acute myocarditis in the upper panel and 6 weeks after significant recovery of function. At the time of acute presentation, note the reduced systolic velocity of 8 cm/s, which increased to 13 cm/s following recovery of function.
FIGURE 17.77. These images were recorded in a young patient presenting 5
days after childbirth with congestive heart failure. A was recorded at time of presentation and reveals moderate to severe mitral regurgitation. The inset at the upper left is the two-dimensional image demonstrating a left ventricular internal dimension of 5.6 cm. Left ventricular systolic function can be appreciated to be low normal in the real-time images. B was recorded 1 month later following recovery of all symptoms. In the central image note the left ventricular internal dimension of 4.9 cm and in the real-time image the normal ventricular function. The inset at the upper left confirms absence of mitral regurgitation. In this case a low-grade peripartum cardiomyopathy has resulted in subclinical left ventricular dilation and only mild systolic dysfunction but altered geometry sufficient to cause significant mitral regurgitation.
Other less common causes of transient and reversible left ventricular systolic dysfunction include occasional patients with pheochromocytoma and catecholamine storm. These conditions result in an echocardiographic picture virtually identical to acute myocarditis with global hypokinesis and tachycardia. Surgical resection of the pheochromocytoma and removal of the excess catecholamine state allow recovery of function in the majority of instances. Other rare causes of acute severe systolic dysfunction include acute toxic exposure such as instances of toxic venom from insect bites.

**PERIPARTUM CARDIOMYOPATHY**

Peripartum cardiomyopathy presents with ventricular dilation, systolic dysfunction, and congestive heart failure in the peripartum period. Most women present shortly after childbirth, although a subset will have the initial clinical and echocardiographic presentation late in the third trimester of pregnancy. The precise physiologic mechanism of this entity remains in dispute. At this point, a firm etiology for peripartum cardiomyopathy has not been established. The severity of left ventricular dysfunction ranges from mild to fulminant, and the time course and extent of recovery is variable.
FIGURE 17.78. Apical four-chamber view recorded in a patient with a history of Chagas myocarditis. Note the discrete apical aneurysm (arrows). (Illustration courtesy of Wilson Mathias, Jr, MD, FACC.)

Video 17-78

Echocardiography and Doppler imaging reveal findings identical to those for any other dilated cardiomyopathy. The degree of chamber dilation is dependent on the timing of the examination with respect to onset. Near-normal chamber sizes may be encountered early in the course of the disease.
As with other forms of cardiomyopathy, mitral regurgitation may be encountered as a secondary finding (Fig. 17.77). The diagnosis of peripartum cardiomyopathy is made in the context of a cardiomyopathy first noted in the peripartum period and is in part a diagnosis of exclusion. To date, there have not been specific findings noted on cardiac MRI.

![Figure 17.79](image_url)

**FIGURE 17.79.** Apical four-chamber view recorded in a patient in the chronic phase of confirmed Chagas cardiomyopathy. In this instance, the left ventricle is dilated with severe global systolic dysfunction. The inset at the upper right confirms a marked reduction in global longitudinal strain with mild preservation of the anterior segments. (Illustration courtesy of Wilson Mathias Jr. M.D., FACC.)

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**CHAGAS MYOCARDITIS**

Chagas disease is the sequelae of an infection with *Trypanosoma cruzi*. While focal apical involvement, resulting in a narrow-neck apical aneurysm (Fig. 17.78), has been considered the classic abnormality, the most common presentation of Chagas myocarditis is of global ventricular dysfunction which mimics postviral myocarditis or idiopathic cardiomyopathy (Fig. 17.79). The disease is endemic to South America and rarely, if ever, is encountered in individuals without travel to endemic areas.
Suggested Readings

**GENERAL**


**PHYSIOLOGY AND PROGNOSIS**


**ADVANCED THERAPY**


**Miscellaneous**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Chapter 18
Hypertrophic and Other Cardiomyopathies

OVERVIEW

This chapter deals with hypertrophic and miscellaneous cardiomyopathies, which generally are characterized by increased left ventricular wall thickness and/or infiltration of the myocardium. Unlike dilated cardiomyopathy (Chapter 17) in which signs and symptoms of systolic dysfunction predominate, the clinical presentation of hypertrophic and infiltrative cardiomyopathies is more varied. Symptoms are often mediated by intracavity obstruction, diastolic dysfunction and/or reduced stroke volume related to small cavitary volume. Hypertrophic cardiomyopathies also pose unique clinical challenges with respect to arrhythmias. For patients with infiltrative cardiomyopathy, consideration needs to be given to the presence of an underlying systemic illness. Echocardiography is an essential and appropriate tool in the management of patients with these cardiomyopathies (Table 18.1).

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy is defined by the presence of localized or generalized left ventricular hypertrophy (≥13-mm wall thickness) in the absence of hypertension or other factors likely to result in a pressure overload or an infiltrative state. Hypertrophic cardiomyopathy occurs in sporadic and familial forms with a prevalence estimated at 1 in 500. The genetics of the disease is variable with respect to the specific gene mutation and degree of
More than 1,500 distinct genetic mutations have been reported. The majority involve mutations of either beta-myosin heavy chain or myosin-binding proteins. All forms of hypertrophic cardiomyopathy have in common inappropriate left ventricular hypertrophy. Histologically, myocyte hypertrophy and abnormal orientation are noted. The classic form, obstructive hypertrophic cardiomyopathy, results in dynamic left ventricular outflow tract obstruction and is associated with ventricular arrhythmias and sudden cardiac death. This classic form was previously referred to as idiopathic hypertrophic subaortic stenosis (IHSS), a term no longer in use. There may be substantial variation in phenotypic expression of this disease, even among affected family members carrying the same mutation. In addition to the classic obstructive form, there are well-described forms which are concentric and which may be associated with little or no obstruction. Other forms include the apical variant, more commonly seen in Asian populations. A pattern of hypertrophic cardiomyopathy with isolated midseptal hypertrophy has also been described, as has hypertrophy limited to the inferior, anterior, or lateral walls. Rarely hypertrophic cardiomyopathy may manifest as isolated hypertrophy of the papillary muscles. Less encountered variants of hypertrophic cardiomyopathy include predominant involvement of the right ventricle.

### Table 18.1

**APPROPRIATENESS USE CRITERIA FOR USE OF ECHOCARDIOGRAPHY IN HYPERTROPHIC AND RESTRICTIVE CARDIOMYOPATHY**

<table>
<thead>
<tr>
<th>Appropriate Use Criteria</th>
<th>Score (1–9)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Suspected Cardiac Etiology—General with TTE</strong></td>
<td></td>
</tr>
<tr>
<td>2. Prior testing that is concerning for heart disease or structural abnormality including but not limited to chest x-ray, baseline scout images for stress echocardiogram, ECG, or cardiac biomarkers</td>
<td>A (9)</td>
</tr>
<tr>
<td><strong>Arrhythmias with TTE</strong></td>
<td></td>
</tr>
<tr>
<td>4. Frequent VPCs or exercise-induced VPCs</td>
<td>A (8)</td>
</tr>
<tr>
<td>5. Sustained or nonsustained atrial fibrillation, SVT, or VT</td>
<td>A (9)</td>
</tr>
<tr>
<td><strong>Lightheadedness/Presyncope/Syncope with TTE</strong></td>
<td></td>
</tr>
<tr>
<td>7. Clinical symptoms or signs consistent with a cardiac diagnosis known to cause</td>
<td>A (9)</td>
</tr>
</tbody>
</table>
lightheadedness/presyncope/syncope (including but not limited to aortic stenosis, hypertrophic cardiomyopathy, or HF)

<table>
<thead>
<tr>
<th>Murmur or Click with TTE</th>
</tr>
</thead>
<tbody>
<tr>
<td>34. Initial evaluation when there is a reasonable suspicion of valvular or structural heart disease</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cardiomyopathies with TTE</th>
</tr>
</thead>
<tbody>
<tr>
<td>86. Initial evaluation of known or suspected cardiomyopathy (e.g., restrictive, infiltrative, dilated, hypertrophic, or genetic cardiomyopathy)</td>
</tr>
<tr>
<td>87. Reevaluation of known cardiomyopathy with a change in clinical status or cardiac exam or to guide therapy</td>
</tr>
<tr>
<td>88. Routine surveillance (&lt;1 yr) of known cardiomyopathy without a change in clinical status or cardiac exam</td>
</tr>
<tr>
<td>89. Routine surveillance (≥1 yr) of known cardiomyopathy without a change in clinical status or cardiac exam</td>
</tr>
<tr>
<td>90. Screening evaluation for structure and function in first-degree relatives of a patient with an inherited cardiomyopathy</td>
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</tbody>
</table>


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**FIGURE 18.1.** Parasternal long-axis view recorded in a patient with classic hypertrophic cardiomyopathy. Note the marked thickening of the interventricular...
As an integral aspect of hypertrophic cardiomyopathy, papillary muscle architecture and position within the left ventricle are often abnormal. Abnormal position of the papillary muscles is a contributor to development of dynamic outflow tract obstruction. An additional recently recognized aspect of hypertrophic cardiomyopathy is the concurrent presence of localized areas of myocardial noncompaction in a percentage of cases.

In addition to variable degrees of left ventricular hypertrophy, there may be primary abnormalities of the mitral valve. This includes pathologic elongation and redundancy of a mitral leaflet (usually the anterior leaflet), as well as abnormal chordal attachments. These anatomical abnormalities may be a contributor to both mitral regurgitation and outflow obstruction and may result in failure of myectomy alone to relieve dynamic outflow tract obstruction. When primary anatomical abnormalities of the mitral valve are identified, the surgical approach to hypertrophic cardiomyopathy may be more complex and include complex mitral valve repair and resection of excess mitral valve tissue. Careful attention to mitral valve anatomy is obviously required in evaluating patients with hypertrophic cardiomyopathy.

**Echocardiographic Evaluation of Hypertrophic Cardiomyopathy**
The initial echocardiographic studies of hypertrophic cardiomyopathy used M-mode echocardiography for diagnosis. With this technique, a septal to posterior wall-thickness ratio of 1.3:1 or more was considered evidence of inappropriate septal hypertrophy and was used to establish the diagnosis. This was referred to as asymmetric septal hypertrophy (ASH), a term which often understates the distribution of the pathologic hypertrophy. It should be emphasized that there are a number of other disease states, such as pulmonary hypertension with right ventricular hypertrophy, and inferior wall infarction in the presence of left ventricular hypertrophy that result in a similar septal to posterior wall-thickness ratio. In the normal aging heart there is often hypertrophy and angulation of the proximal anterior septum which may mimic hypertrophic cardiomyopathy if only the septal to posterior wall ratio is considered. Therefore, septal to posterior wall-thickness ratio alone should not be used as a marker of hypertrophic cardiomyopathy.

**FIGURE 18.2.** Parasternal short-axis view recorded in the same patient presented in Figure 18.1. Note the significant left ventricular hypertrophy, maximum in the anterior ventricular septum (long double-headed arrow) with essentially normal wall thickness in the posterior wall (short double-headed arrow) and the gradation of left ventricular hypertrophy circumferentially throughout the
Two-dimensional echocardiography is the primary tool for screening and evaluation of known or suspected hypertrophic cardiomyopathy. The presence, magnitude, and distribution of left ventricular hypertrophy can be determined. When combined with M-mode echocardiography, color flow and spectral Doppler, echocardiography can fully delineate the entire spectrum of hemodynamic abnormalities seen in hypertrophic cardiomyopathy. Figures 18.1 through 18.12 were recorded in patients with hypertrophic cardiomyopathy and demonstrate the variation in degree and distribution of ventricular hypertrophy. Note the thickening of the proximal anterior septum and relative sparing of the posterior wall in Figures 18.1 and 18.2. M-mode echocardiography (Fig. 18.3) suggests isolated septal hypertrophy with otherwise normal wall thickness. Inspection of the short-axis view in Figure 18.2, however, reveals that the hypertrophy is far more generalized than what would have been appreciated from only the parasternal long-axis view or M-mode echocardiography. Commonly, there is a gradation of hypertrophy with maximal involvement in the anterior septum, substantially less involvement in the posterior wall, and intermediate involvement in the lateral wall and inferior septum. This pattern is more common than isolated septal hypertrophy. Figure 18.4 was recorded in a patient with milder hypertrophic cardiomyopathy. Note the proximal septal hypertrophy but relatively preserved dimension of the left ventricular outflow tract.
FIGURE 18.3. Parasternal long-axis derived M-mode echocardiogram recorded in the same patient presented in Figures 18.1 and 18.2. Note the markedly thickened interventricular septum and the relatively normal thickness posterior wall. Also note the systolic anterior motion of the mitral valve (downward-pointing arrows) which opposes the ventricular septum throughout the majority of ventricular systole suggesting obstructive physiology.
FIGURE 18.4. Parasternal long-axis view recorded in a young patient with a family history of hypertrophic cardiomyopathy. In this parasternal long-axis view recorded in diastole, note the selective increase in proximal septal thickness which measures approximately 13 mm compared to thickness throughout the remainder of the myocardium which is uniformly less than 10 mm. The inset shows Doppler recorded through the left ventricular outflow tract confirming absence of a dynamic gradient in this patient who has not developed an obstructive component.

Video 18-4

Figures 18.7 to 18.9 were recorded in patients with more concentric...
hypertrophic cardiomyopathy. Concentric forms of hypertrophic cardiomyopathy are often not obstructive. Symptoms develop in patients with the nonobstructive form due to pathologic stiffness of the left ventricular myocardium and elevated diastolic pressures as well as pathologically small stroke volume. Occasionally, hypertrophic cardiomyopathy is encountered in which the pathologic hypertrophy is confined to the inferior (Fig. 18.10), anterior (Fig. 18.11), lateral or midseptal wall or to the right ventricular wall. Figure 18.12 illustrates hypertrophic cardiomyopathy predominately involving the right ventricle.
FIGURE 18.5. Parasternal long- and short-axis echocardiograms recorded in a patient with hypertrophic cardiomyopathy. Note the increased thickness of the ventricular septum (double-headed arrow) compared to the posterior wall. The short-axis image confirms the disproportionate thickening of the septum versus the posterior wall but also a gradation of hypertrophy present throughout the septum and inferior and lateral walls. The middle inset is an M-mode echocardiogram depicting marked septal hypertrophy and also absence of systolic anterior motion of the mitral valve. The upper right inset is a cardiac magnetic resonance image demonstrating the same pattern of ventricular hypertrophy.
FIGURE 18.6. Apical four-chamber view recorded in the same patient as depicted in Figure 18.5, demonstrating diffuse hypertrophy of the ventricular walls extending to the apex. The small inset is a continuous-wave Doppler image through the left ventricular outflow tract confirming the absence of a dynamic gradient.
FIGURE 18.7. Transthoracic echocardiogram recorded in a young patient with a
newly recognized hypertrophic cardiomyopathy after presenting with an abnormal electrocardiogram. **A:** In the parasternal long-axis view. Note the massive hypertrophy of the anterior septum which measures 3.7 cm and the complete sparing of the posterior wall which measures <1 cm. Note in the real-time image the lack of systolic anterior motion of the mitral valve. In the upper left inset, note the Doppler profile through the left ventricular outflow tract from an apical view confirming absence of any dynamic obstruction. **B:** Parasternal short-axis view recorded in the same patient depicted in (A). Again note the massive hypertrophy of the anterior ventricular septum (*double-headed arrow*) and the sparing of the true posterior wall (*inward-pointing arrows*) with a gradation of hypertrophy in between. Video 18-7
FIGURE 18.8. Parasternal views of a patient with hypertrophic cardiomyopathy and more symmetric hypertrophy. In the parasternal long- and short-axis views note the marked symmetric hypertrophy of virtually all walls of the ventricle as noted by the double-headed arrows in the parasternal long-axis view. The inset is a short-axis cardiac magnetic resonance image also showing the severe, nearly symmetric hypertrophy.
FIGURE 18.9. Apical four-chamber view recorded in the same patient as depicted in Figure 18.8, demonstrating an even greater degree of ventricular hypertrophy extending into the apical aspect of the right ventricular cavity (double-headed arrow).
FIGURE 18.10. Parasternal short-axis image recorded in a patient with hypertrophic cardiomyopathy confined to the proximal inferior wall and inferior septum (double-headed arrow). There was no evidence of dynamic outflow tract obstruction in this patient.
Three-dimensional echocardiography has shown promise for refined definition of the degree and distribution of hypertrophy and assessment of the geometry of the left ventricular outflow tract including the degree to which the proximal septum protrudes into the outflow tract. It is not of proven incremental benefit, and in many adult patients, acquisition of high-quality, three-dimensional data sets for this purpose have been problematic (Fig. 18.13). Because of these limitations, including the paucity of truly incremental information, it has not seen widespread use in evaluation of these patients.

**Role of Cardiac Magnetic Resonance Imaging**

Cardiac magnetic resonance imaging (CMR) plays a valuable role in evaluating patients with known or suspected hypertrophic cardiomyopathy. Because of its intrinsic three-dimensional acquisition, CMR is able to accurately evaluate the global extent and distribution of left ventricular wall hypertrophy more accurately than two- or three-dimensional echocardiography (Fig. 18.14). CMR can identify the abnormal architecture, hypertrophy, and geometry of papillary muscles and also characterize anatomical abnormalities of the mitral valve which may contribute to mitral regurgitation and require attention at the time of surgical myectomy. CMR allows identification and quantification of abnormal flow velocities within the cardiac chambers and can identify mitral regurgitation, dynamic outflow tract obstruction, and calculate dynamic obstructive gradients. An additional
unique feature of CMR is the ability to identify and quantify areas of scar and fibrosis within the hypertrophied myocardium on the basis of delayed gadolinium enhancement (Fig. 18.15). Identification of scar within the hypertrophied myocardium is an independent and incremental marker of patients at greater risk of malignant ventricular arrhythmias.

FIGURE 18.11. Parasternal short-axis view recorded in a patient with hypertrophic cardiomyopathy confined to the anterior and lateral walls (double-headed arrows). [FIGURE]
FIGURE 18.12. **A:** Apical long-axis view recorded in a patient with hypertrophic cardiomyopathy with predominant right ventricular involvement. In the central image, note the marked hypertrophy at the apparent right ventricular apex (arrows). The upper inset is a cardiac computed tomography image from the same patient, better delineating the marked hypertrophy of the apical right ventricular wall (double-headed arrow). The lower inset is a cardiac magnetic resonance image recorded in the same patient, again demonstrating the marked hypertrophy of the right ventricular aspect of the apical ventricular septum (arrows). **B:** Parasternal short-axis view illustrating right ventricular involvement. Note the diffuse hypertrophy of the left ventricle with relatively preserved ventricular septal thickness (double-headed arrow) but the marked obliteration of the right ventricular outflow tract by hypertrophied right ventricular muscle (arrows).
A limitation of CMR is that many patients with a hypertrophic cardiomyopathy may have an implantable defibrillator in place, and as such, CMR, while feasible, is technically more complex and often complicated by artifact. If CMR is to be employed for anatomical assessment in patients with hypertrophic cardiomyopathy, the initial diagnostic study should be performed prior to implantation of a cardiac device to optimize diagnostic accuracy.
FIGURE 18.13. Three-dimensional echocardiogram recorded in a patient with a classic hypertrophic cardiomyopathy. The image displayed is an extracted parasternal long-axis view from a full-volume loop acquired over four cardiac cycles. Note the pathologic thickness of the ventricular septum and the systolic anterior motion of the mitral valve (arrow), which is more apparent in the real-time image.

Video 18-13

Cardiac computed tomography can also be used to identify left ventricular
hypertrophy as seen in hypertrophic cardiomyopathy (Fig. 18.12). It is less able to evaluate outflow tract obstruction or valvular regurgitation and does not provide information regarding myocardial scar which is of significant prognostic relevance.

**Assessment of the Left Ventricular Outflow Tract in Obstructive Cardiomyopathy**

A major component of hypertrophic cardiomyopathy is dynamic left ventricular outflow obstruction. M-mode echocardiography was initially used to document the presence of outflow tract obstruction by detection of systolic anterior motion (SAM) of the mitral valve and aortic valve notching, or abrupt partial closure, in systole (Figs. 18.3, 18.16 to 18.19). SAM of the mitral valve occurs because of the abnormal geometric relationship of papillary muscles and the mitral supporting apparatus combined with hyperdynamic left ventricular contraction of the hypertrophied septum. There is often a component of abnormal elongation and redundancy of the mitral leaflets as well which contributes to obstruction (Fig. 18.20). This results in anterior systolic displacement of varying portions of the mitral valve apparatus. SAM of the mitral valve can be identified on M-mode, transthoracic or transesophageal imaging and should be characterized by the area of the mitral valve having abnormal motion (chordal or leaflet) and the degree and duration of contact with the ventricular septum. Obstruction is more likely to be present when the mitral leaflet makes direct contact with the ventricular septum for 40% or more of the systolic cycle.
FIGURE 18.14. Cardiac magnetic resonance imaging recorded in a young patient with hypertrophic cardiomyopathy and massive hypertrophy of the proximal septum with relative sparing of the remaining walls. The central illustration is a longitudinal view demonstrating the marked septal hypertrophy (double-headed arrow). Note the normal thickness of the left ventricular apex and lateral wall. The inset at the upper right is a short-axis view, again demonstrating massive hypertrophy of the ventricular septum (double-headed arrow) which bulges into the body of the right ventricle. Again note the relative sparing of the remaining walls. The inset at the lower left is a short-axis view of the left ventricle demonstrating significant late gadolinium enhancement within the markedly hypertrophied ventricle (arrows).
FIGURE 18.15. Cardiac magnetic resonance image with late gadolinium enhancement performed in a patient with an apical hypertrophic cardiomyopathy. Note the focal bright echoes at the apex consistent with myocardial fibrosis and scar (arrows).
FIGURE 18.16. Hypertrophic cardiomyopathy with systolic anterior motion of mitral valve depicted in the parasternal long-axis and apical four-chamber views. In each systolic frame, note the motion of the mitral valve into the left ventricle outflow tract (arrows). The M-mode echocardiogram (small inset) also demonstrates systolic anterior motion of the mitral valve (arrow).
FIGURE 18.17. Apical four-chamber view recorded in a patient with obstructive hypertrophic cardiomyopathy. In this end-systolic frame, notice the disproportionate thickening of the ventricular septum, as well as the anterior motion of the mitral valve (arrow) into the left ventricular outflow tract.
The ejection dynamics of obstructive hypertrophic cardiomyopathy allow for relatively normal early left ventricular ejection during which the aortic valve opens normally. Obstruction occurs in mid- to late-systole concurrent with late-phase left ventricular contraction, at which point flow transiently diminishes. The reduction in flow volume results in partial closure of the aortic valve, often with a secondary opening as final ejection occurs. This results in a single notch, or occasionally several discrete high-amplitude notches, of aortic valve motion (Figs. 18.18 and 18.19). The degree to which there is preclosure and notching of the aortic valve is not uniform among the three aortic valve cusps and confers no quantitative information regarding the magnitude of outflow obstruction.
FIGURE 18.18. Transesophageal echocardiogram visualizing the left ventricular outflow tract in a patient with an obstructive hypertrophic cardiomyopathy. In this still frame recorded in early systole, note the anterior motion of the mitral valve which opposes the ventricular septum (arrow). In the real-time image, note the oscillations of the aortic valve which are better depicted in the M-mode (inset).
FIGURE 18.19. M-mode echocardiogram recorded through the aortic valve in a patient with an obstructive hypertrophic cardiomyopathy. Note the midsystolic notching of the aortic valve (arrow) and the otherwise coarse fluttering of the aortic valve during ventricular systole.
FIGURE 18.20. Transesophageal echocardiogram recorded in a patient with an
elongated mitral valve leaflet seen in hypertrophic cardiomyopathy. **A:** Recorded in diastole illustrates the abnormally elongated anterior mitral valve leaflets (arrows). **B:** This frame was recorded in systole. Note the bowing of the anterior leaflet into the left atrium suggestive of mitral prolapse (arrow) and the components of the elongated leaflet prolapsing into the left ventricular outflow tract in systole (left arrow).  

FIGURE 18.21. Parasternal long-axis view (systolic frame) recorded with color
flow Doppler imaging in a patient with hypertrophic cardiomyopathy and systolic anterior motion of the mitral valve. Note the marked turbulence in the left ventricular outflow tract, the relatively narrow width of the turbulent jet at the level of the mitral valve (arrows) and the posteriorly directed mitral regurgitation jet (horizontal arrow).

Video 18-21

Doppler interrogation of the left ventricular outflow tract provides documentation and quantitation of outflow tract obstruction. Dynamic outflow tract obstruction results in turbulence in the outflow tract, which can be detected with color flow Doppler imaging (Fig. 18.21). Pulsed Doppler imaging can be used to track the ejection velocities along the left ventricular outflow tract at which point, when significant dynamic outflow tract obstruction is present, the velocity will exceed the Nyquist limit and aliasing will occur (Fig. 18.22).

Continuous-wave Doppler provides high-fidelity analysis of left ventricular outflow tract ejection dynamics and gradients, but as a standalone technique does not identify the location of obstruction. In hypertrophic cardiomyopathy with SAM of the mitral valve, the level of anatomic obstruction is rarely in question, and continuous-wave Doppler, combined with anatomic assessment, typically allows a full assessment of the location and degree of outflow tract obstruction. Figure 18.23 presents continuous-wave Doppler at rest and with Valsalva from which peak velocities can be recorded without aliasing. There are several characteristics of the continuous-wave Doppler profile related to dynamic outflow tract obstruction. In these figures, note the relatively late peak of the maximal gradient. This has been
described as a “dagger-shaped” profile in distinction to the spectral profile of mitral regurgitation or aortic stenosis (Fig. 18.24), which is more symmetric. The late peaking of the outflow tract gradient is evidence of the dynamic nature of the gradient that develops toward mid- and end-systole rather than being related to fixed obstruction in which the gradient occurs earlier in systole at the time of maximal flow. In obstructive hypertrophic cardiomyopathy, the maximal gradient occurs in late systole, after the majority of left ventricular ejection has occurred. As such, it is not truly obstructive with respect to flow volume, because the majority of the left ventricular stroke volume has been ejected at the time that the gradient develops. Often, there is evidence of presystolic forward flow in the left ventricular outflow tract (Fig. 18.23). This occurs when atrial contraction results in acceleration of flow, which is transmitted to the outflow tract because of a highly noncompliant left ventricle.

FIGURE 18.22. Apical four-chamber view recorded in a patient with an obstructive hypertrophic cardiomyopathy, demonstrating the impact of Doppler region of interest on detection of a dynamic outflow tract gradient. The central frame was recorded in mid systole and systolic anterior motion of the mitral valve is noted (small arrow). The inset at the upper right is a pulsed-wave Doppler
recorded at a mid-septal position demonstrating a nonaliased signal without evidence of an increased velocity. The inset at the lower right is again a pulsed Doppler interrogation with the sample volume at the level of systolic anterior motion of the mitral valve demonstrating aliasing of the signal related to high-velocity dynamic outflow tract obstruction. The inset at the upper left was recorded with continuous-wave Doppler through the left ventricular outflow tract and demonstrates a late-peaking dynamic gradient of 48 mm Hg. Video 18-22
FIGURE 18.23. Continuous-wave Doppler image recorded through the left ventricular outflow tract in a patient with hypertrophic cardiomyopathy. Note the relatively late-peaking systolic gradient with peak pressure gradient of 112 mm Hg at baseline. Also note the prominent presystolic flow in the outflow tract due to atrial contraction against a highly noncompliant and hypertrophied left ventricle. With Valsalva the gradient increases to 172 mm Hg. In the M-mode (bottom panel), note the notching of aortic valve in systole (arrows).

While generally safe in patients with hypertrophic cardiomyopathy,
exercise testing should be performed in a physician-monitored setting by a team familiar with the physiologic and arrhythmic manifestations of hypertrophic cardiomyopathy. In general, exercise testing will be used in an effort to provoke an outflow tract gradient and not for consideration of myocardial ischemia. As such, Doppler interrogation is usually prioritized over wall motion analysis (Fig. 18.25).

It may be clinically useful to attempt to provoke an outflow tract gradient. Physiologically, any maneuver that increases contractility, reduces left ventricular volume, or decreases resistance to left ventricular outflow may unmask an occult gradient. Maneuvers to unmask an outflow tract gradient include exercise, the Valsalva maneuver (Figs. 18.23 and 18.25), amyl nitrite inhalation, isoproterenol infusion, or rapid standing from a squatting position. There are multiple physiologic changes which can result in a secondary alteration of the magnitude of the dynamic left ventricular outflow tract gradient. Following a PVC and a postcompensatory pause it is common to see elevation of the left ventricular outflow tract gradient in the postextrasystolic beat (Fig. 18.26). Other factors which can transiently elevate a dynamic gradient include physiologic stress. Figure 18.27 was recorded in a patient with a stable hypertrophic cardiomyopathy. At the time of presentation with a complex fracture his outflow tract gradient increased dramatically before reverting to baseline with adequate pain control.

Occasionally, one encounters an individual in whom the two-dimensional echocardiographic anatomy is consistent with hypertrophic cardiomyopathy but in whom no evidence of obstruction can be found. It should be emphasized that many of the signs, symptoms, and adverse clinical sequelae of hypertrophic cardiomyopathy are independent of outflow obstruction and are related to diastolic dysfunction, reduced stroke volume, or secondary pulmonary hypertension (Fig. 18.28). Absence of obstruction does not preclude a diagnosis of hypertrophic cardiomyopathy.

Detailed evaluation of myocardial deformation with strain imaging potentially provides a window into the preclinical diagnosis of hypertrophic cardiomyopathy. In general, hypertrophic cardiomyopathy will be associated with abnormal systolic and diastolic strain velocities when compared to normal individuals. Unfortunately, there may be overlap between the strain values noted in hypertrophic cardiomyopathy, hypertensive cardiovascular disease, restrictive cardiomyopathy, and other conditions. This overlap
reduces the independent value of strain analysis. However, if dealing with a patient with no confounding comorbidities (i.e., hypertension, aortic stenosis, etc.), mildly reduced strain in the presence of equivocal hypertrophy in an otherwise healthy young individual with a positive family history for hypertrophic cardiomyopathy may provide circumstantial evidence of a preclinical state (Fig. 18.29). An occasional diagnostic dilemma is separating hypertrophic cardiomyopathy from an infiltrative cardiomyopathy related to Fabry disease, glycogen storage disease, or amyloid. In general, strain values are substantially lower in patients with the infiltrative cardiomyopathies than in hypertrophic cardiomyopathy. Finally in patients with known hypertrophic cardiomyopathy progressively more abnormal strain values (less negative) have been associated with a worse prognosis.
FIGURE 18.24. Comparison of the spectral display of mitral regurgitation (upper panel), dynamic outflow tract obstruction (middle panel), and valvular aortic stenosis (lower panel): The images have been aligned so that for each image the first QRS complex is at roughly the same location on the figure. Note the earlier onset of flow in the mitral regurgitation signal as compared with dynamic obstruction or valvular aortic stenosis. The dynamic outflow tract obstruction profile shows a classically late-peaking dagger profile compared with the symmetric flow profile in mitral regurgitation and valvular aortic stenosis.
Mitral Regurgitation in Hypertrophic Cardiomyopathy

Mitral regurgitation is common in obstructive hypertrophic cardiomyopathy and its etiology is often multifactorial. In some instances, there is a concurrent anatomical abnormality of the mitral leaflets contributing to regurgitation. More often, mitral regurgitation is due to dynamic
malcoaptation that occurs during SAM of the valve (Fig. 18.30). On occasion, one can directly visualize the mid-systolic separation of the mitral leaflets (Fig. 18.31). The severity of mitral regurgitation can range from mild to severe, and mitral regurgitation may be an independent contributor to development of symptoms. The jet typically arises centrally but often takes an eccentric course in the left atrium (Fig. 18.30). It may predominate in mid to late systole during the time of maximal SAM, rather than being holosystolic. Because dynamic outflow tract obstruction also occurs in these individuals, intracavitary left ventricular pressure increases in mid and late systole. This results in an atypical mitral regurgitation contour in which the maximal mitral regurgitation velocity is late rather than early, as is seen in structural mitral regurgitation (Figs. 18.30 and 18.32). Occasionally, confusion arises when looking for an outflow tract gradient if one mistakenly interrogates mitral regurgitation with a late peak and confuses it with the dynamic outflow tract obstruction. The mitral regurgitation signal may have a later onset than the outflow tract flow profile, and frequently the peak velocities are in a supraphysiologic range (Fig. 18.32). When one encounters a hypertrophic cardiomyopathy with mitral regurgitation and a late-peaking velocity of >6 m/s, confusion with the mitral regurgitation jet should be considered. An additional clue to the etiology of the signal is the prolonged nature of the mitral regurgitation signal, which may extend into the isovolumic relaxation period.
FIGURE 18.27. Two-dimensional echocardiogram recorded in a patient with a well-documented obstructive hypertrophic cardiomyopathy. The central illustration is a parasternal long-axis view recorded at end systole demonstrating systolic anterior motion of the mitral valve (arrow). The upper right inset is a continuous-wave Doppler from the apex, through the left ventricular outflow tract at baseline, documenting a left ventricular outflow tract gradient of 16 mm Hg. The inset at the upper left was recorded when the patient subsequently presented with a femoral fracture and was recorded at time of physiologic stress related to pain. Note the increase in the dynamic left ventricular outflow tract gradient to 96 mm Hg related to the increased adrenergic state. This gradient reverted to baseline after control of pain.
FIGURE 18.28. Apical four-chamber and short-axis views in a patient with concentric nonobstructive hypertrophic cardiomyopathy. The small inset is a continuous-wave recording of the tricuspid regurgitation jet revealing a peak tricuspid regurgitation jet-derived gradient of 74 mm Hg consistent with significant secondary pulmonary hypertension.
FIGURE 18.29. Composite four-panel view recorded in a young patient with hypertrophic cardiomyopathy. Note that the global hypertrophy is slightly more prominent in the ventricular septum. In the real-time image note the normal regional and global left ventricular systolic function. The central image is the bull’s-eye plot of global longitudinal strain derived from multiple views. The ejection fraction is calculated at 75.5% and there is normal global longitudinal strain of –25.3%. Note, however, the selective decrease in longitudinal strain in the midportion of the ventricular septum.
FIGURE 18.30. Apical four-chamber view recorded in a patient with an obstructive hypertrophic cardiomyopathy with a combination of dynamic outflow tract obstruction and functional mitral regurgitation. In the central figure, notice the moderate to severe mitral regurgitation with a somewhat eccentric posterolaterally directed jet. The inset at the upper left is the continuous-wave Doppler profile of the mitral regurgitation jet revealing an atypical late-peak systolic jet with a supraphysiologic velocity of 7 m/s corresponding to a pressure gradient of approximately 200 mm Hg, which is the gradient between the left ventricle and the
left atrium rather than the outflow tract gradient. 

Video 18-30

coming soon
FIGURE 18.31. Transesophageal echocardiogram recorded in a patient with obstructive hypertrophic cardiomyopathy and secondary mitral regurgitation. **A:** Recorded in systole and depicts anterior motion of the mitral valve with septal contact. Note that in this mid- to late-systolic frame, the anterior motion of the valve has pulled it away from the posterior leaflet resulting in coaptation failure (arrow) and mitral regurgitation as noted in (**B**) with color flow Doppler imaging. The inset schematizes the pathology which is more apparent in the real-time image. PMV, posterior mitral valve leaflet; SAM, systolic anterior motion.
coming soon

Video 18-31a

coming soon

Video 18-31b
FIGURE 18.32. Continuous-wave Doppler recorded in two patients with hypertrophic cardiomyopathy and concurrent mitral regurgitation. In the upper panel note the unusual contour of the continuous-wave Doppler with a peak velocity of 8.3 m/s, corresponding to a pressure gradient of 276 mm Hg. A gradient of this magnitude would be inconsistent with a true outflow tract gradient.
Note also the unusual contour of the dense late systolic peaking gradient and the suggestion of a fainter early more holosystolic signal (horizontal arrows). The slender late-peaking supraphysiologic gradient is typical of mitral regurgitation in the presence of obstructive hypertrophic cardiomyopathy. The lower panel was recorded in a similar patient during which the interrogation line was shifted slightly from the outflow tract. At the left note the typical late-peaking systolic velocity of approximately 3 m/s consistent with dynamic outflow tract obstruction. As the sample volume shifted slightly note the slender late-peaking peak velocity of 6 m/s which represents mitral regurgitation (horizontal left-pointing arrow). Superimposed on the late-peaking 6 m velocity the dynamic outflow tract gradient can also be faintly appreciated (rightward-pointing arrow).

**Variants of Hypertrophic Cardiomyopathy**

A less frequent form of hypertrophic cardiomyopathy is the isolated apical variant. This form is often associated with deep symmetric T-wave inversion in the anterior precordial leads on the electrocardiogram. Figures 18.33 to 18.36 were recorded in patients with apical hypertrophic cardiomyopathy. The distribution of the hypertrophy can be highly variable and ranges from localized to symmetric and massive in extent. In the “classic form” one notes normal wall thickness at the base of the heart and pathologic thickness toward the apex resulting in a “spade-shaped” left ventricular cavity (Fig. 18.33). This variant of hypertrophic cardiomyopathy is typically not obstructive and is often incidentally encountered in asymptomatic individuals being evaluated for an abnormal electrocardiogram. Figure 18.34 was recorded in a patient with a profound degree of apical and distal hypertrophy with sparing of the base. Notice in this diastolic frame, the plane of the aortic valve and the approximate 2-cm distance of relatively thin septum and posterior wall after which left ventricular hypertrophy results in near cavity obliteration.
FIGURE 18.33. Apical four-chamber view recorded in a patient with apical hypertrophic cardiomyopathy. This image was recorded in systole and reveals obliteration of the apex by hypertrophied myocardium.

Video 18-33

Apical hypertrophic cardiomyopathy can occasionally be overlooked on echocardiography, especially when scanning with low-frequency transducers. In this instance, the low-frequency ultrasound penetrates the relatively less
echogenic myocardium, and only the epicardium is visualized. This results in it being misidentified as the endocardial border. Several additional maneuvers can be used to identify an apical or midventricular hypertrophic cardiomyopathy when it is not apparent on an initial clinical scan. The first is to use relatively shallow focal depths and high-frequency transducers. Additionally, by employing color flow Doppler imaging in the apex, at a relatively low Nyquist limit, one can appreciate the blood pool tissue boundary and often identify a convergence zone near the apex that represents an area of left ventricular narrowing at the apical or midventricular level (Fig. 18.35). Spectral Doppler imaging can be used to confirm a localized apical gradient. Scanning with color tissue Doppler imaging may also allow detection of the more subtle myocardial echoes (Fig. 18.36).

FIGURE 18.34. Parasternal long-axis view recorded in a patient with profound hypertrophy of all distal wall segments. In this end-diastolic frame, notice the massive hypertrophy of the distal three-quarters of the left ventricle. The plane of the aortic valve is noted by the vertical arrow and the double-headed arrow just beneath the aortic valve denotes the extent of normal thickness of the proximal septal myocardium. Note the normal thickness of the proximal posterior wall as well. The remainder of the left ventricle is profoundly hypertrophied with near total cavity obliteration even in this diastolic frame. The full thickness of the
myocardium can be appreciated by the inward-pointing arrows at the margin of the left ventricular cavity.

FIGURE 18.35. Transthoracic echocardiogram recorded in a patient with a hypertrophic cardiomyopathy with predominantly apical hypertrophy and apical displacement of a papillary muscle. A: In this apical long-axis view note the hypertrophy of the entire septum extending into the apex and the apically displaced papillary muscle (downward-pointing arrow). B: Apical long-axis view recorded with color flow Doppler illustrating the narrowed ventricular chamber at the apex (arrow) which is a result of septal hypertrophy as well as the apically displaced and hypertrophied papillary muscle.
FIGURE 18.36. Apical four-chamber view recorded in a patient with an apical variant of hypertrophic cardiomyopathy. **A:** Recorded with standard B-mode imaging and reveals apparent apical hypertrophy. **B:** Recorded with color Doppler tissue imaging in real time. Note the enhanced ability to detect the fainter myocardial echoes related to apical hypertrophy with this technique. The small inset is a cardiac magnetic resonance imaging in a longitudinal view from the same patient also showing isolated apical hypertrophy. 

Contrast echocardiography, using transpulmonary agents to opacify the left ventricle, can also be used to confirm the presence of apical hypertrophic cardiomyopathy. After opacification of the left ventricular cavity with contrast, the true extent of hypertrophy can be appreciated, and the abnormal contour of the left ventricular cavity can be clearly documented (Figs. 18.37
A final indirect observation is that of prominent intramyocardial vasculature seen in the markedly hypertrophied myocardium (Figs. 18.39 and 18.40). This is indirect evidence that the thick walls are constituted by myocardium and not hypertrophied from an infiltrative process.

**Midcavitary Obstruction**

An additional form of hypertrophic cardiomyopathy involves selective hypertrophy and obstruction at the mid-left ventricular level. As with the apical variant, this type of hypertrophic cardiomyopathy may be more difficult to identify because there typically will not be evidence of SAM of the mitral valve or outflow tract turbulence. Because image detail is dependent on lateral resolution, when imaging from the apex, the actual degree of narrowing at the mid-left ventricular level may be underappreciated. Evaluation of the color flow signal in systole may often be the first evidence of midcavitary obstruction (Figs. 18.41 and 18.42). Color flow Doppler imaging will often identify a narrow constricted area of the mid-left ventricular cavity in systole. Continuous-wave Doppler will identify a high-velocity jet at the mid-ventricular level. On occasion, the dynamic nature of both diastolic inflow and ejection flow in mid- and apical-hypertrophic cardiomyopathies results in an unusual and nearly unique flow profile with continuous-wave Doppler directed through the midline of the left ventricle (Fig. 18.42B, inset). This profile represents early high velocities followed by interruption of flow, a second systolic peak, and then diastolic forward flow. This unusual configuration has been referred to as a “lobster claw” flow profile.

In some individuals, this pattern may represent the effects of long-standing hypertension with relatively small left ventricular cavities. It is quite likely that there is a distinct anatomic subtype of hypertrophic cardiomyopathy resulting in this pattern as well. As with the apical variant, intravenous contrast for left ventricular opacification can be used to identify the true boundary of the left ventricular cavity and the degree to which there is narrowing at the mid-left ventricular level.
FIGURE 18.37. A: Apical four-chamber view recorded in a patient with an apical hypertrophic cardiomyopathy, not clearly evident on routine transthoracic imaging. In the real-time image, there is a suggestion of apical hypertrophy (double headed arrow) and cavity obliteration. In B, recorded with left ventricular contrast, note the marked increase in left ventricular apical wall thickness (double-headed white arrows) and the “spade shaped” left ventricular cavity. [Video 18-37a]
Video 18-37b
FIGURE 18.38. Apical four-chamber view recorded in a patient with apical hypertrophic cardiomyopathy. A: Note the vague suggestion of apical hypertrophy. B: Recorded after an intravenous injection of contrast for left ventricular opacification after which the full thickness of the apical myocardium is better appreciated (double-headed arrows).

FIGURE 18.39. Apical four-chamber view recorded with and without color Doppler flow imaging at the apex of the left ventricle. Note the symmetric left ventricular hypertrophy consistent with a hypertrophic cardiomyopathy. In the right panel, recorded with color Doppler flow imaging, note the prominent intramyocardial flow signals consistent with prominent intramyocardial coronary arteries (arrows).
FIGURE 18.40. Apical four-chamber view recorded in a patient with apical hypertrophic cardiomyopathy after injection of contrast for left ventricular opacification. In the central image note the pathologically thickened apical myocardium within which there are multiple large intramyocardial coronary vessels (arrows). The actual anatomical boundary of the left ventricular apex is denoted by the longer downward-pointing arrow. The inset at the upper left is an expanded view of the same area better demonstrating multiple prominent intramyocardial blood vessels. The phasic flow in these vessels can be appreciated in the real-time image.
FIGURE 18.41. Apical long-axis view recorded in a patient with a hypertrophic obstructive cardiomyopathy in whom the maximum level of obstruction is at the mid-left ventricular cavity. Notice in this end-systolic frame the narrowed contour of the systolic ejection flow (arrows). In the upper left inset, note the continuous-wave Doppler recorded through the long axis of the left ventricle with a classic late-peaking systolic gradient of approximately 36 mm Hg. Video 18-41
End-Stage Hypertrophic Cardiomyopathy

One occasionally encounters a patient who presents with inappropriate ventricular hypertrophy (i.e., in the absence of hypertension) and left ventricular systolic dysfunction (Fig. 18.43). This pattern can represent the end stage of a hypertrophic cardiomyopathy in which hyperdynamic left ventricular contraction has “burned out” and the patient is left with global ventricular hypokinesis. Advanced “restrictive” diastolic dysfunction is commonly present as well. Because of the decrease in contractility, SAM and dynamic outflow tract obstruction may no longer be present and the patient presents as having a mildly dilated but hypertrophied cardiomyopathy. The diagnosis of end-stage hypertrophic cardiomyopathy can be made only when previous clinical and echocardiographic evidence has documented a typical hypertrophic cardiomyopathy, but is occasionally suspected when patients present who have no other etiology for the combined hypertrophy and systolic dysfunction.

**FIGURE 18.42.** Apical four-chamber view recorded without (A) and with (B) color Doppler flow imaging in a patient with hypertrophic cardiomyopathy. Notice in A the suggestion of diffuse left ventricular hypertrophy with a relatively normal chamber size. In B, recorded with Doppler color flow imaging at end systole, notice the systolic flow pattern with narrowing at the midlevel of the left ventricle (inward-pointing arrows). The upper left inset is a continuous-wave Doppler recorded through the long axis of the left ventricle revealing an abnormal systolic flow pattern within the cavity of the left ventricle with a double peak resembling a “lobster claw.”
Video 18-42a

Video 18-42b
FIGURE 18.43. Apical four-chamber view recorded in a patient previously with classic obstructive hypertrophic cardiomyopathy which has now progressed to a “restrictive phase.” Note the biatrial enlargement and the global left ventricular systolic dysfunction in the real-time image. The upper inset is the mitral valve inflow pattern, revealing a markedly shortened deceleration time of 106 cm/s consistent with restrictive physiology. The lower inset is the lateral annular Doppler e’ velocity which is pathologically reduced at 5 cm/s. [Video 18-43]
In more advanced stages of hypertrophic cardiomyopathy reduction in global longitudinal strain (GLS) may be noted with little or no reduction in systolic function as measured by ejection fraction (Fig. 18.44). Studies have suggested that significantly more abnormal GLS confers a worsened prognosis compared to patients with preserved strain.

An additional clinical finding in patients with long-standing hypertrophic cardiomyopathy is development of atrial fibrillation. On transesophageal echocardiography these patients not infrequently have significant spontaneous echo contrast within the body of the left atrium and left atrial appendage (Fig. 18.45) and may be at higher risk of clot formation and thromboembolic events.

Additionally, one occasionally encounters patients with long-standing hypertrophic cardiomyopathy and myocardial infarction but without obstructive coronary artery disease. The etiology of the infarct remains conjectural but may be related to compression of the intramyocardial coronary arteries or to functional ischemia in a setting when coronary blood supply has failed to keep in balance with the demands of pathologic hypertrophy. This phenomenon is most often seen in the apical variant of hypertrophic cardiomyopathy. Identification of the small apical aneurysms can be problematic as many of these may be outside of traditional apical imaging planes. Figures 18.46 and 18.47 were recorded in patients with apical hypertrophic myopathy who were incidentally noted to have small discrete apical aneurysms. Figure 18.47 was recorded in a patient in whom the aneurysm was not apparent on routine transthoracic imaging but was clearly identified after injection of intravenous contrast for left ventricular opacification. Note that the contrast-enhanced image also suggests the presence of a small thrombus within the localized aneurysm. Virtually identical anatomy was noted on a cardiac CT, including presence of thrombus.
FIGURE 18.44. Parasternal long-axis view recorded in a patient with a well-established, long-standing hypertrophic cardiomyopathy. Note the hypertrophy of both the ventricular septum and posterior wall and in the real-time image the apparently preserved left ventricular systolic function. The inset at the lower left is a “bull's-eye” plot of global longitudinal strain demonstrating pathologically reduced global strain of –10.2%, with a pattern of uniformly diminished regional strain in all segments.
Screening of Family Members

Once diagnosed, the patient with hypertrophic cardiomyopathy will require lifelong surveillance for the development of progressive gradients and/or mitral regurgitation. Current recommendations are that all first-degree relatives be screened for occult hypertrophic cardiomyopathy. Screening potentially could take place with any imaging modality; however, in view of ease of performance and cost considerations, two-dimensional echocardiography is the standard for routine surveillance. Current recommendations are that all first-degree relatives be screened annually until the age of 18. While no longer accurate, it was previously believed that if hypertrophic cardiomyopathy did not develop by that age, it was unlikely to become manifest later in life. Current recommendations are for screening of first-degree relatives every 3 to 5 years indefinitely after age 18, as there are well-documented instances of hypertrophic cardiomyopathy first becoming manifest up to the seventh decade of life. When a screening echocardiogram reveals an equivocal abnormality, repeat echocardiography at intervals of less than 3 to 5 years may be prudent.
FIGURE 18.45. Transesophageal echocardiogram recorded in a patient with a long-standing hypertrophic cardiomyopathy and subsequent development of atrial fibrillation. Note the dense spontaneous echo contrast in the body of the left atrium. The arrows denote an area of denser mobile spontaneous contrast better seen in the real-time image. The inset at the upper right is a view of the left atrial appendage demonstrating dense spontaneous echo contrast and “sludge” filling the left atrial appendage. [Video 18-45 LAA]
FIGURE 18.46. Apical four-chamber view recorded in a patient with documented apical hypertrophic cardiomyopathy. This image was recorded several years after initial diagnosis and reveals a discrete apical aneurysm (arrows) which is a well-known complication of this form of hypertrophic cardiomyopathy.

Video 18-46

Recent studies in well-defined patient subsets with genetic evidence of hypertrophic cardiomyopathy (abnormal genotype), but no evidence of
pathologic hypertrophy (normal phenotype), have revealed subtle abnormalities of contractility and relaxation, which can be detected with advanced imaging techniques (Fig. 18.29). In these instances, myocardial strain values have been noted to be abnormal compared to normal controls. These abnormalities are not specific for preclinical hypertrophic cardiomyopathy and need to be interpreted in context with family history and/or genetic testing. More recently, abnormalities in left ventricular twist or torsion have also been reported in hypertrophic cardiomyopathy. Whether this observation could also serve as a marker of preclinical disease remains conjectural.

FIGURE 18.47. Apical four-chamber view recorded in a patient with an apical variant hypertrophic cardiomyopathy and a localized small apical aneurysm containing thrombus. The central image is an apical view recorded in systole demonstrating obliteration of the ventricular cavity at the apical third of the left ventricle (inward-pointing arrows). The outer boundary of the left ventricular apical aneurysm is noted by the three small arrows and there is a distinct contrast-filling defect within the aneurysm representing thrombus (long arrow). The inset at the lower right is the noncontrast-enhanced apical image from which the apical aneurysm was not apparent. The inset at the lower left is a cardiac CT of the same patient demonstrating the small apical aneurysm with a filling defect of slightly greater signal intensity than the surrounding myocardium (arrows).
consistent with a small thrombus within the localized aneurysm.

**Table 18.2** CONDITIONS WHICH MAY MIMIC HYPERTROPHIC CARDIOMYOPATHY

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive heart disease with left ventricular hypertrophy</td>
</tr>
<tr>
<td>Left ventricular hypertrophy with inferior myocardial infarction</td>
</tr>
<tr>
<td>Left ventricular hypertrophy with anteroseptal ischemia</td>
</tr>
<tr>
<td>Right ventricular hypertrophy</td>
</tr>
<tr>
<td>Anomalous muscle bundles</td>
</tr>
<tr>
<td>Cardiac amyloid</td>
</tr>
</tbody>
</table>
Conditions Mimicking Hypertrophic Cardiomyopathy

There are several conditions that may mimic the echocardiographic appearance of hypertrophic cardiomyopathy (Table 18.2). Any situation which results in relatively greater septal than posterior wall thickness potentially could be confused for isolated pathologic septal hypertrophy. Occasionally, one encounters a patient with left ventricular hypertrophy related to hypertension and concurrent coronary disease with an inferior myocardial infarction (Fig. 18.48). The reduction in wall thickness of the posterior wall related to myocardial infarction, in conjunction with the hypertension-related hypertrophy of the remaining walls, creates a pattern which mimics classic hypertrophic cardiomyopathy. By noting the akinesis and pathologic scarring of the posterior wall, as well as the clinical scenario, this situation should not be confused for a true hypertrophic cardiomyopathy.
FIGURE 18.48. Parasternal long-axis view recorded in a patient with left ventricular hypertrophy related to hypertension and a prior inferior myocardial infarction. Note the apparent asymmetric septal hypertrophy (double headed arrow), with a septal to posterior wall ratio exceeding 1.3:1. In this instance, the finding is related to pathologic thinning of the posterior wall (inward pointing arrows) combined with hypertension-related hypertrophy of the septum and does not represent hypertrophic cardiomyopathy.
FIGURE 18.49. Transthoracic parasternal (A) and transesophageal longitudinal...
view (B) recorded in a patient with a fixed subvalvular obstruction mimicking hypertrophic cardiomyopathy. **A:** Note the ventricular hypertrophy with a greater degree of septal than posterior wall thickening suggesting the presence of hypertrophic cardiomyopathy. In the small inset, note the continuous-wave Doppler image recorded through the left ventricular outflow tract with a peak velocity of 4 m/s, suggesting an outflow tract gradient of 64 mm Hg. **B:** Note the discrete fibromuscular ridge protruding into left ventricular outflow tract (arrow) which has resulted in a pattern mimicking obstructive hypertrophic cardiomyopathy.

In adult patients with a discrete subvalvular membrane, the actual membrane may be difficult to visualize. In many instances septal hypertrophy progresses to the edge of the membrane and may further obscure it, especially
on transthoracic imaging (Fig. 18.49). Rarely, the septal hypertrophy may contribute a dynamic component to the obstruction. A valuable clue to the presence of a fixed subvalvular membrane is the presence of concurrent aortic insufficiency which is rare in hypertrophic cardiomyopathy but very common in patients with fixed outflow tract obstruction due to a discrete membrane. Transesophageal echocardiography is usually diagnostic. This lesion is further discussed in Chapter 19.

Anatomic variants or other primary diseases may mimic the echocardiographic appearance of hypertrophic cardiomyopathy. One of the more commonly encountered is a prominent muscle bundle or trabeculation lying along the right ventricular side of the anterior ventricular septum (Fig. 18.50). With either M-mode echocardiography or isolated parasternal long-axis imaging, the overlying trabeculation may be confused with an intrinsic portion of the ventricular septum. This results in overestimation of septal thickness, mimicking true septal hypertrophy, which when compared with the normal thickness of the posterior wall leads to the erroneous diagnosis of hypertrophic cardiomyopathy. Similarly, any entity resulting in right ventricular hypertrophy will also result in septal hypertrophy. In this instance, the septal hypertrophy represents the contribution of right ventricular hypertrophy rather than intrinsic disease of the left ventricular septum. Full evaluation of the right ventricle will often reveal evidence of right ventricular hypertrophy and Doppler evidence of right ventricular hypertension.

Additionally, there will not be evidence of dynamic left ventricular outflow tract obstruction. It is quite common to see disproportionate septal hypertrophy meeting the classic criteria of the septal to posterior wall thickness of 1:3:1 in patients with pulmonary hypertension. Recognition of the underlying disease as pulmonary hypertension with right ventricular hypertrophy should avoid confusion with hypertrophic cardiomyopathy. A rare situation which may mimic a hypertrophic cardiomyopathy is in an individual with a spontaneously closed perimembranous ventricular septal defect. The mechanism of closure of perimembranous defect is either by tissue growth with aneurysm formation or by a portion of the tricuspid valve forming a seal over the defect. In either instance, the angulation of the septum may be dramatically altered, and a septal remnant may protrude into the left ventricular outflow tract (Fig. 18.51).
FIGURE 18.50. Normal patient in whom a prominent right-sided trabeculation has resulted in the appearance of septal hypertrophy, mimicking a hypertrophic cardiomyopathy. Careful scrutiny of the septal echoes, however, reveals that the increased thickness is constituted almost entirely by right ventricular trabeculation and does not represent hypertrophy of the left ventricular portion of the septum. The true septal dimension is noted by the longer arrows, whereas the apparent (septal and trabeculation) dimension is noted by the two shorter inward-pointing arrows. [Video 18-50]
Several chronic conditions may also mimic obstructive hypertrophic cardiomyopathy. The first is the so-called acquired hypertrophic cardiomyopathy of the hypertensive elderly (Fig. 18.52). This is a variation of hypertensive cardiovascular disease in which there has been relatively greater hypertrophy of the ventricular septum, which when combined with increased septal angulation seen in the elderly results in outflow tract obstruction. The obstruction occasionally reaches levels similar to those seen in a true genetically based hypertrophic cardiomyopathy. SAM of the mitral valve can result in secondary mitral regurgitation. The diagnosis is established clinically when one encounters the anatomic appearance of an obstructive hypertrophic cardiomyopathy in an elderly patient with long-standing hypertension, but no family history or other features consistent with true hypertrophic cardiomyopathy.

On occasion, cardiac amyloid may also be confused for hypertrophic cardiomyopathy especially when the distribution of amyloid infiltration is not uniform (Fig. 18.53). Doppler tissue imaging may detect markedly reduced annular velocities which, while not specific, may point in the direction of an infiltrative rather than hypertrophic cardiomyopathy. Evaluation of global strain usually reveals a distinct pattern of apical sparing seen in amyloid, but not in hypertrophic cardiomyopathy.

Highly trained athletes may develop a pattern of ventricular hypertrophy, which may include chamber dilation as well as increased wall thickness. The “athlete’s heart” can be confused for hypertrophic cardiomyopathy. This may be problematic as many of these patients may be screened for underlying cardiomyopathy as part of a preparticipation medical clearance. The increased wall thickness in an athlete’s heart is usually ≤13 mm, whereas hypertrophic cardiomyopathy often has substantially greater wall thickness. In the athlete’s heart there will be no evidence of outflow tract obstruction. Recent data also suggest that Doppler tissue profiles will reveal higher systolic and diastolic annular and wall velocities in the athlete’s heart than in hypertrophic cardiomyopathy.
FIGURE 18.51. Parasternal long-axis and off-axis apical views recorded in a patient with a spontaneously closed perimembranous ventricular septal defect, which has resulted in a pattern mimicking hypertrophic cardiomyopathy. **A:** Note the distinct bulge of a discrete portion of the proximal septum into the left ventricle outflow tract (*arrow*). Also note the abnormal angulation between the aorta and septum and the normal thickness of all other ventricular walls. **B:** Recorded from an apical view with inferior angulation, again, revealing the septal bulge into the left ventricular outflow tract. It also reveals a thin, discrete membrane (*arrow*) connecting the right side of the proximal ventricular septum to the aorta, which is a sequela of the spontaneously closed perimembranous VSD. The small inset is a continuous-wave Doppler image revealing a peak gradient through the outflow tract of <2 m/s, which does not have a dynamic configuration.

In patients with intravascular volume depletion, especially if concurrently
on inotropic agents, a hyperdynamic ventricle may be associated with evidence of dynamic outflow tract obstruction. This syndrome is not infrequently encountered in intensive care units where a hypotensive patient with relatively low intravascular volume is placed on inotropic support. Often, there is a history of hypertension, and the relatively low intravascular volumes with augmented contractility result in hyperdynamic motion of the ventricle with an acquired dynamic outflow tract obstruction. The acquired dynamic outflow tract obstruction and SAM of the mitral valve can occasionally result in mitral regurgitation and detection of clinically significant murmurs. The combination of mitral regurgitation, a small ventricular cavity, and outflow tract obstruction leads to progressive hypotension for which an inappropriate increase in inotropic agents is occasionally employed. Detection of a small hyperdynamic ventricle with outflow tract obstruction in this setting is an indication for volume resuscitation and discontinuation or decrease in inotropic support. This issue is discussed further in Chapter 23 dealing with ICU applications.
FIGURE 18.52. Parasternal long-axis view recorded in an elderly hypertensive patient with “hypertensive hypertrophic cardiomyopathy of the elderly.” The combination of septal angulation and disproportionate proximal septal hypertrophy (double headed arrow) results in an anatomic pattern mimicking classic hypertrophic cardiomyopathy. Systolic anterior motion of the mitral valve and varying degrees of outflow tract obstruction may also be encountered.
Occasionally, ischemia or infarction in the left anterior descending coronary artery distribution may mimic obstructive cardiomyopathy. This can occur either as a consequence of an acute coronary syndrome or be provoked at the time of dobutamine stress echocardiography. The distal ischemia results in an exaggerated angulation of the anterior septum which, when combined with hyperdynamic contractility at the base of the heart, results in dynamic outflow tract obstruction with SAM of the mitral valve and, on occasion, mitral regurgitation. Treatment is obviously directed at resolution of the ischemic insult and/or withdrawal of inotropic agents. A similar phenomenon has, on occasion, been noted in the apical ballooning syndrome (Takotsubo).

FIGURE 18.53. Parasternal long-axis view recorded in a patient presenting with diastolic dysfunction and found to have marked ventricular hypertrophy with greater septal than posterior wall thickness. Note the abnormal myocardial texture which is characteristic of amyloid but which may also be seen in hypertrophic

Video 18-52
cardiomyopathy. In the real-time image, notice the absence of systolic anterior motion. The small inset is an annular Doppler tissue image revealing a pathologically reduced annular E/A ratio with an annular E velocity of 4 cm/s, more consistent with an infiltrative than a hypertrophic process.

Video 18-53

**Monitoring Therapy for Hypertrophic Cardiomyopathy**

Obstructive hypertrophic cardiomyopathy often represents a difficult management challenge. Medical therapy directed at decreasing contractility with beta-blockers or calcium channel blockers may provide benefit with respect to resting and exercise outflow tract gradients and result in symptomatic relief. Reduction in outflow tract gradients by surgical myectomy is considered the standard of care for patients who remain symptomatic despite appropriate medical therapy.

Following successful surgical myectomy, there is an immediate reduction in myocardial mass in the proximal anterior septum with instantaneous improvement of abnormal hemodynamics including obstruction (Fig. 18.54). As a result of surgical myectomy, it is not uncommon to sever one or more small intramyocardial coronary arteries. The severed vessel creates a fistula into the left ventricular tract which can be detected with color flow Doppler (Fig. 18.55). On occasion the fistula flow may be confused for aortic insufficiency. Most of these fistulae resolve with time.
FIGURE 18.54. Transthoracic parasternal echocardiograms recorded before (A) and after surgical myectomy for obstructive hypertrophic cardiomyopathy. A: Notice the thickness of the proximal septum and the peak gradient of 100 mm Hg demonstrated on continuous-wave Doppler image. B: Note the abrupt tapering of the proximal anterior septum which is the result of the surgical myectomy and the reduction of the outflow tract gradient to <16 mm Hg. The upper left inset is the premyectomy magnetic resonance image, also revealing proximal septal hypertrophy. 🎥
FIGURE 18.55. Parasternal long-axis view recorded in a patient with a hypertrophic cardiomyopathy shortly after surgical myectomy. In the central image, notice the abnormal geometry of the ventricular septum consistent with the myectomy. Note the color Doppler signal arising from the proximal portion of the ventricular septum consistent with an iatrogenic coronary fistula into the left ventricular outflow tract. The lower left inset is an expanded view of the same area with a slightly different angulation, again depicting the flow from the coronary fistula (shorter arrow). Trivial aortic insufficiency is also present (longer arrow). The upper right inset is a pulse Doppler of the area of the fistula, confirming both systolic and diastolic flow.
Video 18-55

With alcohol septal ablation, a “controlled” myocardial infarction is created in the proximal septum, but there is no immediate reduction in proximal septal mass. Over time, there is scarring and reduction in septal thickness proximally (Fig. 18.56). Typically, alcohol septal reduction results in an immediate decrease in the left ventricular outflow tract gradient with some further improvement noted over time as septal thickness decreases. With either form of septal reduction, there can be subsequent long-term regression in wall thickness in the remaining left ventricular walls related to absence of outflow tract gradient over time (Fig. 18.57). CMR with gadolinium contrast often reveals significant areas of septal scar which may provide a continued substrate for ventricular arrhythmias.

INFLTRATIVE AND RESTRICTIVE CARDIOMYOPATHY

Cardiomyopathy with isolated restrictive physiology may be of several etiologies including infiltrative or idiopathic. In the pure form, systolic function is preserved, and heart failure symptoms are due to diastolic dysfunction. Currently, this clinical syndrome is referred to as “heart failure with preserved ejection fraction” (HFpEF). The classic restrictive cardiomyopathy is infiltrative in nature as typified by cardiac amyloidosis. Although cardiac amyloid is the prototypical disease causing restrictive cardiomyopathy, it is by no means the most common situation in which to identify heart failure with preserved systolic function. A number of diseases including end-stage hypertensive cardiovascular disease, hypertrophic cardiomyopathy, idiopathic restrictive cardiomyopathy, and restrictive heart disease of the elderly may all present with similar pathophysiologic derangement and symptoms of congestive heart failure. Additionally, the late stages of dilated and ischemic cardiomyopathy may be associated with “restrictive physiology” as discussed in Chapters 15 and 17.

The underlying abnormality in restrictive cardiomyopathy (infiltrative or otherwise) is stiffening of the left ventricular myocardium and subsequent congestive heart failure due to elevated ventricular filling pressures. In many
of the restrictive cardiomyopathies, especially later in their course, a component of systolic dysfunction also develops. Detailed evaluation of left ventricular mechanics with newer methods, such as analysis of left ventricular strain, confirm subclinical systolic dysfunction in many cases of restrictive cardiomyopathy. Pathologic stiffening of the left ventricle shifts the left ventricular compliance curve to the left and upward, such that for any given intraventricular volume, left ventricular diastolic pressure is elevated. The elevated diastolic pressure is transmitted to the left atrium and pulmonary veins where it results in pulmonary congestion. In the pure, isolated form of restrictive cardiomyopathy, the internal dimensions of the left and right ventricles are normal, and there is secondary dilation of both atria which may be extreme. This secondary atrial dilation is commonly associated with atrial fibrillation and stasis of blood. Secondary pulmonary hypertension is also common.
FIGURE 18.56. Parasternal long-axis view recorded in a patient before (A) and 4 months after (B) alcohol septal ablation of the proximal septum for hypertrophic cardiomyopathy. Both images are recorded in early systole. A: Note the marked hypertrophy of the proximal septum that narrows the left ventricular outflow tract. B: Note the relative thinning of the proximal septum and substantial widening of the left ventricular outflow tract. Dotted lines denote the original boundary of the hypertrophied proximal septum.

Video 18-56a

Video 18-56b
FIGURE 18.57. Parasternal long-axis view recorded in the same patient depicted in Figure 18.56. This echocardiogram was recorded 10 years following alcohol septal ablation and reveals normal left ventricular internal dimensions, as well as overall normal wall thickness representative of progressive regression of left ventricular hypertrophy following relief of dynamic left ventricular outflow tract obstruction. [Video]
Echocardiographic Evaluation of Restrictive Cardiomyopathy

The echocardiographic hallmark of restrictive cardiomyopathy is normal ventricular size and systolic function with evidence of pathologic diastolic stiffening. In the majority of cases, diastolic dysfunction is often accompanied by increased wall thickness, whether due to left ventricular hypertrophy, as in end-stage hypertensive cardiovascular disease, or infiltration, as typified by cardiac amyloid. Biatrial enlargement is ubiquitous. Varying degrees of concurrent systolic dysfunction may be noted in more advanced cases and secondary pulmonary hypertension is not uncommon.

Cardiac Amyloidosis

Figures 18.53 and 18.58 to 18.62 were recorded in patients with cardiac amyloid and illustrate the ventricular hypertrophy with abnormal myocardial texture. Abnormal myocardial texture was initially described using early generation scanners, on which the myocardium was described as diffusely bright with a finely “speckled” appearance. When scanning with modern scanners in tissue harmonic mode, myocardial intensity is enhanced and the appearance of a bright myocardial signature is not specific for amyloid infiltration. It is often useful to scan in both harmonic and fundamental modes if there is a question of abnormal myocardial texture. In addition to cardiac amyloid, hypertrophic cardiomyopathy and hypertrophy seen in end-stage renal disease often have a similar tissue appearance. Findings in cardiac amyloid vary with its severity and duration. In early phases, abnormal texture may be a subtle finding and Doppler inflow patterns may suggest delayed relaxation rather than a restrictive pattern (Fig. 18.63). Patients with cardiac amyloidosis often have pathologically low voltage on ECG. The combination of low-ECG voltage in the presence of apparent left ventricular hypertrophy is often an early clue to the presence of cardiac amyloid.

Analysis of ventricular dynamics with advanced techniques, such as speckle tracking for strain analysis, play a significant role in the evaluation of patients with restrictive and infiltrative cardiomyopathies and may enhance specificity for the diagnosis of cardiac amyloid. In general, GLS is reduced in restrictive cardiomyopathies and particularly in cardiac amyloid. Reduction in strain parameters has been shown to precede other echocardiographic
evidence of cardiac involvement in systemic amyloidosis. A pattern of apical sparing of GLS has been reported as a specific marker of cardiac amyloid when compared to patients with hypertrophy related to hypertensive cardiovascular disease, aortic stenosis, or hypertrophic cardiomyopathy (Figs. 18.61 and 18.62). Additionally, lower values of GLS confer a substantially worse prognosis with respect to mortality.

**Restrictive Cardiomyopathy**

Figure 18.64 was recorded in an elderly patient with an idiopathic restrictive cardiomyopathy. In this instance, mild left ventricular hypertrophy without abnormal texture is present and there is marked dilation of both atria. Additional features may include secondary pulmonary hypertension and atrial fibrillation. In some instances in which an idiopathic restrictive cardiomyopathy has been detected in a relatively young patient, the underlying substrate may have been a previously unrecognized hypertrophic cardiomyopathy.
Doppler evaluation is essential to confirm the diagnosis of restrictive cardiomyopathy. Early in the course of an infiltrative process such as amyloid, mitral inflow shows a pattern of delayed relaxation. In advanced restrictive myopathy, one classically encounters a pathologically elevated E/A ratio of mitral valve inflow (typically ≥2.0) with a shortened deceleration time (typically <160 ms) and reduced annular diastolic velocities (Fig. 18.65). In distinction to constrictive pericarditis, there is less respiratory
variation in E-wave velocity. Concurrent with abnormalities in mitral valve inflow, pulmonary vein flow may reveal a blunted systolic forward flow and an accentuated retrograde A-wave. Color M-mode imaging of mitral valve inflow can also be used to document the abnormal filling pattern in restrictive cardiomyopathy. Evaluation of hepatic vein flow also provides valuable clues as to the presence of restrictive cardiomyopathy. In the presence of a restrictive cardiomyopathy there is expiratory dependent reversal of flow in the hepatic veins (Fig. 18.66) which is a distinguishing feature between constrictive and restrictive physiology.
FIGURE 18.59. Ancillary imaging recorded in the same patient as depicted in Figure 18.58. The top panel is a mitral inflow revealing absence of a distinct A wave and a short deceleration time of 100 ms. The second panel was recorded from the right ventricular outflow tract and depicts the continuous-wave spectral profile of pulmonic insufficiency. Note the notching in the pulmonic insufficiency display timed with right atrial contraction. Because of atrial contraction against a noncompliant right ventricle, pulmonary insufficiency is interrupted in late diastole resulting in this pattern. The third panel is a lateral mitral annulus tissue Doppler tissue imaging showing low systolic and diastolic velocities with an annular e’ of approximately 10 cm/s. The bottom panel is a color M-mode Doppler image also revealing abbreviated diastolic inflow.
Restrictive cardiomyopathy is often a global process, and similar pathology can be noted in the right ventricle, including varying degrees of hypertrophy and infiltration and abnormalities of tricuspid inflow and hepatic vein flow, all of which mirror to those seen on the left side.

A not uncommon clinical dilemma involves distinguishing a restrictive cardiomyopathy from constrictive pericarditis. Both entities often present with evidence of low-output and congestive heart failure with preserved ventricular function. Signs and symptoms of right heart failure with marked volume overload often predominate. If classic anatomic findings are present, there should be little confusion differentiating between constrictive pericarditis and restrictive cardiomyopathy. As such, when a patient presents with symmetrically hypertrophied walls with abnormal myocardial texture, low-ECG voltage, bialtrial enlargement, and a restrictive mitral inflow pattern, the diagnosis of cardiac amyloid is reasonably assured, and constrictive pericarditis is less of a clinical consideration. This issue is discussed in more detail in Chapter 9 dealing with pericardial diseases.

**Endocardial Fibroelastosis and Hypereosinophilic Syndrome**

Endocardial fibroelastosis occurs in several forms, including congenital and acquired tropical and nontropical varieties. Endocardial fibroelastosis is also associated with the hypereosinophilic syndrome which results in inflammation of the endocardium and subsequent creation of a thick endocardial layer. Because of the inflammatory process, there is overlying thrombus and the appearance of an obliterative apical process (Fig. 18.67). The process can involve both ventricles. Both global systolic dysfunction and variable degrees of diastolic dysfunction occur. In late stages, this entity has the appearance of a dilated cardiomyopathy with restrictive physiology. Occasionally seen in hypereosinophilia syndrome is selective involvement of the posterior mitral valve leaflet, resulting in mitral regurgitation. (See Chapter 23 for further discussion of this topic.)
FIGURE 18.60. Apical four-chamber view recorded in a patient with cardiac amyloidosis. Note the biventricular hypertrophy with abnormal myocardial texture and the marked biatrial enlargement. On the mitral inflow signal, note the markedly elevated E/A ratio of approximately 4.0 with a short deceleration time. The inset in the upper right is an annular Doppler tissue imaging display revealing an annular e’ of <10 cm/sec.

Video 18-60

Muscular Dystrophy/Glycogen Storage Disease
Several of the muscular dystrophies as well as Friedrich ataxia may have cardiac involvement that may mimic hypertrophic or dilated cardiomyopathy. Friedrich ataxia is an autosomal recessive disorder with progressive neurologic involvement. Initial manifestations of ataxia typically appear in late adolescence or early adulthood. Cardiac manifestations include left ventricular hypertrophy mimicking hypertrophic cardiomyopathy without obstruction (Fig. 18.68).

**FIGURE 18.61.** Apical four-chamber view with composite Doppler and longitudinal strain data from a patient with documented cardiac amyloidosis. In the apical four-chamber view, note the biatrial enlargement, the dilated right ventricle, and the normal size and geometry of the left ventricle with left ventricular hypertrophy. The upper right inset is mitral valve inflow revealing a short deceleration time and absent A wave. The lower right panel is a lateral annular Doppler tissue velocity revealing a pathologically reduced $e’<4$ cm/s. In the upper left, note the bull’s-eye pattern of global longitudinal strain derived from apical four-, two-, and long-axis views confirming apical sparing consistent with cardiac amyloidosis.
coming soon

Video 18-61
FIGURE 18.62. Transthoracic apical images recorded in a patient with cardiac amyloidosis. A is an apical long-axis view illustrating left ventricular hypertrophy with pathologically increased echo intensity of the myocardium. Also note the left atrial dilation. B is an apical four-chamber view recorded in the same patient at the time of acquisition of data for longitudinal strain. Note the biatrial enlargement and the normal left ventricular internal dimensions. The global longitudinal strain reveals preservation of apical strain with diminution in the basal segments as reflected in the bull’s-eye plot.

In addition to muscular dystrophies, there are a number of heritable metabolic disorders which are associated with cardiovascular disease, among them Fabry disease. Fabry disease has been associated with a variety of abnormalities which can be detected by echocardiography including patterns
of both concentric and asymmetric hypertrophy mimicking hypertrophic cardiomyopathy, as well as regional wall motion abnormalities which may either be typically or atypically located for a coronary disease distribution. Figures 18.69 through 18.71 were recorded in patients with documented Fabry disease. In Figure 18.70, note the overall pattern of left ventricular hypertrophy with the regional wall motion abnormality and thinning of the inferoposterior wall suggestive of inferoposterior myocardial infarction with concurrent left ventricular hypertrophy. This echocardiogram was recorded in a young individual free of coronary artery disease. Various patterns of left ventricular hypertrophy have been described in Fabry disease, including those mimicking obstructive and nonobstructive hypertrophic cardiomyopathy, as well as left ventricular hypertrophy with coronary artery disease.

**FIGURE 18.63.** Pulsed Doppler imaging of mitral inflow (A) and annular Doppler tissue imaging (DTI) (B) recorded in a patient with cardiac amyloid, revealing
grade 1 diastolic dysfunction. Note the reduced mitral E/A ratio, which is paralleled by the Doppler tissue imaging of annular motion in diastole.

A LAMP2 gene mutation results in a form of lysosomal storage disease resulting in an infiltrative cardiomyopathy which may mimic hypertrophic cardiomyopathy or cardiac amyloid. Electrocardiographic voltage is preserved or increased. The same mutation may result in the appearance of a dilated cardiomyopathy. Clinically it is more severe in males than in females and is associated with musculoskeletal and developmental abnormalities. The degree of wall thickening is often marked and is associated with abnormal myocardial texture (Fig. 18.72). CMR can demonstrate the extent of wall thickening and late gadolinium enhancement (Fig. 18.73).

**FIGURE 18.64.** Apical four-chamber view recorded in an elderly patient with an idiopathic restrictive cardiomyopathy. Notice the marked biatrial enlargement. In the real-time image, note the normal systolic function of the left ventricle. The upper left inset is the transmitral flow in this patient with atrial fibrillation. Deceleration time is shortened at 133 ms. The lower right inset was recorded from a pulmonary vein. Notice the blunted antegrade systolic flow (arrow).
coming soon

Video 18-64
FIGURE 18.65. Pulsed Doppler imaging of mitral inflow (A) and annular Doppler tissue imaging (B) recorded in a patient with restrictive cardiomyopathy and evidence of significant diastolic dysfunction. A: Note the mitral E/A ratio of approximately 3.5 and the short deceleration time, typical of a restrictive process. B: Note the marked reduction in annular e’ velocity. In this example, the ratio of E/e’ is more than 25, indicative of a marked elevation in left atrial pressure.
FIGURE 18.66. Hepatic vein pulsed Doppler recordings from two patients with documented restrictive cardiomyopathy showing the variability in inflow patterns that can be seen. A: Note the loss of smooth multiphasic flow out of the hepatic vein and the distinct inspiratory reversal of flow (downward-pointing arrow). B: Recorded in a patient with cardiac amyloid and abnormal hepatic vein flow. Note the lack of any respiratory variation and the forward flow out of the hepatic vein, which is confined exclusively to the systolic portion of the cardiac cycle. Note that there is little or no flow during diastole (D) (double-headed arrow).
FIGURE 18.67. Apical four-chamber view recorded in a patient with hypereosinophilic syndrome and endocardial fibrosis. Note the homogeneous mass obliterating the left ventricular apex (arrows), which represents a combination of inflammatory material and superimposed thrombus. [Video 18-67]
FIGURE 18.68. Transthoracic echocardiogram recorded in a patient with Friedrich ataxia and marked left ventricular hypertrophy, mimicking a hypertrophic cardiomyopathy. A: Parasternal long-axis view demonstrating abnormal
myocardial texture and left ventricular hypertrophy. In the real-time image note the normal ventricular function and the absence of systolic anterior motion to suggest outflow tract obstruction. The side inset at the upper right is a parasternal short-axis view from the same patient demonstrating symmetric left ventricular wall thickening with abnormal texture and a prominent papillary muscle (arrow). B: Apical long-axis view recorded in the same patient. In the real-time image notice the preserved left ventricular systolic function and the absence of systolic anterior motion of the mitral valve. The inset at the lower left is a continuous-wave Doppler through the left ventricular outflow tract confirming the absence of any dynamic obstruction. The inset at the upper right is the bull's-eye plot of global longitudinal strain demonstrating a mild apical sparing and overall reduction of global longitudinal strain which measured –12.4%. [Video]
FIGURE 18.69. Parasternal long-axis view recorded in a patient with Fabry disease. Note the left ventricular hypertrophy and the selective thinning and akinesia of the proximal posterior wall (arrows), better appreciated in the real-time image.
FIGURE 18.70. Cardiac magnetic resonance imaging recorded in the same patient presented in Figure 18.69. This cardiac magnetic resonance imaging in a
long-axis equivalent view was recorded 2 years prior to the echocardiographic images presented previously and confirms symmetrically increased left ventricular wall thickness (double-headed arrows). At this time, the focal wall motion abnormality in the inferoposterior wall was not yet present.

**FIGURE 18.71.** An apical four-chamber view is presented in a patient with Fabry cardiomyopathy depicting left ventricular hypertrophy, mimicking hypertrophic cardiomyopathy. In the upper right inset, note the pathologically reduced annular e' and, in the upper left inset, the bull's-eye plot of global longitudinal strain revealing globally reduced longitudinal strain.
FIGURE 18.72. **A:** Parasternal long-axis view recorded in a young patient with a LAMP2 mutation. Note the massive left ventricular hypertrophy with markedly abnormal myocardial texture (double headed arrows). In **B**, again note the marked symmetric left ventricular hypertrophy with a pathologically reduced left ventricular end-diastolic volume.

coming soon
FIGURE 18.73. Cardiac magnetic resonance image recorded in the same patient presented in Figure 18.72. Note the markedly thickened left ventricular wall thickness in virtually all segments. In the side insets, note the marked degree of late gadolinium enhancement throughout virtually all segments of the left ventricular myocardium consistent with a diffuse infiltrative process.
FIGURE 18.74. Apical four-chamber view recorded in a patient with Uhl anomaly. Note the unusual, “banana-shaped” left ventricle which appears to prolapse over the right ventricle. Note the globular right ventricle. This echocardiogram was recorded in a patient with Uhl anomaly with absence of functioning myocardium at the right ventricular apex. Also see Figures 18.75 and 18.76.

Video 18-74

Uhl Disease
Uhl disease is a heritable or congenital abnormality of the right ventricular myocardium in which the typical myocardial structure is lost and replaced with fatty infiltration. Synonyms for this phenomenon include idiopathic right ventricular dysplasia. There may be overlap between this syndrome and that of arrhythmogenic right ventricular dysplasia. From an echocardiographic perspective, one can identify a dilated, hypokinetic right ventricle, often with abnormal geometry. Because the supporting architecture of the right ventricle is lost, there may be abnormal left ventricular geometry as well (Figs. 18.74 through 18.76). CMR as well as cardiac computed tomography can also reliably identify the abnormal right ventricular geometry. CMR can also identify regions of fatty infiltration of the myocardium.
**FIGURE 18.75.** Cardiac CT with contrast recorded in a patient with Uhl anomaly. Note the unusual, “banana-shaped” left ventricle and the globular right ventricle. At the base of the right ventricle (outward-pointing arrows), note the normal tissue character of the right ventricular myocardium. At the more apical half of the right ventricular free wall (downward-pointing arrows), note the absence of myocardial tissue indicative of fatty replacement.

**FIGURE 18.76.** Cardiac magnetic resonance imaging performed in a patient with Uhl anomaly. This MRI corresponds to the echocardiogram presented in Figure 18.74 and the CT depicted in Figure 18.75. Note the unusual “banana-shaped” left ventricle and the globular right ventricle with absent myocardial tissue toward the apex.
Suggested Readings

GENERAL


HYPERTROPHIC CARDIOMYOPATHY


**INFLITRATIVE AND RESTRICTIVE CARDIOMYOPATHY**


**CARDIAC AMYLOID**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Congenital heart diseases (CHDs) are broadly defined as those cardiac anomalies that are present at birth. By their very nature, such defects have their origin in embryonic development. Most congenital cardiac lesions constitute gross structural abnormalities with a spectrum of associated hemodynamic derangements. It is not surprising that the various echocardiographic techniques are ideally suited to the study of patients with CHD. Perhaps nowhere in cardiology have these methods played a more vital role in diagnosis and management. More recently, cardiac MRI has assumed an increasingly important role in this area and especially for the comprehensive evaluation of adult patients following surgical repair or palliation. This group represents a growing proportion of all patients with CHD and MRI provides several unique advantages for their assessment.

The echocardiographic approach to patients with congenital heart lesions differs substantially from that used to evaluate other forms of cardiac disease. Guidelines for the use of echocardiography in CHD have been published and appropriate use criteria for this growing patient population are provided in Table 19.1. Imaging in children has both advantages and disadvantages compared with adults. The smaller patient size permits the use of higher-frequency transducers, thereby enhancing image quality. The presence of less heavily calcified bone and the absence of hyperinflated lungs in most children increase the available acoustic windows and generally contribute to improved image quality. Unfortunately, small patients also create practical problems for image acquisition. Children are more likely to be uncooperative and may have other malformations (such as a chest deformity) that complicate imaging.

In the near future, there will be more adults with CHD than children. A
formal subspecialty now exists devoted to the care of these patients and dedicated adult congenital heart clinics are present in many tertiary medical centers. Older patients with CHD present an entirely different array of challenges to the clinician and echocardiographer. The decision to intervene in these patients frequently hinges on the adequacy of previous interventions and the presence and severity of pulmonary vascular disease. In patients who have undergone surgery, an accurate assessment may be difficult and other imaging modalities, especially MRI, are often utilized for the evaluation of these individuals. When details of the clinical history are unavailable, the cardiac imager is often called on to determine which surgical procedures have been performed. The options for further intervention often depend on the imaging results. As the patient with CHD ages, the superimposition of other medical conditions (such as hypertension or coronary disease) further complicates evaluation and management. Both image acquisition and interpretation can be challenging and time-consuming. The diversity and complexity of congenital cardiac malformations obviate even the most basic assumptions regarding chamber orientation and great vessel relationships. Therefore, the initial evaluation of the patient with suspected CHD mandates a thorough and systematic approach, often using additional views beyond those obtained during the standard examination.

This chapter focuses on the role of echocardiography in the adolescent and adult with CHD. It is not intended as an exhaustive description of all forms of CHD. Lesions that are seen more commonly in adult patients are emphasized, whereas those considered less relevant are covered only superficially.

**THE ECHOCARDIOGRAPHIC EXAMINATION: A SEGMENTAL APPROACH TO ANATOMY**

The initial echocardiographic examination of the patient with suspected CHD requires a sequential and systematic approach to cardiac anatomy (Table 19.2). Such a method is necessary to detect cardiac malpositions and to diagnose complex CHD. The first step in this sequential approach is to determine atrial situs and to assess the venous inflow patterns to the atria. Then, atrioventricular connections are defined and ventricular morphology and position are determined. Finally, ventriculoarterial relationships are
evaluated. In most cases, this approach permits the identification of even the most complex forms of CHD.

<table>
<thead>
<tr>
<th>Table 19.1</th>
<th>APPROPRIATE USE CRITERIA FOR ADULT CONGENITAL HEART DISEASE USING TTE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indication</td>
<td>Score (1–9)</td>
</tr>
<tr>
<td>92. Initial evaluation of known or suspected adult congenital heart disease</td>
<td>A (9)</td>
</tr>
<tr>
<td>93. Known adult congenital heart disease with a change in clinical status or cardiac examination</td>
<td>A (9)</td>
</tr>
<tr>
<td>94. Reevaluation to guide therapy in known adult congenital heart disease</td>
<td>A (9)</td>
</tr>
</tbody>
</table>
| 95. Routine surveillance (≤2 yrs) of adult congenital heart disease following complete repair  
  • Without residual structural or hemodynamic abnormality  
  • Without a change in clinical status or cardiac examination | I (3)       |
| 96. Routine surveillance (≥2 yrs) of adult congenital heart disease following complete repair  
  • Without residual structural or hemodynamic abnormality  
  • Without a change in clinical status or cardiac examination | U (6)       |
| 97. Routine surveillance (<1 yr) of adult congenital heart disease following incomplete or palliative repair  
  • With residual structural or hemodynamic abnormality  
  • Without a change in clinical status or cardiac examination | U (5)       |
| 98. Routine surveillance (≥1 yr) of adult congenital heart disease following incomplete or palliative repair  
  • With residual structural or hemodynamic abnormality  
  • Without a change in clinical status or cardiac examination | A (8)       |


| Table 19.2 | A SEGMENTAL APPROACH TO CARDIAC SITU AND |


Cardiac Situs

Determination of atrial situs is best accomplished by using the subcostal views. In atrial situs solitus, the normal situation, the morphologic right atrium is to the right and the morphologic left atrium is to the left. In situs inversus, the opposite occurs, creating a mirror image effect. Atrial and visceral situs are almost always concordant. Thus, a right-sided liver and left-sided stomach are usually associated with atrial situs solitus. In the rare cases when atrial and abdominal situs are discordant, the likelihood of complex congenital lesions is high. By using two-dimensional echocardiography, the location and morphology of the atria can be determined. The morphologic right atrium always contains the eustachian valve, and its appendage is shorter and broader than that of the left atrium. The left atrium lacks the eustachian valve and has a more rounded shape than the right atrium. The left atrial appendage is long and thin and has a narrower atrial junction than that of the right atrial appendage.
Although venous inflow does not define atrial morphology, the patterns of systemic and pulmonary venous return are helpful in determining situs. This spatial relationship is best evaluated using a transverse imaging plane through the upper abdomen. Normally, the abdominal aorta lies to the left and the inferior vena cava lies to the right of the spine. Compared with the vena cava, the aorta appears larger, more rounded, and more pulsatile. When in doubt, color flow imaging can be used to differentiate between the two vessels by demonstrating higher-velocity and primarily systolic flow in the aorta (Fig. 19.1). The opposite spatial relationship is characteristic of situs inversus. By tracing the course of the inferior vena cava and hepatic veins in the subcostal long-axis view, the right atrium generally can be identified in its usual position anterior and to the right of the left atrium (Fig. 19.2).

The entrance of the pulmonary veins into the left atrium can sometimes be
visualized using the apical and suprasternal window (Fig. 19.3). Color Doppler imaging is particularly helpful in identifying the pulmonary veins as they enter the left atrium. In adults, it is usually impossible to record the insertion of all four pulmonary veins using transthoracic echocardiography. With transesophageal echocardiography, however, the pulmonary venous drainage pattern can be defined more precisely. Because of the possibility of anomalous pulmonary venous drainage, the relationship between the pulmonary veins and the left atrium is not constant, and their connections should not be used to define atrial morphology.

FIGURE 19.2. A: Subcostal long-axis view of a normal subject. The IVC can be seen entering the RA. B: Color flow Doppler shows hepatic vein flow.
FIGURE 19.3. Apical four-chamber view from a patient demonstrates the entrance of a pulmonary vein (asterisk) into the LA.

**Ventricular Morphology**

Once visceroatrial situs and venous connections are established, the orientation and morphology of the ventricles should be determined. During normal embryogenesis, the straight heart tube folds to the right (a D-loop) and then pivots to occupy a position within the left side of the chest. This positioning results in the right ventricle developing anteriorly and to the right of the left ventricle. The base-to-apex axis points leftward and most of the cardiac mass lies within the left side of the chest. If the initial fold in the heart tube is leftward, an L-loop develops, with the morphologic right ventricle to
the left of the morphologic left ventricle. Thus, atrioventricular discordance occurs in the presence of situs solitus and an I-loop or situs inversus and a d-loop.

Ventricular morphology is readily assessed with two-dimensional echocardiography. Features that are useful in distinguishing the right and left ventricles are listed in Table 19.3. The presence of muscle bundles, particularly the moderator band, gives the right ventricle a trabeculated endocardial surface (Fig. 19.4). In contrast, the left ventricle is characterized by a smooth endocardial surface. This distinction is apparent using echocardiography and serves as one of the more reliable characteristics when determining ventricular morphology. The structure and position of the atrioventricular valves are additional echocardiographic clues that are useful in distinguishing the right and left ventricles. If two ventricles are present, the atrioventricular valves associate with the corresponding ventricle and identification of the mitral and tricuspid valves defines the respective chambers. The tricuspid valve is more apically displaced and has three leaflets (and three papillary muscles) and chordal insertions into the septum. The mitral valve has a more basal septal attachment and has two leaflets, which insert into two papillary muscles but not the septum. All these features can be assessed with echocardiography. The four-chamber view allows the echocardiographer to determine ventricular morphology and the relative positions of the atrioventricular valves. The short-axis views permit definition of the papillary muscles and chordal insertions. The relative positions of the atrioventricular valves and the presence or absence of chordal insertions into the septum are the most helpful echocardiographic features when attempting to determine ventricular identity.

| **Table 19.3** ECHOCARDIOGRAPHIC CHARACTERISTICS OF RIGHT AND LEFT VENTRICLES |
|-----------------------------|-----------------------------|
| **Right Ventricle**         | **Left Ventricle**          |
| Trabeculated endocardial surface | Smooth endocardial surface |
| Three papillary muscles     | Two papillary muscles       |
| Chordae insert into ventricular septum | Ellipsoidal geometry |
Great Artery Connections

The final step in the segmental approach to cardiac anatomy involves identification of the great arteries and their respective connections. In the normal heart with concordant connections, the morphologic left ventricle gives rise to the aorta and the pulmonary artery serves as the outlet of the right ventricle. In the presence of normal ventricular orientation, this arrangement results in an anterior and leftward pulmonary artery and a posterior and rightward aorta with a left-sided aortic arch and descending aorta. The great arteries originate in orthogonal planes creating a “sausage and circle” appearance on short-axis imaging, which results from the rotation during development of the right ventricular outflow tract and pulmonary artery (the “sausage”) around the ascending aorta (the “circle”). Discordant ventriculoarterial connections, or transposition, occur when the great arteries arise from the opposite ventricle. Two forms of transposition occur. In d-transposition, the atrioventricular relationship is normal and the morphologic right ventricle is located to the right of the morphologic left ventricle. The great arteries are discordant, with the aorta arising from the right ventricle and the pulmonary artery from the left ventricle. This creates two parallel circuits, which is incompatible with life. In l-transposition, atrioventricular discordance is present (because of formation of an l-loop during embryogenesis) so that the morphologic right ventricle lies to the left of the morphologic left ventricle. The great arteries are also discordant, but because the ventricles are reversed, normal blood flow is preserved. That is, blood flows from right atrium to left ventricle to pulmonary artery, and from left atrium to right ventricle to aorta.
FIGURE 19.4. A four-chamber view from a normal subject demonstrating a moderator band in the right ventricular apex (arrow).
FIGURE 19.5. Parasternal short-axis echocardiograms from a healthy subject (A) and a patient with D-transposition of the great arteries (B). In the healthy subject, the AV is posterior and the right ventricular outflow tract and PA appear to wrap around the aorta. With transposition, the aorta is anterior and the two great vessels arise in parallel.

Two-dimensional echocardiography permits accurate identification of the great arteries and their origins and relationship. The short-axis view at the base of the heart is most helpful when assessing these features. In the normal heart, the pulmonary valve lies slightly anterior and to the left of the aortic valve (Fig. 19.5A). The pulmonary artery then courses posteriorly and bifurcates, with the right pulmonary artery passing immediately below the aortic arch. An abnormal relationship between the great arteries is illustrated in Figure 19.5B, from a patient with D-transposition. In this condition, the two arteries arise in parallel with the aorta more anterior. These findings are best appreciated in the parasternal long- and short-axis and subcostal views. The proximal aorta is optimally recorded from the parasternal window and the suprasternal notch (Fig. 19.6). To identify the great arteries, the course of the vessel and the presence or absence of a bifurcation are the most reliable echocardiographic signs. From the suprasternal short-axis view, the presence of a right aortic arch also can be detected by assessing the course of the brachiocephalic vessels as they leave the arch.

ABNORMALITIES OF RIGHT VENTRICULAR INFLOW
The right ventricular inflow tract and tricuspid valve are visualized using the apical and subcostal four-chamber views, the short-axis view at the base, and the medially angulated parasternal long-axis view. The most important congenital pathologic entities involving the tricuspid valve are Ebstein anomaly and tricuspid atresia (discussed subsequently). Ebstein anomaly consists of apical displacement of the septal and posterior (and sometimes the anterior) leaflets of the tricuspid valve into the right ventricle, often with some tethering of the leaflets to the right ventricular wall. Typically, the leaflets are elongated and redundant with abnormal chordal attachments. This results in “atrialization” of the basal portion of the right ventricle as the functional orifice is displaced apically relative to the anatomic annulus. Ebstein anomaly is a spectrum of abnormalities, depending on the extent of apical displacement of the valve, the distal attachments of the leaflets, the size and function of the remaining right ventricle, the degree of tricuspid regurgitation, and the presence of right ventricular outflow tract obstruction (usually from the redundant anterior tricuspid valve leaflet).

FIGURE 19.6. Suprasternal long- (A) and short-axis (B) views from a healthy subject. The right pulmonary artery (RPA) passes below the aortic arch (AA) and above the left atrium. The SVC can be seen to the right of the aortic arch.
The best echocardiographic view for the evaluation of Ebstein anomaly is the four-chamber view. The characteristic features identified in this plane are shown schematically in Figure 19.7. Of principal importance is the accurate recording of the level of insertion of the septal leaflet of the tricuspid valve relative to the annulus. Apical displacement of this insertion site is optimally assessed in this view and is the key to diagnosis. Because the tricuspid valve is normally positioned more apically than the mitral valve, abnormal apical displacement is relative, and some investigators have suggested measuring the distance between insertion sites of the two atrioventricular valves. When normalized for body surface area, a distance of greater than 8 mm/M² is indicative of Ebstein anomaly. Other investigators have advocated a maximal displacement of more than 20 mm as the diagnostic criterion in adults. Figure 19.8 is an example of relatively mild Ebstein anomaly. Apical displacement is apparent, but there is little tethering of the tricuspid valve leaflets and only mild tricuspid regurgitation.
The four-chamber and medially angulated parasternal views may be used to assess the severity of Ebstein anomaly and to determine surgical options. The degree of atrialization of the ventricle, the extent of leaflet tethering, and the magnitude of deformity or dysplasia of the valve leaflets are important features with implications for surgical repair (Fig. 19.9). In this example, tethering of the tricuspid leaflets is present, but little atrialization of the right ventricle occurs. The right heart is dilated and the septum is displaced leftward. The extent of chordal attachments between the anterior leaflet and the anterior free wall should be assessed in multiple views. If tethering is significant, valve replacement rather than repair may be required. The greater the degree of atrialization is, the worse the prognosis. Figure 19.10 is an example of an extreme form of Ebstein anomaly, with displacement of the tricuspid leaflets well into the right ventricular apex and marked tethering of the valve tissue. If the area of the functional right ventricle is less than one-third of the total right ventricular area, overall prognosis is poor. Because of the complexity of right ventricular geometry, an accurate measure of the size of the functional right ventricle is difficult, and all available views should be used. Doppler echocardiography should be used to detect tricuspid regurgitation, which is commonly seen in patients with Ebstein anomaly. A redundant anterior tricuspid valve leaflet may cause functional right ventricular outflow tract obstruction, which can also be detected with Doppler imaging. In severe cases, pulmonary atresia may be present, although it is rarely seen in adults.

Ebstein anomaly may be associated with a variety of other abnormalities that can be detected with echocardiography, namely, atrial septal defect, mitral valve prolapse, and left ventricular dysfunction. The etiology of the left ventricular dysfunction is not known, but its presence is associated with a poor prognosis. Surgical options in patients with Ebstein anomaly include tricuspid valve repair or replacement. After surgical repair, echocardiography plays a role in assessing the success of the procedure and the function of the tricuspid valve.
FIGURE 19.8. A: A four-chamber view from a patient with Ebstein anomaly. The *arrow* indicates the degree of apical displacement of the tricuspid valve insertion site. In B, mild tricuspid regurgitation is indicated by the *arrow*. Video 19-8A
FIGURE 19.9. From a patient with Ebstein anomaly, a greater degree of tricuspid valve displacement is shown. In A, the RV is dilated and the septum bows toward...
the left ventricle. In B, the tricuspid valve is displaced well into the right ventricle, as indicated by the arrows. The functional right ventricle is markedly reduced. In C, tethering of the tricuspid valve leaflets is apparent. Moderate tricuspid regurgitation is noted in D.  

Video 19-9A

Video 19-9B
FIGURE 19.10. An extreme example of Ebstein anomaly. From the apical four-
chamber view (A), the tricuspid valve leaflets (arrows) are displaced far into the right ventricular apex. In B, note the origin of the tricuspid regurgitation jet far into the right ventricular cavity (arrow).

Table 19.4

### LEVELS OF OBSTRUCTION OF LEFT VENTRICULAR INFLOW

<table>
<thead>
<tr>
<th>Pulmonary veins</th>
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<tbody>
<tr>
<td>Pulmonary vein stenosis (discrete)</td>
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<tr>
<td>Hypoplastic pulmonary veins</td>
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<tr>
<td>Extrinsic compression</td>
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<tr>
<th>Left atrium</th>
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<tr>
<td>Cor triatriatum</td>
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<td>Supravalvular stenosing ring</td>
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<tr>
<th>Mitral valve</th>
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<tbody>
<tr>
<td>Hypoplastic mitral valve</td>
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<tr>
<td>Congenital mitral stenosis</td>
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<tr>
<td>Parachute mitral valve</td>
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<td>Anomalous mitral arcades</td>
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<td>Double-orifice mitral valve</td>
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**ABNORMALITIES OF LEFT VENTRICULAR INFLOW**

**Pulmonary Veins**

Obstruction of left ventricular inflow can occur at several levels (Table 19.4). Pulmonary vein stenosis may be seen as an isolated entity or in association
with other congenital lesions. In one form, discrete areas of stenosis involving one or more pulmonary veins occur at or near the junction with the left atrium. Alternatively, hypoplasia of the pulmonary veins may be present. The echocardiographic diagnosis of the discrete form of pulmonary vein stenosis is contingent on the ability to visualize the entrance of the veins into the left atrium, which is optimally recorded using the apical and subcostal four-chamber views (Fig. 19.11). In younger patients, a posteriorly angulated suprasternal short-axis view (sometimes referred to as the “crab view”) can also be obtained. Usually, only the right or left upper pulmonary veins are imaged (see Fig. 19.3). Because of the proximity of the transducer to the left atrium, transesophageal echocardiography is superior for recording the insertion of the pulmonary veins (Fig. 19.12). An approach to pulmonary vein visualization using this technique is covered in Chapter 7. In most patients, all four veins can be visualized. Echocardiography has also been used for the diagnosis of pulmonary vein obstruction from compression by an extrinsic mass or secondary to stricture after an atrial fibrillation ablation procedure.
FIGURE 19.11. Pulmonary venous flow can be recorded from the apical four-chamber view with the sample volume placed in the mouth of the pulmonary vein as it enters the left atrium.
FIGURE 19.12. A: A transesophageal echocardiogram shows the entrance of the right lower pulmonary vein (RLPV) and the right upper pulmonary vein (RUPV) into the left atrium. B: Flow in the left upper pulmonary vein is recorded from transesophageal echocardiography. In this example, moderately increased flow velocity is the result of left-to-right shunting through an atrial septal defect. $PV_S$, $PV_D$, and $PV_A$ refer to pulmonary vein flow during systole, diastole, and atrial systole, respectively.
Visualizing pulmonary vein stenosis with two-dimensional echocardiography is rarely possible, and Doppler imaging is the primary means of securing a noninvasive diagnosis. Color Doppler imaging is useful when attempting to identify venous inflow and to detect the turbulent flow associated with stenosis. Because of the increase in velocity distal to the stenosis, color Doppler imaging may record a jet of blood entering the left atrium near the posterior wall. Turbulent flow in the posterior left atrium may be the initial echocardiographic abnormality and should suggest the possibility of a stenotic pulmonary vein. Then, pulsed Doppler imaging can be used to assess the inflow pattern and determine flow velocity. Normally, biphasic antegrade pulmonary venous flow (during ventricular systole and early diastole) is recorded (Fig. 19.12B).

**Left Atrium**

Obstruction of left ventricular filling also occurs at the atrial level, usually because of a fibrous membrane that impedes the flow of blood through the chamber. These membranes may be located in the middle of the atrium, effectively partitioning the left atrium into two chambers (a condition known as cor triatriatum), or they may occur at or near the level of the mitral annulus (a supravalvular stenosing ring). Such membranes are readily detected and localized with two-dimensional echocardiography. The membrane is visualized as a linear, echogenic structure extending from the anterosuperior to the posterolateral wall. In most cases, the superior “chamber” receives the pulmonary veins and the inferior “chamber” is associated with the atrial appendage and mitral valve (which is usually normal). Because of the orientation of the membrane, the four-chamber view is often optimal because it places the membrane perpendicular to the beam (Fig. 19.13). Note in Figure 19.14 the improved visualization of the membrane from an apical window compared with the parasternal view. The obligatory perforation connecting the two is most often posterior and may be multiple. This communication may be difficult to record with echocardiography. Color Doppler imaging usually permits localization of the opening in the membrane so that the pressure gradient can be assessed with pulsed Doppler imaging. Figure 19.15 is an example of cor triatriatum assessed from the transthoracic approach. The atrial membrane is clearly visualized from multiple views.
When the transthoracic study is suboptimal, transesophageal echocardiography should be used for evaluating this entity.

**FIGURE 19.13.** A: From a patient with cor triatriatum, the membrane in indicated by the arrow, separating the left atrium into an inferior and superior portion. The gap through the membrane is apparent. In B, flow around the membrane can be demonstrated using color Doppler. Pulsed Doppler (C) indicates low-velocity flow, ruling out a high-grade obstruction across the membrane. [Video]
FIGURE 19.14. Cor triatriatum is demonstrated from the parasternal long-axis (A) and four-chamber (B) views. The membrane (arrows) within the left atrium is much better seen from the apical window. In such cases, color Doppler imaging is useful to demonstrate turbulent flow through the defect in the membrane (arrow).
coming soon

Video 19-14
FIGURE 19.15. In this patient with cor triatriatum, the linear echo seen within the left atrium represents a membranous partition in the chamber. This membrane is visualized from the apical long-axis (A) and the four-chamber view (B). In panel C, color flow imaging demonstrates left atrial flow around the membrane and through the mitral valve, confirming incomplete partitioning of the atrium.

Video 19-15

Distinguishing among the different levels of left ventricular inflow obstruction requires a combination of two-dimensional imaging and Doppler imaging and is best accomplished using the parasternal long-axis and apical four-chamber views. An example of a supravalvular stenosing ring, in the setting of Shone complex, is presented in Figure 19.16. In this case, both a subaortic membrane and a supravalvular stenosing ring are present. In contrast to cor triatriatum, these supravalvular membranes are closer to the mitral valve and may actually adhere to the valve leaflets. In the example presented, the membrane was not well visualized in the long-axis view, although restricted mobility of the mitral leaflets was apparent. Absence of anterior leaflet doming excludes the possibility of rheumatic mitral stenosis, and the presence of the supravalvular membrane was detected from the apical window. By using color Doppler imaging, identification of flow acceleration and turbulence at the level of the annulus rather than the leaflet tips is an additional clue to distinguish a supravalvular ring from mitral valve stenosis. Continuous-wave Doppler imaging can then be used to assess the severity of the obstruction (see Fig. 19.16D). The proximity of the membrane to the valve can lead to leaflet damage, the result of high-velocity turbulent flow. Leaflet thickening and mitral regurgitation may develop as a consequence.
Caution must be used when diagnosing a supravalvular stenosing ring with echocardiography. Differentiating between a thickened and calcified mitral annulus and a stenosing ring may be difficult, leading to both false-positive and false-negative results. Associated anomalies are seen frequently with both cor triatriatum and supravalvular stenosis. Atrial septal defect and persistent left superior vena cava are especially common and are readily detected with echocardiography.

**FIGURE 19.16.** An example of Shone complex. **A:** Restricted mitral valve motion during diastole is present, but the stenosing ring is not visualized from this view. **B:** The restricted leaflet motion, as well as the presence of the fibrous ring (arrows) and its relationship to the mitral valve, is better seen from the apical four-chamber view. **C:** Color Doppler imaging demonstrates turbulent antegrade flow during diastole through the abnormal mitral valve. **D:** Continuous-wave Doppler imaging demonstrates a significant pressure gradient across the mitral valve.
Mitral Valve

Congenital mitral stenosis is far less common than rheumatic mitral valve disease. Several anatomic variations exist (Table 19.4), and all can be diagnosed accurately with echocardiography. Because rheumatic mitral stenosis is so much more common in adults, however, the diagnosis of congenital mitral stenosis is often missed. Figure 19.17 is an example of a parachute mitral valve. In this condition, all the chordae insert into a single, large papillary muscle (see Fig. 19.17B). The parasternal short-axis view is most helpful in determining the number, size, and location of the papillary muscles. The long-axis view reveals deformity and thickening of the mitral valve, restricted leaflet excursion, and chordal thickening and fusion. The degree of stenosis is variable and is best assessed with Doppler imaging. Because many of these features are common to rheumatic mitral valve disease, proper diagnosis is sometimes difficult and relies on detecting the presence of a single papillary muscle.
**FIGURE 19.17.** An example of parachute mitral valve. **A:** The long-axis view reveals thickened mitral leaflets that dome in diastole. **B:** A short-axis view at the midventricular level demonstrates the chordae converging on a single papillary muscle (*arrow*). **C:** The orifice of the abnormal mitral valve is shown from the short-axis view. Although the orifice is large, a mild degree of subvalvular gradient was present.

**Video 19-17**

**FIGURE 19.18.** Parasternal short-axis views from two patients with a double-orifice mitral valve (MV).

Other congenital forms of mitral stenosis include anomalous mitral arcade and double-orifice mitral valve. In arcade-type mitral stenosis, the chordae insert into multiple small papillary muscles. Both stenosis and regurgitation
are possible. Double-orifice mitral valve occurs because of duplication of the mitral orifice with or without fusion of subvalvular chordal structures. Usually, all the chordae associated with each orifice insert into the same papillary muscle, a situation similar to parachute mitral valve. The diagnosis is made by visualization of two separate orifices in the short-axis view (Fig. 19.18). The presence and severity of stenosis are variable in this condition. Other forms of congenital mitral valve pathology, including mitral valve prolapse and cleft mitral valve, are discussed in Chapter 11.

**ABNORMALITIES OF RIGHT VENTRICULAR OUTFLOW**

**Right Ventricle**

Narrowing of the right ventricular outflow tract can occur on several levels, and obstruction may be present at multiple sites within an individual patient. Subvalvular pulmonary stenosis usually involves the infundibulum and is less common than valvular stenosis. Infundibular pulmonary stenosis may be the result of discrete fibromuscular narrowing or hypertrophied muscle bundles (also called double-chambered right ventricle) (Fig. 19.19). In many cases, a ventricular septal defect is also present. Right ventricular outflow tract narrowing is occasionally secondary to stenosis at a more distal level. For example, valvular pulmonary stenosis may lead to right ventricular hypertrophy, the development of subvalvular muscle bundles, and subsequent outflow tract narrowing.

Two-dimensional echocardiography is well suited to the evaluation of the right ventricular outflow tract. The parasternal short-axis and the subcostal four-chamber views are ideal for assessing the complex geometry of this region and for determining the level and severity of stenosis. The use of Doppler imaging to measure the pressure gradient may be challenging, however. Orienting the ultrasound beam parallel to the outflow tract jet requires considerable effort and the use of all available windows. Furthermore, localization of the site of stenosis may be difficult if narrowing occurs at more than one level. Typically, subvalvular stenosis is a dynamic form of obstruction with maximal velocity occurring in late systole, a pattern that is analogous to the outflow jet seen in hypertrophic cardiomyopathy of
the left ventricle. The magnitude of reduction in pulmonary artery flow can affect development of the pulmonary arteries, which can be an important factor in surgical planning. Therefore, an evaluation of children with any form of right ventricular outflow tract obstruction should include an assessment of the pulmonary arteries. This includes patients with tetralogy of Fallot, in whom the type and timing of surgical repair are determined in part by the size of the pulmonary arteries.

**Pulmonary Valve**

Stenosis of the pulmonary valve is a fairly common congenital lesion that may occur in isolation or in association with other cardiac defects. The most frequently encountered form is characterized by fusion of the cusps and incompletely formed raphae, resulting in a dome-like structure with a narrowed orifice. Typically, the valve annulus is normal in size. With severe stenosis, right ventricular hypertrophy may lead to variable degrees of subvalvular narrowing.

In adults, the morphology of the stenotic pulmonary valve is best visualized in the parasternal short-axis plane through the base of the heart. With two-dimensional echocardiography, the cusps appear thickened, have decreased excursion, and dome in systole (Fig. 19.20). Poststenotic pulmonary artery dilation is frequently evident, but its presence does not correlate with severity. In most cases, right ventricular size and function are normal, and trabeculation of the right ventricular walls is increased (see Fig. 19.20A). Calcification of the valve is characteristic in adults, but not children, with this disorder. Less common, dysplasia of the pulmonary valve will cause valvular stenosis at birth due to myxomatous thickening of the leaflets (Fig. 19.21). When pulmonary stenosis is severe, evidence of right ventricular pressure overload will be present. The degree of septal flattening and right ventricular enlargement correlate roughly with the severity of stenosis. Figure 19.22 is an example of extreme right ventricular pressure overload secondary to severe valvular pulmonary stenosis.
FIGURE 19.19. A series of short-axis images demonstrate infundibular right ventricular narrowing. **A:** Note the presence of muscle bundles in the area of the right ventricular outflow tract (arrow). **B:** The relationship of the subvalvular narrowing to the pulmonary valve (arrow). **C:** Color Doppler imaging demonstrates turbulence in this area. Dynamic subvalvular stenosis is present with a late-peaking gradient.
FIGURE 19.20. An example of valvular pulmonary stenosis. A: From the basal short-axis view, doming of a mildly thickened pulmonary valve can be seen (arrow). B: Doppler demonstrates a peak gradient of 48 mm Hg and a mean gradient of 26 mm Hg. 

Video 19-19
FIGURE 19.21. An example of dysplastic pulmonary valve stenosis. A: The pulmonary valve (arrow) is markedly thickened and immobile. Doming during systole is present. B: A maximal pressure gradient of approximately 65 mm Hg.

Although two-dimensional echocardiography is essential for the morphologic diagnosis of pulmonary stenosis, the technique is limited for assessing the severity of obstruction. Neither the degree of cusp thickening nor the presence of right ventricular hypertrophy provides a quantitative measure of severity. Doppler imaging is the technique of choice to measure the severity of pulmonary stenosis. Using the modified Bernoulli equation, the peak instantaneous pressure gradient can be calculated (Figs. 19.20 to 19.22). Several clinical studies have demonstrated an excellent correlation
between Doppler imaging and catheterization-derived pressure gradients in patients with pulmonary stenosis. In most patients, optimal alignment of the Doppler beam with the stenotic jet is from the parasternal short-axis view. In some individuals, use of a lower interspace is necessary to better align with a superiorly directed jet. In patients with pulmonary artery dilation, anterior displacement of the valve precludes proper beam alignment from the parasternal window. In this situation, the subcostal or suprasternal approach is usually adequate. In children, particularly, the subcostal approach provides optimal beam alignment and permits detection of the maximal jet velocity.

In children with pulmonary stenosis, surgical valvotomy or balloon valvuloplasty is often performed to relieve the obstruction. After such interventions, Doppler echocardiography may be used for serial evaluation and to detect residual stenosis (Fig. 19.23). The magnitude of associated pulmonary insufficiency and abnormalities of right ventricular diastolic filling can also be assessed. In patients with combined valvular and infundibular stenosis, the presence of serial obstructions may result in overestimation by continuous-wave Doppler imaging of the catheterization-derived pressure gradient.

Pulmonary Artery
Pulmonary artery stenosis (also referred to as peripheral or supravalvular pulmonary stenosis) can occur at any level and often involves multiple sites. Several morphologic forms exist, including discrete membrane-like lesions, long tubular stenoses, and tubular hypoplasia. These anomalies are frequently associated with other congenital cardiac and extracardiac lesions (e.g., Williams syndrome). The ability to detect pulmonary artery stenoses with echocardiography depends on the location of the lesions. Proximal lesions can be visualized from the parasternal short-axis window. Figure 19.24 is an example of peripheral pulmonary stenosis involving the right branch. In most such cases, the diagnosis is apparent from two-dimensional echocardiographic imaging. Color Doppler imaging should be used to demonstrate turbulence and acceleration of flow within the stenotic segment. The echocardiographer must bear in mind, however, that a more common cause of turbulent flow within the main pulmonary artery is patent ductus arteriosus. More peripheral stenoses may be impossible to visualize,
especially in older patients. In children, the subcostal four-chamber and the suprasternal views may permit detection of distal lesions. The diagnosis should be considered in a patient with unexplained right ventricular hypertrophy, particularly in the presence of a pulsatile proximal pulmonary artery.

**FIGURE 19.22.** A: A patient with severe pulmonary stenosis demonstrates septal flattening with a dilated and hypertrophied right ventricle. These findings are consistent with right ventricular pressure overload. B: Severe pulmonary stenosis is confirmed with a maximal pressure gradient of approximately 95 mm Hg. Note the presence of presystolic flow through the pulmonary valve at the time of right atrial systole (arrow). Video 19-22
FIGURE 19.23. A case of pulmonary stenosis is shown before (pre) (A) and after (post) (B) valvuloplasty. The procedure resulted in a decrease in pulmonary valve gradient from 90 to 25 mm Hg.

ABNORMALITIES OF LEFT VENTRICULAR OUTFLOW

Congenital abnormalities of left ventricular outflow usually involve obstruction of flow, and several important forms exist. These lesions may be categorized as subvalvular, valvular, or supravalvular (which includes coarctation of the aorta) (Table 19.5). The subvalvular forms are heterogeneous and include hypertrophic cardiomyopathy, which is discussed in Chapter 18. The most important forms are the valvular lesions, which are common causes of stenosis in children (the unicuspid or congenitally stenotic aortic valve) and in adults (the bicuspid valve). The form of supravalvular obstruction encountered most frequently in the adult patient is coarctation of the aorta. This section includes a discussion of the lesions that occur at each of these different levels in order, but the focus is on those anomalies that are most common in adults.

Subvalvular Obstruction

Two types of subvalvular aortic stenosis are discussed here: the discrete form and the fibromuscular type of subaortic obstruction. Together, these lesions account for less than 20% of all cases of left ventricular outflow obstruction in children and both are uncommon in adult patients. Discrete subaortic stenosis results from a thin, fibrous membrane or ridge that forms a crescentic barrier within the outflow tract just below the aortic valve. The membrane
usually extends from the anterior septum to the anterior mitral leaflet. The degree of obstruction to flow is variable, and aortic regurgitation develops in approximately 50% of patients. With two-dimensional echocardiography, these membranes are seen as a discrete linear echo in the left ventricular outflow tract perpendicular to the interventricular septum. Because the membranes are parallel to the beam, recording these structures from the parasternal long-axis window may require the use of multiple transducer positions. In many cases, the membranes are detected more easily from the apical views (where the ultrasound beam is oriented perpendicular to the structure, Fig. 19.25). Transesophageal echocardiography has also been used in the assessment of patients with subvalvular obstruction. Doppler imaging plays an essential role in the evaluation of these patients. After the location and orientation of the jet are visualized with color flow imaging, continuous-wave Doppler imaging can be used to estimate the peak pressure gradient across the membrane (Fig. 19.26). In the absence of aortic valve stenosis, this value correlates well with the catheterization-derived measure of obstruction. In the presence of multiple serial stenoses, however, Doppler imaging may overestimate the catheterization-measured gradient. The presence and severity of aortic regurgitation can also be assessed with Doppler techniques (Fig. 19.25). Figure 19.27 is an example of a subaortic membrane evaluated with transesophageal imaging. Note how the attachment of the membrane to the anterior mitral leaflet deforms the valve, especially during systole. M-mode echocardiography can also be helpful in assessing subvalvular obstruction (Fig. 19.27C). Mid systolic partial closure with reopening of the leaflets in late systole is indicative of a subvalvular pressure gradient. Figure 19.28 is an example of recurrent subaortic stenosis, several years after surgical resection. In this case, the subaortic membrane is contiguous with the valve cusps, resulting in both stenosis and regurgitation.

Membranous subaortic stenosis is distinguished from a subaortic fibromuscular ridge or tunnel with two-dimensional echocardiography. Tunnel-type subaortic obstruction, rarely seen in adults, is characterized by diffuse thickening and narrowing of the left ventricular outflow tract with associated concentric left ventricular hypertrophy. A fibromuscular ridge may also obstruct the outflow tract (Fig. 19.29). This entity is similar to discrete membranous subaortic stenosis, but the obstruction is thicker and less discrete and appears more muscular.

<table>
<thead>
<tr>
<th>Table 19.5</th>
<th>CLASSIFICATION OF THE VARIOUS CONGENITAL FORMS OF LEFT VENTRICULAR OUTFLOW TRACT OBSTRUCTION</th>
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<tbody>
<tr>
<td>Subvalvular</td>
<td>Discrete membranous stenosis&lt;br&gt;Fibromuscular tunnel&lt;br&gt;Hypertrophic obstructive cardiomyopathy</td>
</tr>
<tr>
<td>Valvular</td>
<td>Unicuspid&lt;br&gt;Bicuspid&lt;br&gt;Dysplastic</td>
</tr>
<tr>
<td>Supravalvular</td>
<td>Discrete (membranous or “hourglass”)&lt;br&gt;Aortic hypoplasia or atresia&lt;br&gt;Interrupted aortic arch&lt;br&gt;Coarctation of the aorta</td>
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These different forms of subaortic obstruction probably exist as a continuum, with a thin discrete membrane at one extreme and a diffuse tunnel at the other. Differentiating among individual cases may, therefore, be difficult and somewhat arbitrary. All these forms of subaortic obstruction are frequently associated with ventricular septal defects. Occasionally, other congenital cardiac anomalies are associated with subvalvular left ventricular outflow tract obstruction, including accessory mitral valve chordae, anomalous papillary muscle insertion, and abnormal insertion of the anterior
Valvular Aortic Stenosis

Aortic stenosis may be present at birth (a congenitally stenotic aortic valve) or may develop over time in a congenitally abnormal, but not stenotic, valve. In the former, the valve may be acommissural (resembling a volcano and more typical of pulmonary stenosis) or unicuspid unicommissural (with a slit-like orifice, resembling an exclamation point, Fig. 19.30). A bicuspid or tricuspid valve can also be stenotic at birth because of commissural fusion or dysplasia. Most often, such valves will be functionally normal at birth but gradually become stenotic over time because of progressive fibrosis and calcification. In other cases, degeneration of the valve leads to predominant aortic regurgitation. Quadricuspid valves are rare and have a similar natural history (Fig. 19.31).

FIGURE 19.25. A patient with membranous subaortic stenosis is shown. In A, in the long axis, the membrane (arrow) is subtle and easily missed. In B, color
Doppler shows turbulent flow acceleration in the left ventricular outflow tract below the aortic valve, suggesting the presence of subaortic flow disturbance. A magnified view (C), clearly shows the membrane (arrow) and its relation to the valve. In D, continuous-wave Doppler reveals a mean gradient of 23 mm Hg.
FIGURE 19.26. These two cases demonstrate the continuum between a discrete subaortic membrane and a fibromuscular ridge. **A**: A discrete membrane is demonstrated. Note how the membrane attaches to and deforms the base of the
anterior mitral leaflet. A 60 mm Hg peak systolic gradient is confirmed (B). C: A fibromuscular ridge (arrow) in association with a membrane is located just below the aortic valve. In this patient, the peak gradient across the subvalvular obstruction is approximately 52 mm Hg (D).

Bicuspid aortic valve is estimated to occur in 1% to 2% of the general population, making it the single most common congenital cardiac anomaly. As just noted, these valves often are functionally normal at birth (Fig. 19.32). Two-dimensional echocardiography plays a major role in detection of this entity. Direct visualization of the aortic cusps is possible from the parasternal short-axis view through the base of the heart. During diastole, the cusps of a normal tricuspid valve are closed within the plane of the scan and the commissures form a “Y” (sometimes referred to as an inverted Mercedes-Benz sign). A true bicuspid valve has two cusps of nearly equal size, two associated sinuses, and a single linear commissure. A raphe may be present and, if present, creates the illusion of three separate cusps. By observing valve opening in systole, however, the number of distinct cusps is apparent. Fusion of two of the cusps may create the appearance of a bicuspid valve, but the presence of three distinct sinuses will establish this difference. Confirming the presence of a bicuspid aortic valve with echocardiography requires high-resolution images from the short-axis view for adequate visualization of valve morphology. A unicuspid valve has a single slit-like commissure, and the opening is eccentric and restricted. The stenotic tricuspid valve has three cusps with variable degrees of commissural fusion. Thus, an accurate assessment of functional anatomy requires an analysis of the number of apparent cusps, the degree of cusp separation, and a recording of their mobility and excursion during systole.
FIGURE 19.27. A subaortic membrane is demonstrated using transesophageal echocardiography. A: From a long-axis view, the membrane can be seen in the left ventricular outflow tract extending from the septum (arrow) to the anterior mitral leaflet. Note how the mitral leaflet is deformed by the attachment of the membrane. B: Color Doppler during systole demonstrates turbulent flow within the left ventricular outflow tract, beginning at the level of the membrane. C: With a subaortic membrane, M-mode echocardiography demonstrates the characteristic mid systolic partial closure and coarse fluttering of the aortic valve cusps.
FIGURE 19.28. Recurrence of a resected subaortic membrane is shown. In A, the delicate membrane was not visualized in the long-axis view. Using transesophageal echocardiography (B), the membrane (arrows) can be seen encroaching on the aortic valve leaflets and restricting their motion. Moderate aortic regurgitation is present (C). In D, The mean gradient is 45 mm Hg, indicating severe outflow tract obstruction.
Video 19-28b

coming soon

Video 19-28c

coming soon
FIGURE 19.29. A transesophageal echocardiogram, using both two-dimensional and three-dimensional imaging, is recorded in a patient with a fibromuscular ridge. 

A: In the long-axis view, fibrous thickening of the basal septum, just below the aortic valve is indicated by the arrow. 

B: The same long-axis view is shown using three-dimensional imaging. The relationship between the aortic cusps (arrows) and the narrowed outflow tract (white arrowhead) is demonstrated. 

C: Recorded from a short-axis view just above the aortic valve, this three-dimensional echocardiogram illustrates the subaortic orifice located just below the aortic cusps (indicated by the three white arrows).
FIGURE 19.30. A unicuspid aortic valve is evaluated with transesophageal echocardiography. **A:** From a short-axis view, the eccentric, oval-shaped orifice is shown during systole (arrows). **B:** Color flow imaging demonstrates turbulent, eccentric antegrade flow. **C:** From a long-axis view, the systolic doming of the aortic valve is apparent.
FIGURE 19.31. An example of a quadricuspid aortic valve is demonstrated on transesophageal echocardiography. The valve is recorded from the long-axis (A) and short-axis (B) views. Mild aortic regurgitation is present (C).
FIGURE 19.32. A functionally normal bicuspid aortic valve from a young patient. A: Long-axis view demonstrates doming of the valve in systole. B: Basal short-axis view confirms that the valve is bicuspid but with no evidence of stenosis.
Whereas the short-axis view is useful for determining the number of commissures and the degree, if any, of commissural fusion, movement of the cusps out of the imaging plane during systole precludes accurate determination of the presence and severity of stenosis. In fact, normal systolic excursion of the bodies of the cusps recorded from the short-axis view may lead to underestimation of the severity of congenital aortic stenosis. Thus, the short-axis view is useful when evaluating aortic valve anatomy but should never be used to exclude the possibility of congenital aortic stenosis. The long-axis views have several advantages for this purpose. The thickness and excursion of the cusps can be assessed. Normally, they appear as thin, delicate structures that appear to open completely in systole and are aligned parallel to and against the aortic walls. With congenital aortic stenosis, the cusps are thickened and appear to dome during systole, the result of restricted motion of the tips relative to the more mobile bodies of the cusps (Fig. 19.33). A qualitative estimate of severity is possible, based on the thickness and immobility of the cusps, the extent of leaflet tip separation in systole, the degree of left ventricular hypertrophy, and the presence of poststenotic aortic root dilation.

Doppler imaging should be used to complete the noninvasive assessment of aortic stenosis and to provide a quantitative evaluation of severity. The apical, right parasternal, and suprasternal windows should be used to ensure that the maximal velocity is obtained. Then, through the use of the modified Bernoulli equation, the peak pressure gradient can be calculated. Both peak
instantaneous and mean pressure gradients can be derived, and in children, the mean gradient is often used for clinical decision making. The values obtained with this approach correlate well with catheterization-derived gradients. Inherent differences exist between the two methods, and discrepancies should not necessarily be viewed as an error on the part of one or the other technique. In children especially, anxiety and increased activity during the examination will lead to an increase in flow velocity (both proximal and distal to the valve) and will thereby increase the measured pressure gradient. To calculate aortic valve area, the continuity equation can be used. It should be emphasized that the application of Doppler imaging to quantify aortic stenosis is similar in children and adults. The basic principles underlying these applications are covered in detail in Chapters 8 and 10.

**FIGURE 19.33.** A: A bicuspid aortic valve is seen from a high parasternal long-
axis view. The valve is thickened and domes in systole. The ascending aorta is
dilated and there is effacement of the sinotubular junction. In B, the short-axis
view shows thickening and calcification. In C, a mean gradient of over 60 mm Hg
is demonstrated indicating severe aortic stenosis.

Supravalvular Aortic Stenosis
The least common site of left ventricular outflow obstruction is in the
supravalvular area. Three morphologic types of supravalvular aortic stenosis
have been described: (1) fibromuscular thickening producing an hourglass-
shaped narrowing above the sinuses (the most common form); (2) a discrete
fibrous membrane in a normal-sized aorta, usually located near the
sinotubular junction; and (3) diffuse hypoplasia of the ascending aorta, often
involving the origins of the brachiocephalic arteries. Because of the presence of stenosis above the aortic valve and coronary ostia, two additional features often accompany these anomalies: (1) dilation of the coronary arteries, sometimes with ostial obstruction; and (2) thickening and fibrosis of the aortic cusps, usually with an element of aortic regurgitation. Williams syndrome includes supravalvular aortic stenosis, elfin facies, mental retardation, and occasionally, peripheral pulmonary stenosis. Isolated supravalvular aortic stenosis with or without peripheral pulmonary stenosis may be inherited as an autosomal dominant trait.

The parasternal long-axis view or a high right parasternal view is most helpful for diagnosing supravalvular aortic stenosis. In the normal aorta, the vessel diameter is greatest at the level of the sinuses. At the sinotubular junction, the diameter decreases slightly and approximates the size of the aortic annulus. With supravalvular aortic stenosis, an hourglass deformity occurs that is characterized by a segment of gradual tapering and then widening of the lumen. The aortic walls usually appear thickened and echogenic. Aortic cusp fibrosis is often present, but poststenotic dilation of the ascending aorta is not a feature of this anomaly. A hypoplastic aorta is characterized by more diffuse and extensive narrowing with variable involvement of the branch vessels.

**COARCTATION OF THE AORTA**

This relatively common condition is the result of localized narrowing of the descending aorta near the origin of the ductus arteriosus. The lesion consists of a ridge-like indentation of the posterolateral wall of the aorta resulting from thickening and infolding of the aortic media. It is typically located just distal to the origin of the left subclavian artery and the specific location may be “preductal” or “postductal” depending on the position of the ridge of tissue relative to the ductus (or ligamentum) arteriosus. It is often associated with other forms of CHD, especially bicuspid aortic valve and mitral valve malformations.

Echocardiographic detection of coarctation requires both an index of suspicion and careful recording of the descending aorta from the suprasternal window. In children, the evaluation of this portion of the aorta is relatively
straightforward. In adults, however, the assessment can be technically demanding and both false-negative and false-positive results occur. The goal is to record the arch and descending aorta in the long axis from the suprasternal notch. False-negative results usually result from an inability to image the most distal portion of the arch (where the narrowing occurs). False-positive findings are the result of a tangential imaging plane through the vessel, creating the illusion of narrowing. The origins of the carotid and subclavian arteries serve as landmarks when localizing the juxtaductal area. The location of the left subclavian artery relative to the coarctation is an important factor in surgical management. If an area of stenosis is suspected, care should be taken to ensure proper beam alignment. If the aortic lumen can be seen beyond the narrowing, the likelihood of a false-positive result is reduced (Fig. 19.34). Dilation and exaggerated pulsation of the proximal aortic arch are further evidence of significant coarctation.

An example of coarctation of the aorta in an adult patient is shown in Figure 19.35. Note the location of a shelf-like constriction just beyond the origin of the left subclavian artery. When two-dimensional echocardiographic imaging is diagnostic of (or suspicious for) coarctation, Doppler should be performed to aid in the diagnosis and to provide an estimation of the pressure gradient. As a first step, color Doppler imaging can be used to detect acceleration and turbulence within the region of narrowing (Fig. 19.34B). The absence of Doppler evidence of acceleration and turbulence of flow should alert the examiner to the possibility of a false-positive two-dimensional echocardiographic result. Color Doppler imaging also permits more accurate alignment of the continuous-wave Doppler beam. Figure 19.36 includes two examples of Doppler recordings of flow across an aortic coarctation. To estimate the peak pressure gradient, the Bernoulli equation can be used. When this equation is applied to aortic coarctation, however, it may be inappropriate to ignore the proximal aortic flow velocity. As a general rule, if this proximal velocity is less than 1.5 m/s, it can be ignored and the simplified equation can be used. If it is greater than 1.5 m/s, the expanded Bernoulli equation is necessary. In this way, a more accurate pressure gradient is obtained. The persistence of a high-velocity flow signal into diastole is another useful clue to the severity of the stenosis. A pressure gradient throughout the cardiac cycle indicates a more severe form of obstruction compared with a pressure gradient that is confined to systole (Fig.
In this example, severe coarctation is present. Following balloon dilation (Fig. 19.37B), the peak gradient is reduced and diastolic antegrade flow is no longer present. Because coarctation gradients are flow dependent, low-level exercise, usually in the form of leg lifts, can be performed to assess the response to stress. In many cases, exercise will not cause a significant increase in the peak gradient but will result in the development or increase in the diastolic gradient. In borderline cases, this response can be helpful in clinical decision making.

Although Doppler imaging is sensitive for the detection of coarctation, false-negative results can occur in the presence of a patent ductus arteriosus. Left-to-right runoff of blood flow through the ductus reduces the jet velocity through the coarctation and leads to an underestimation of the pressure gradient. This can also occur in the presence of well-developed collaterals. In such cases, the Doppler gradient will be an underestimation of the actual severity of obstruction.

False-positive results are even less common. Occasionally, a mild increase (1.5 to 2 m/s) in descending aortic flow velocity will be misinterpreted as evidence of coarctation. In the absence of turbulence or echocardiographic evidence of vessel narrowing, this should generally be attributed to normal acceleration around the arch. Long-term follow-up after repair of aortic coarctation relies heavily on echocardiographic methods for the detection of restenosis. In this example, an endovascular stent is visualized in the descending aorta (Fig. 19.38A) with a peak systolic gradient of approximately 30 mm Hg (Fig. 19.38B).

**ABNORMALITIES OF CARDIAC SEPTATION**

Defects in septation between the cardiac chambers constitute the largest single group of congenital cardiac malformations. These developmental anomalies may involve the atrial septum, the ventricular septum, or the conotruncus (the infundibulum or outlet portion of the ventricles). Within each category, specific lesions are designated on the basis of their embryologic origin and anatomic site. These anomalies often occur in association with other complex lesions; the focus of this section is on those conditions in which septation defects are the primary cardiac anomaly.
FIGURE 19.34. Coarctation of the aorta is evaluated from the suprasternal window. **A:** A long-axis view of the aortic arch suggests tapering of the descending aorta just beyond the origin of the left subclavian artery (arrow). **B:** Color flow imaging is useful to confirm turbulence and acceleration of flow at the level of the coarct (arrow). **C:** Then, continuous-wave Doppler imaging is used to quantify the pressure gradient. In this case, a peak systolic gradient of 50 mm Hg was recorded. TA, transverse aorta.
Atrial Septal Defect

There are four types of atrial septal defects, which correspond to abnormal development at specific stages of embryogenesis and to specific locations within the atrial septum (Fig. 19.39A). The most common type is the ostium secundum defect, located in the area of the fossa ovalis or middle of the atrial septum. In the adult population, this type comprises approximately two-thirds of all cases. The ostium primum defect involves the lower (or primum) portion of the atrial septum and accounts for approximately 15% of atrial septal defects seen in adults. This type may occur alone or in association with defects in the inlet portion of the ventricular septum and atrioventricular valves (i.e., as a component of an endocardial cushion defect). The sinus venosus defect is slightly less common (approximately 10% of cases) and occurs in the superior and posterior septum, near the junction of the superior vena cava. Defects in the area of the coronary sinus occur rarely, as an unroofed coronary sinus.

Atrial septal defects usually are single and vary considerably in size. Direct visualization of the atrial septum with two-dimensional echocardiography is the most accurate means by which to diagnose these lesions. The presence of an atrial septal defect is often first suspected, however, on the basis of indirect echocardiographic findings. Right ventricular dilation in an otherwise healthy young patient should always suggest this possibility. Abnormal motion of the interventricular septum is another clue to its presence. Typically, septal motion in the presence of an
atrial septal defect is characterized by brisk anterior movement in early systole or flattened motion throughout systole.

**FIGURE 19.35.** Coarctation of the aorta is demonstrated from transthoracic echocardiography. In **A**, a suprasternal view suggests narrowing of the proximal descending aorta, although image plane misalignment can lead to both false positive and negative results. In **B**, turbulence and flow acceleration suggests that significant stenosis may be present. In **C**, Doppler demonstrates a 35 mm Hg peak gradient across the coarctation with persistent antegrade flow throughout diastole.
Figure 19.36. A: Continuous-wave Doppler imaging demonstrates a peak systolic pressure gradient of 35 mm Hg across the coarctation. Superimposed within the systolic flow signal is a darker jet (arrow) that corresponds to flow proximal to the stenosis. Note the absence of flow during diastole. B: A more severe case of coarctation, with a peak gradient of 74 mm Hg. Note the persistence of low-velocity flow throughout diastole.

Figure 19.37. Balloon angioplasty can be used to treat coarctation of the aorta. These Doppler recordings were obtained before (pre) (A) and after (post) (B) balloon dilation of a coarct. The procedure resulted in a decrease in the peak gradient from approximately 100 to 25 mm Hg.

Two-dimensional echocardiography permits a more direct assessment of an atrial septal defect (Fig. 19.39B). As with M-mode echocardiography, right ventricular dilation and paradoxic septal motion can be detected. In the parasternal short-axis view, the abnormal ventricular septal geometry indicative of a right ventricular volume overload can be confirmed. This abnormal geometry is characterized by leftward displacement (or flattening) of the septum in diastole, the result of a right ventricular diastolic volume overload. During systole, the normal transseptal pressure gradient is restored and the septum regains its normal circular geometry. Rounding of the septum in early systole causes it to be displaced anteriorly (from its abnormal posterior position in late diastole). Figure 19.40 is from a patient with right ventricular volume overload due to an atrial septal defect. Septal flattening in diastole is present but reverses in systole, with restoration of normal circular geometry.

Two-dimensional echocardiography is the standard technique for direct visualization of atrial septal defects. To assess the presence, location, and size of an atrial septal defect, multiple echocardiographic views are required, and
an appreciation of the advantages and limitations of each is essential. In the apical four-chamber view, the atrial septum is located in the far field, relatively parallel to the ultrasound beam. Although the diagnosis of an ostium primum defect can often be made with confidence from this view, detection of a secundum defect is considerably more difficult. Shadowing and echo dropout (particularly in the area of the fossa ovalis) create the potential for false-positive results. To aid in diagnosis, saline contrast and/or color flow imaging can be performed. These techniques will usually allow distinction between echo dropout and a true septal defect (Fig. 19.41).

The subcostal four-chamber view places the atrial septum perpendicular to the ultrasound beam and thereby obviates many of the limitations of the apical approach (Fig. 19.42). From this window, the fossa ovalis is seen as a thin central region within the atrial septum. The presence and approximate size of secundum defects can be assessed accurately in more than 90% of cases. This view is also ideal when distinguishing among defects of the primum, secundum, and sinus venosus types. In fact, this is the only transthoracic view in which sinus venosus defects are consistently visualized. Although challenging in adults, careful interrogation of the most superior and posterior portions of the atria may allow detection of sinus venosus defects (Figs. 19.43 and 19.44). In a minority of adult patients, the entrance of the superior vena cava and pulmonary veins can be identified, thereby permitting diagnosis of anomalous pulmonary venous drainage (although this diagnosis usually requires transesophageal imaging). Finally, the subcostal views are helpful for the detection of an atrial septal aneurysm. These aneurysms consist of thin, billowing tissue in the area of the fossa ovalis that moves with the cardiac and respiratory cycles and usually protrudes into the right atrial cavity.
FIGURE 19.38. This patient with coarctation of the aorta was treated with an endovascular stent, which can be seen in A. The proximal and distal ends are approximated by the arrows. In B, Doppler shows a peak gradient of 30 mm Hg with no diastolic antegrade flow.
FIGURE 19.39. These schematics illustrate the different types of atrial septal defect. **A:** The relationship of the different types of atrial septal defects viewed from the perspective of the right heart. **B:** The differences among the types of atrial septal defect (ASD) from a subcostal four-chamber perspective. See text for details. RUPV, right upper pulmonary vein.
Regardless of the view, transthoracic image quality may preclude an acute diagnosis in some adult patients. To overcome this problem, the first step should involve color Doppler imaging and contrast echocardiography (Fig. 19.45). By aligning the Doppler sample volume perpendicular to the atrial septum in the subcostal view, flow across the defect can be recorded (Fig. 19.46). In the usual case, pulsed Doppler imaging will demonstrate low-velocity, left-to-right flow extending from mid systole to mid diastole, with a second phase of flow coincident with atrial systole. A brief period of right-to-left shunting may also be recorded in early systole. Because the pressure difference between the atria is relatively small, a high-velocity jet will not be present. The respiratory phase will also affect the flow pattern. Care must be taken to avoid confusing the low-velocity shunt flow with normal venous and atrioventricular valve flow. Although color flow imaging can confirm the presence of an atrial septal defect, false-positive results can occur because of improper gain settings. In addition, caval flow streaming along the right side of the atrial septum can sometimes be mistaken for flow through an atrial septal defect. For these reasons, the use of saline contrast is recommended to either confirm or exclude the presence of a defect. A venous contrast
injection can also demonstrate the biphasic nature of atrial septal defect shunting (Fig. 19.47).

FIGURE 19.41. A secundum atrial septal defect can be difficult to visualize from the four-chamber view (A), even when large. In B, color Doppler clearly shows left-to-right shunting across the atrial septum. Video 19-41a
FIGURE 19.42. A secundum atrial septal defect is often best seen from the subcostal view. In A, the right ventricle is dilated, but distinguishing between a septal defect and dropout is difficult. In B and C, the diagnosis is made with confidence using a combination of two-dimensional imaging (arrow) and color Doppler.
Video 19-42a
coming soon
Video 19-42b
coming soon
FIGURE 19.43. A sinus venosus defect. **A:** This four-chamber view demonstrates a dilated right heart but suggests that the atrial septum is intact. **B:** Color Doppler imaging reveals a defect in the most superior portion of the atrial septum, near the entrance of the superior vena cava (arrow). **C:** Flow through anomalous pulmonary vein as it enters the left atrium at the site of the defect (arrows).
As a next step, quantitation of shunt size can be determined with Doppler techniques. This assessment requires determination of left and right ventricular stroke volume, which can be derived from aortic and pulmonary flow velocity profiles. In children, this method has been used to estimate the direction and magnitude of the shunt (i.e., the net shunt ratio or \( \frac{Q_p}{Q_s} \)). Correlation between Doppler imaging and catheterization techniques for this measurement is good. In adults, however, technical problems limit the accuracy and utility of this approach.

Contrast echocardiography is a valuable method for detecting and characterizing intracardiac shunting. The apical four-chamber view usually is optimal because it allows simultaneous visualization of all four chambers. After intravenous injection of agitated saline, the right side of the heart is rapidly and completely opacified. The demonstration of contrast echoes in the left atrium suggests right-to-left shunting at the atrial level (Fig. 19.48). This phenomenon occurs both in the presence and absence of elevated pressure in the right side of the heart, even when the predominant shunt is left to right. The magnitude of this shunt, however, is often small and transient and may easily be missed.

Contrast-containing blood within the left atrium also occurs in the presence of a pulmonary arteriovenous malformation. Direct evidence of a left-to-right shunt relies on the appearance of noncontrast-containing blood within the right atrium (a so-called negative contrast effect). Unfortunately, noncontrast-enhanced blood may enter the right atrium across an atrial septal
defect, via the coronary sinus, through a left ventricle-to-right atrium communication, or from the inferior vena cava. Slow motion and frame-by-frame analysis of the echocardiogram is necessary to distinguish among these possibilities. It should be recognized that contrast echocardiography has certain limitations for detecting atrial septal defects. First, the method is not quantitative. Shunting is a transient phenomenon reflecting the instantaneous pressure gradient across the atrial septum. The appearance of right-to-left shunting should not be misconstrued as evidence of pulmonary hypertension. Conversely, an apparent “negative” contrast effect within the right atrium must be analyzed carefully to avoid false-positive results. Finally, evidence of shunting at the atrial level may occur with a patent foramen ovale and does not by itself confirm the presence of an atrial septal defect. These concepts are also discussed in Chapter 3.

FIGURE 19.44. From the apical four-chamber view (A), marked dilation of the right atrium and right ventricle is evident, but the atrial septum appears intact. B: By superior angulation of the scan plane, color Doppler imaging (arrow) was able to demonstrate a sinus venosus defect. 

RV, LV, RA, LA
The most accurate technique for evaluating the integrity of the interatrial septum is transesophageal echocardiography. The proximity and orientation of the septum relative to the esophagus permit the entire structure to be adequately visualized in virtually every patient (Fig. 19.49). The presence, location, and size of the defect can be determined with confidence. When percutaneous device closure is contemplated, the test is often required to accurately size the defect and to determine the feasibility of successful closure. Atrial septal defects are not necessarily round, so their dimensions should be measured in multiple planes to ensure proper sizing. Figure 19.50 is an example of incremental information provided by the transesophageal study. In this patient, a secundum defect was detected on a chest wall study and device closure was planned. The presence of a second atrial septal defect was confirmed with the transesophageal echocardiogram, and the plan was altered accordingly. In addition, transesophageal echocardiography is often used when contrast echocardiography demonstrates shunting, but a defect cannot be visualized on transthoracic imaging. In this situation, the transesophageal approach is necessary to differentiate between a patent foramen ovale and a true atrial septal defect. Thus, for the diagnosis of an atrial septal defect, the sensitivity of transesophageal echocardiography approaches 100%. Figure 19.51 shows a large atrial defect evaluated with transesophageal echocardiography. Very little atrial septal tissue is present, creating what is essentially a single, common atrium. As would be expected, significant pulmonary hypertension is documented (see Fig. 19.51C).
In adult patients, transesophageal echocardiography is particularly advantageous in the assessment of sinus venosus defects. This is primarily because these defects are the ones most likely to be missed on a transthoracic study. In addition, the possibility of partial anomalous pulmonary venous drainage is best evaluated using this technique. Typically, the right upper pulmonary vein will drain into the confluence created by the septal defect and the entrance of the superior vena cava. Although this can usually be seen in children from a chest wall study, in adults, this determination is rarely possible without resorting to transesophageal imaging. Figure 19.52 provides an example of sinus venosus atrial septal defect detected using transesophageal echocardiography. Note the relationships among the defect, the superior vena cava, and the superior rim of the atrial septum. Figure 19.53 is another example of a sinus venosus defect recorded with transesophageal three-dimensional imaging.

**FIGURE 19.45.** To make a definitive diagnosis of atrial septal defect with transthoracic echocardiography, both saline contrast (A) and color Doppler (B) should be utilized. In A, a four-chamber view during saline contrast injection shows bubbles traversing the interatrial septum and flowing into the left ventricle. In B, a subcostal four-chamber view illustrates the challenge of diagnosing the defect with two-dimensional imaging (left). However, on the right, using color Doppler, a clear left-to-right flow is recorded crossing the septum.
Video 19-45a

Video 19-45b
Diagnosis of an ostium primum atrial septal defect is easily accomplished with two-dimensional echocardiography. Such defects result from failure of partitioning of the atrioventricular canal and frequently involve the ventricular septum as well. Thus, an ostium primum defect may occur alone (partial atrioventricular canal) or in association with defects in the inlet ventricular septum (complete atrioventricular canal or endocardial cushion defect). Absence of tissue in the most inferior portion of the atrial septum (at the level of insertion of the septal leaflets of the atrioventricular valves) is diagnostic and serves to distinguish ostium primum from secundum defects.
This determination can be made from any of several views, although the apical four-chamber view is often best (Figs. 19.54 and 19.55). The presence of any atrial septal tissue above the base of the atroioventricular valves excludes the diagnosis of a primum defect. Complete atroioventricular canal defects are also associated with a lack of separate fibrous atroioventricular valve rings. As a consequence, both atroioventricular valves lie in the same plane (rather than more apical displacement of the tricuspid valve). This finding is also readily apparent from the four-chamber view. To fully characterize the extent of the defect, transesophageal imaging is usually required (Fig. 19.56). This allows complete assessment of the atrial and ventricular septum as well as the mitral and tricuspid valves. In this example, transthoracic imaging demonstrated the septal defect, but transesophageal imaging was required to fully characterize the atroioventricular valves, which is essential for surgical planning.

Once an ostium primum atrial septal defect is detected, it is essential to assess for the presence of associated abnormalities, including: (1) an inlet ventricular septal defect; (2) a cleft mitral valve; (3) the presence and severity of atroioventricular valve regurgitation; and (4) partial attachment of the septal leaflet of the mitral valve to the interventricular septum. Cleft mitral valve, often seen in the presence of an ostium primum defect, is detected more easily from the parasternal short-axis view by careful scanning the tips of the mitral leaflets (Fig. 19.57). The cleft will generally be recognized as a gap at approximately the 12 o’clock position. Mitral regurgitation is invariably present and often oriented in an eccentric direction.

The management of patients with an atrial septal defect continues to evolve. A key factor in clinical decision making is the presence and severity of pulmonary hypertension. Figure 19.58 is an example of a large secundum defect in a middle-aged woman. The study demonstrates significant enlargement of the right side of the heart and evidence of severe pulmonary hypertension. In Figure 19.59, a dilated, hypertrophied, and hypokinetic right ventricle is present. Doppler demonstrates significantly elevated pulmonary artery pressure (Fig. 19.59B and C). Both surgical repair and percutaneous techniques are now available for closing atrial septal defects. Many patients are able to undergo surgery without the need for cardiac catheterization, based on a thorough echocardiographic assessment. Echocardiography plays a vital role in the percutaneous approach to atrial septal defect closure (Fig.
In these patients, transesophageal echocardiography is critical for selecting candidates for repair based on the size and location of the defect as well as the presence of an adequate rim of septal tissue to allow stabilization of the device. Three-dimensional echocardiography has also become a valuable tool for sizing atrial septal defects and also provides accurate information about the rim of septal tissue that surrounds the defect. Then, during the procedure to close the defect, either transesophageal or intracardiac echocardiography can be used to guide device deployment and to determine success (Fig. 19.61). Figure 19.62 is a transesophageal three-dimensional echocardiogram used to guide percutaneous closure of a defect.

**Ventricular Septal Defect**

This lesion is one of the most common cardiac anomalies encountered in the pediatric population. The interventricular septum is composed of a membranous portion and a muscular portion (Fig. 19.63). The membranous septum is small and located directly below the aortic valve. Its right ventricular surface is adjacent to the septal leaflet of the tricuspid valve. On the left, the membranous septum forms the superior border of the left ventricular outflow tract. The remainder of the interventricular septum is composed of muscular tissue that extends out from the membranous septum in an inferior, apical, and anterior direction. Three regions are identified: the inlet septum (lying posterior to the membranous septum and between the two atrioventricular valves), the trabecular septum (extending from the membranous septum toward the cardiac apex), and the outlet or infundibular septum (extending anteriorly from the membranous septum and lying above the trabecular septum and below the great arteries). The outlet septum straddles the crista supraventricularis.
Saline contrast demonstrates bidirectional shunting through a secundum atrial septal defect. In A, the arrow indicates noncontrast-containing blood flowing from the left atrium to the right. In B, right-to-left shunting is demonstrated (arrow). Such phasic changes in flow direction are largely respiratory cycle dependent.
FIGURE 19.48. Contrast echocardiography can be used to demonstrate intracardiac shunting through an atrial septal defect. In this example, sequential images after intravenous contrast injection demonstrate the appearance of bubbles in the right side of the heart. A negative contrast effect is indicated by the arrow (A). Sequential frames (B–D) show increasing opacification of the right heart as more contrast-containing blood flows across the defect.
Ventricular septal defects are rarely limited to the membranous septum but more often extend into one of the three muscular regions. To describe such defects, the designation “perimembranous” is preferred to “membranous.” Perimembranous defects are by far the most common variety of ventricular septal defect, accounting for approximately 80% of all cases. Next most common are the trabecular ventricular septal defects, which may be multiple and vary considerably in size and location. Defects of the inlet and outlet septa are less common. Inlet ventricular septal defects occur infrequently in isolation but may be a component of endocardial cushion defects. Outlet ventricular septal defects, when they abut both semilunar valves, are referred to as supracristal or doubly committed subarterial defects. These anatomic distinctions have important clinical implications with regard to the chance of spontaneous closure, the surgical approach, risk of conducting system involvement, and likelihood of associated valvular dysfunction (e.g., aortic regurgitation).

The accuracy of echocardiography for detecting a ventricular septal defect depends on its size and location. The ventricular septum is curved and therefore does not lie in a single plane. Multiple views are required to examine the entire septal region, and a single imaging plane will neither interrogate the complete structure nor detect every defect (Fig. 19.64). Visualization of a ventricular septal defect in more than one imaging plane is the most direct means of diagnosis. In general, false-negative findings are more common than false-positive results. The sensitivity of two-dimensional
echocardiography for diagnosis of a ventricular septal defect depends on location. Sensitivity is highest for inlet and outlet defects (approaching 100%), slightly less for perimembranous defects (80% to 90%), and least for trabecular defects (as low as 50% in some earlier studies but considerably higher with modern equipment and techniques). The reasons for this low detection rate are that trabecular defects can occur anywhere within a fairly large area, are sometimes small, and may be multiple. Furthermore, the shape of the defect is often complex, and the orifice may be obscured in systole because of myocardial contraction.

Perimembranous defects are visible in the parasternal long- and short-axis views but generally are not seen from the four-chamber view. Slight medial angulation of the long-axis plane is required to record this area. When this adjustment is done, the membranous septum is located superior to and just below the aortic valve. From this perspective, however, distinguishing between perimembranous and outlet defects (both above and below the crista supraventricularis) may not be possible. For this purpose, the short-axis view is superior. When the scan plane is oriented just below the aortic annulus, both the membranous and outlet septa are visualized. Perimembranous defects are located medially, usually near the septal leaflet of the tricuspid valve (Figs. 19.65 and 19.66).
FIGURE 19.49. A large secundum atrial septal defect with right ventricular enlargement is shown. In A, transthoracic echocardiography reveals a dilated right heart with right ventricular hypokinesis. In B–D, three transesophageal echocardiographic images show the details of the defect at 0, 30, and 90 degrees. In E, an en face three-dimensional view of the defect is provided. This is essential to size the defect and to determine the feasibility of percutaneous closure.
coming soon

Video 19-49a

coming soon

Video 19-49b

coming soon
FIGURE 19.50. A: This transesophageal echocardiogram demonstrates two separate small secundum atrial septal defects (arrows). B: Left-to-right shunting is confirmed with color flow imaging (arrows).
Outlet defects are more anterior and leftward, relative to the aortic annulus (Figs. 19.67 and 19.68). The short-axis view further permits classification of outlet defects as being either above or below the crista supraventricularis. Defects below the crista are to the right of midline, whereas supracristal ventricular septal defects are far leftward and adjacent to the pulmonary valve (Fig. 19.69). Supracristal defects are optimally detected from a high parasternal long-axis or parasternal short-axis view. In the long-axis plane, lateral angulation and rotation permit visualization of both the aortic and pulmonary valves, with the defect adjacent to both. Supracristal defects are often relatively small and may be missed, particularly, if color flow imaging is not used. Once detected, a careful interrogation of the aortic valve is mandatory to exclude cusp prolapse and associated aortic regurgitation. This finding may be accompanied by Valsalva sinus enlargement, usually involving the right sinus. Figure 19.70 is an example of a supracristal ventricular septal defect with associated pulmonic stenosis. In this case, a combination of valvular and supravalvular stenosis was present. Significant pulmonic regurgitation was also noted (see Fig. 19.70D).
FIGURE 19.51. A very large secundum atrial septal defect is demonstrated using transesophageal echocardiography. A: From the four-chamber view, only a small portion of primum atrial septum (arrow) is present and both the right atrium and right ventricle are markedly enlarged. B: By angling rightward, the very large septal defect is apparent. C: A high-velocity tricuspid regurgitation jet confirms severe pulmonary hypertension.
FIGURE 19.52. Transesophageal echocardiography is often required to detect and characterize a sinus venosus defect in adult patients. **A:** The defect is visualized at the junction of the superior vena cava. Flow through the defect is confirmed with color Doppler imaging (**B**). 

The apical four-chamber view permits visualization of both the inlet and trabecular ventricular septum. By tilting the scanning plane inferiorly, the inlet portion of the septum is imaged in the area between the atrioventricular valves. In infants and young children, scanning anteriorly also allows recording of the outlet portion. Although the septum is parallel to the beam in this projection, the four-chamber view is ideal for detecting inlet ventricular septal defects. This view should also be used to assess the relative position of
the two atrioventricular valves. In the presence of an uncomplicated inlet ventricular septal defect, the normal apical displacement of the tricuspid valve is preserved. If both valves are in the same plane, an atrioventricular canal defect should be considered. Because most inlet defects are large, care must be taken to avoid confusing this lesion with a double-inlet left ventricle. Figure 19.71 is an example of a large defect extending from the outlet to inlet portion of the septum. The patient was unrepaired and had Eisenmenger syndrome. There was no evidence of an atrial septal defect.

Malalignment between the septa can also be detected from the four-chamber view (Fig. 19.72). When the atrial and ventricular septa are not aligned, it is essential that the chordal attachments of the atrioventricular valves are carefully assessed. It is crucial to differentiate between a straddling atrioventricular valve (in which some chordae traverse the defect to insert into the opposite ventricle) and an overriding valve (which overlies the defect but has no chordae extending through to the opposite ventricle). In the former case, the presence of chordae crossing the defect greatly complicates surgical repair (Fig. 19.73). Chordal attachments crossing an inlet ventricular septal defect may obscure the defect, leading to a false-negative interpretation. Figure 19.56 is another example of an atrioventricular canal with a portion of the mitral valve overriding the defect.
FIGURE 19.53. A sinus venosus atrial septal defect is evaluated with transesophageal echocardiography. In A, flow through the defect is demonstrated. In the left panel, the relationship of the superior vena cava (#) and right upper pulmonary vein (*) are shown. In B, the arrow indicates the entrance of the pulmonary vein draining into the confluence created by the septal defect. In C, further angulation of the probe demonstrates SVC flow entering the right atrium (arrow). [Video]
coming soon

Video 19-53a
FIGURE 19.54. A primum atrial septal defect is demonstrated on transthoracic echocardiography. Note the location of the defect (arrow) relative to the septal leaflets of the mitral and tricuspid valves.
Defects in the trabecular, or muscular, portion of the muscular septum may be difficult to record with two-dimensional echocardiography. All available imaging planes should be used to exclude the possibility of small defects in this region (Fig. 19.74). Trabecular defects may appear as narrow, irregular channels through the muscular septum. Thus, the orifice on one side of the septum may be displaced from the orifice on the other side, precluding visualization of the entire course in one plane. Once a trabecular defect is identified, it is essential to recognize the possibility of multiple defects and a careful search should be undertaken. Figure 19.75 is an example of a large muscular defect in the mid portion of the septum.

Whenever a ventricular septal defect is suspected, Doppler imaging is crucial as an aid in diagnosis and to characterize the flow direction and velocity. Flow through a small restrictive ventricular septal defect is recorded with Doppler imaging as a turbulent, high-velocity systolic jet crossing the septum from left to right. To detect such jets, the right ventricular septal surface is carefully and systematically scanned with color Doppler imaging. Small defects appear as thin, high-velocity jets of turbulent flow within (and on the right ventricular side of) the septum (Fig. 19.76).

Larger defects are characterized by a wider jet when imaged with color Doppler imaging (Fig. 19.77). When the location of the defect is unknown, the left parasternal, apical, and subcostal windows should be used for screening. Once the jet is identified, the Doppler beam can be oriented parallel to flow to permit recording of the peak jet velocity. With restrictive
defects, the jet velocity is high, reflecting the high-pressure gradient between the ventricles during systole (Fig. 19.78). With larger defects, the pressure gradient is less, and, hence, the jet velocity is lower. In the presence of a large ventricular septal defect and elevated right ventricular pressure, there may be relatively little flow across the defect. The flow can be assessed by using pulsed Doppler and color flow imaging and indicates the presence of Eisenmenger physiology. Figure 19.79 includes two examples of jet velocity through a ventricular septal defect. In the first example, a small perimembranous defect is associated with a high jet velocity, indicating low right ventricular systolic pressure. In the second case, a large muscular defect has led to Eisenmenger syndrome and a resulting lower transseptal gradient and jet velocity.
FIGURE 19.55. Systolic (A) and diastolic (B) frames of the apical four-chamber view from a patient with a large primum atrial septal defect (arrows) and partial atrioventricular canal. Note that the mitral and tricuspid valves arise at the same level and the entire primum septum is absent. In C, a large left-to-right shunt (arrow) is apparent.
The pressure gradient (PG) between the ventricles can be estimated using the modified Bernoulli equation:

\[
\text{PG (mm Hg)} = 4 \times (\text{peak velocity})^2 \quad \text{[Eq. 19.1]}
\]

If the systolic blood pressure is determined by cuff recording of the upper extremity and no left ventricular outflow tract obstruction is present, the left ventricular (LV) systolic pressure can be determined. Then, right ventricular (RV) systolic pressure is calculated from the equation(s):

\[
\text{PG} = \text{LV (systolic) pressure} - \text{RV (systolic) pressure}, \quad \text{or} \quad \text{[Eq. 19.2]}
\]

\[
\text{RV pressure} = \text{LV pressure} - \text{PG}, \quad \text{or by substitution} \quad \text{[Eq. 19.3]}
\]

\[
\text{RV pressure} = \text{cuff systolic blood pressure} - [4 \times (\text{peak velocity})^2] \quad \text{[Eq. 19.4]}
\]

In the absence of right ventricular outflow tract obstruction, this value is equal to the pulmonary artery systolic pressure. Thus, a noninvasive estimate of the presence and severity of pulmonary hypertension can be made. Alternatively, right ventricular systolic pressure can be calculated from the peak velocity of the tricuspid regurgitation (TR) jet using a similar equation (Fig. 19.80):
FIGURE 19.56. A patient with complete atrioventricular canal is evaluated with transthoracic and transesophageal echocardiography. A: From the apical four-chamber view, the defect is poorly characterized at the level of the inlet septum (arrow). B: Color Doppler imaging was unable to fully characterize the shunt. C: Using transesophageal imaging, the extent of the abnormality is better appreciated. In diastole, the common atroventricular valve (white arrows) straddles the defect. The primum atrial septal defect is indicated by the arrowhead. In systole, the inlet ventricular septal defect is indicated by the arrow.
FIGURE 19.57. Primum atrial septal defect is often associated with a cleft mitral valve. A: The mitral orifice is demonstrated from the short-axis view. B: By scanning slightly more apically, the cleft in the anterior leaflet is demonstrated (arrow). C: Such patients often have a posteriorly directed jet of mitral regurgitation.

FIGURE 19.58. Severe pulmonary hypertension developed in this patient with a large secundum atrial septal defect. A: Absence of tissue in the region of the atrial septal is evident and the right side of the heart is dilated. B: Color Doppler imaging demonstrates both tricuspid regurgitation (mosaic pattern) and low-velocity systolic flow (in red) through the defect. C: High-velocity tricuspid regurgitation is demonstrated, indicating a right ventricular systolic pressure of
greater than 100 mm Hg.
FIGURE 19.59. A patient with an unrepaired atrial septal defect and Eisenmenger physiology is shown. In A, the right ventricle is dilated, hypertrophied, and hypokinetic. In B, the tricuspid regurgitation jet has a peak velocity of 4 m/s, indicating a right ventricular systolic pressure of 64 mm Hg. In C, pulmonic regurgitation is present with an end-diastolic velocity (arrow) of more than 2 m/s, indicating elevated pulmonary artery diastolic pressure.
RV systolic pressure = RA pressure + [4 × (TR velocity)²]  [Eq. 19.5]

By using one or both of these approaches, an accurate measure of right ventricular pressure can be obtained in most patients.

A variety of associated lesions or complications occur in the setting of a ventricular septal defect, most of which are readily detected using echocardiography. Among the most common is the ventricular septal aneurysm, a thin membrane of tissue that usually arises from the margin of the defect, sometimes by incorporation of a portion of tricuspid septal leaflet tissue. Such aneurysms are commonly associated with perimembranous ventricular septal defects. Although aneurysms are usually patent, they represent one mechanism for spontaneous closure of a ventricular septal defect. The parasternal long- and short-axis views are most useful in detecting a ventricular septal aneurysm (Fig. 19.81). They are seen as thin, membranous pouches that bulge through the defect often with a windsock appearance. They may be highly mobile, often protruding through the defect into the right ventricle during systole. Once detected, they should be interrogated with color flow imaging (Fig. 19.82) to determine the patency of the aneurysm. If the tricuspid valve is involved, the presence and severity of associated tricuspid regurgitation should be determined.
FIGURE 19.60. Percutaneous closure of an atrial septal defect using an Amplatzer® device is demonstrated in two patients. Such devices appear on echocardiography as echogenic structures within the area of the atrial septum. A: Two devices were needed to occlude two separate defects. Color Doppler imaging can be used to detect residual shunting across the defects. B: A single device is indicated by the arrows.

FIGURE 19.61. A–F: During device closure of an atrial septal defect, intracardiac echocardiography is often used to guide deployment of the device. This series of echocardiograms demonstrates placement of an Amplatzer closure device across
a secundum atrial septal defect. After the left atrial device is positioned, the structure is secured against the atrial septum before the right atrial component is engaged. Then, the deployment catheter is released, allowing the device to straddle the septum and obscure the defect. See text for details. (continued)

An unusual type of ventricular defect involves a direct communication between the left ventricle and the right atrium, sometimes called a Gerbode defect. This can occur because the more apically positioned septal leaflet of the tricuspid valve creates a small region of septum between the left ventricle and the right atrium (Fig. 19.83). In the illustration provided, the septal defect can be seen below the aortic valve but above the tricuspid valve. Color Doppler imaging demonstrates a degree of left-to-right shunting that enters both the right atrium and the ventricle.

Another complication associated with ventricular septal defects is aortic regurgitation, which occurs most commonly with outlet defects in which the support of the valve is undermined by an absence of myocardium below the annulus (Fig. 19.84). Perimembranous defects are also associated with aortic regurgitation. Prolapse of an aortic cusp through the defect occasionally is recorded. The finding of aortic regurgitation in a patient with a ventricular septal defect has important implications. Surgical closure is often recommended, even in the absence of a large shunt, to reduce the risk of progressive aortic valve dysfunction.
FIGURE 19.62. A secundum atrial septal defect is closed using an Amplatzer device. **A:** The atrial septal defect (arrow) is visualized from transesophageal imaging. **B:** Left-to-right shunting through the defect is demonstrated with color flow imaging. **C:** Using three-dimensional imaging multiple occluder devices are demonstrated across the defect, still attached to their delivery catheters (arrows). **D:** The spatial relationship between the three deployed occluder devices and the rim of atrial septal tissue that they straddle (arrow) is well visualized with the
three-dimensional approach. E: All three devices are demonstrated.

FIGURE 19.63. Schematic of the right ventricular surface of the interventricular septum diagramming common locations of ventricular septal defects. FO, foramen ovale; PM, papillary muscle; RAA, right atrial appendage; region 1, membranous interventricular septum; region 2, outflow interventricular septum; region 3, trabecular septum; region 4, inflow septum; region 5, subarterial region; region 6,
distal multiple “Swiss cheese” septal defects.

FIGURE 19.64. Schematic diagram of the location of the various types of ventricular septal defect when viewed using two-dimensional echocardiography. See text for details.

FIGURE 19.65. A perimembranous ventricular septal defect. The long-axis view is angulated medially to record the membranous septum (A). In the basal short-
axis view, the location of the defect can be seen relative to the aortic annulus and the tricuspid valve (B).
FIGURE 19.66. A perimembranous ventricular septal defect is demonstrated with color flow imaging from the long-axis (A) and short-axis (B) views. C: Continuous-wave Doppler imaging demonstrates a peak pressure gradient between the left and right ventricles of greater than 110 mm Hg.
FIGURE 19.67. An outlet type of ventricular septal defect. A: From the long-axis view, note the similarity between this type of defect and a perimembranous defect. The distinction is apparent from the short-axis views (B, C). The defect is more anterior and leftward (arrow) relative to the tricuspid valve. D: A high-velocity jet confirms that the defect is small and restrictive with normal right heart pressure.

Video 19-67
FIGURE 19.68. An example of an outlet ventricular septal defect is visualized in the long-axis (A) and basal short-axis views (B).
A supracristal ventricular septal defect is recorded in a young child. In A, a basal short-axis view reveals a high velocity left-to-right jet just below the aortic annulus and leftward, adjacent to the pulmonary valve (PV). Panels B through E were recorded with transesophageal echocardiography. In B, a short-axis view again shows the close relationship between the defect (small arrow) and pulmonary valve. Shunt flow through the defect is then demonstrated in C. In D, a long-axis view also shows left-to-right shunting through the septal defect (arrow). Mild aortic regurgitation, a common finding in supracristal defects, is demonstrated in panel E. (Courtesy of C. Cua, MD.)
After surgical repair, echocardiography can be used to determine the integrity of the ventricular septal defect patch (Fig. 19.85). Color flow imaging is the most sensitive technique for detection of a residual shunt, which is recorded as a turbulent, high-velocity jet at the periphery of the patch (Fig. 19.86). The width of the jet has been correlated with the magnitude of the shunt and the likelihood of the need for reoperation. Percutaneous closure of ventricular septal defects is now possible. Figure 19.87 is an example of closure of a perimembranous defect using an Amplatzer device.

**Endocardial Cushion Defect**

Division of the common atrioventricular canal into left and right sides occurs by fusion of the superior and inferior endocardial cushions. Failure to do so results in an atrioventricular septal defect with various combinations of ostium primum atrial septal defect, inlet ventricular septal defect, and structural abnormalities of the atrioventricular valves. Thus, an endocardial cushion defect is a spectrum of lesions including partial atrioventricular canal (implying separate atrioventricular orifices), complete atrioventricular canal (a common atrioventricular orifice), and isolated inlet ventricular septal defect.
FIGURE 19.70. A patient with a supracristal ventricular septal defect and right ventricular outflow tract obstruction. From the parasternal long-axis view (A) turbulent flow is seen within the right ventricular outflow tract, but no clear left-to-right shunt is recorded. B: From the basal short-axis view, just below the aortic annulus, the defect can be seen in the area between the aortic and pulmonic valves (arrow). The presence and location of this defect is more convincingly demonstrated using color Doppler imaging (C) with significant left-to-right shunting. D: Doppler imaging demonstrates a peak gradient of 52 mm Hg due to a combination of valvular and subvalvular pulmonic stenosis.
FIGURE 19.71. A large, unrepaired inlet ventricular septal defect is shown. The defect (see arrows) extends from the inlet to outlet regions of the septum, and can be seen in both the long-axis (A) and four-chamber (B) views. In C, the short-axis view demonstrates septal flattening in systole, due to right ventricular pressure overload. [Video 19-71a] [Video 19-71b]
FIGURE 19.72. A large inlet ventricular septal defect with associated malalignment of the septum (arrow). The abnormal relationship between the ventricular septum and the aortic valve is demonstrated in this four-chamber view.
FIGURE 19.73. An inlet ventricular septal defect in association with atrioventricular canal. Note the presence of chordae crossing the defect (arrow). A large primum atrial septal defect is also noted.

Video 19-73

FIGURE 19.74. A ventricular septal defect involving the midportion of the
interventricular septum is demonstrated. The long-axis (A), four-chamber (B), and short-axis (C) views are shown. Color Doppler reveals left-to-right shunting through the defect. 

Video 19-74a

coming soon

Video 19-74b

coming soon
A large muscular ventricular septal defect is associated with right ventricular enlargement and hypertrophy. The defect is recorded in the long-axis (A) and four-chamber (B) views.

FIGURE 19.75. A large muscular ventricular septal defect is associated with right ventricular enlargement and hypertrophy. The defect is recorded in the long-axis (A) and four-chamber (B) views.
FIGURE 19.76. Small ventricular septal defects may not be apparent on two-dimensional imaging (A), but their presence can be confirmed using color Doppler imaging (B). In this example, the septum appears intact, but medial angulation and the use of color Doppler imaging confirm the presence of a small defect.
Two-dimensional echocardiography permits detailed assessment of virtually every morphologic feature of endocardial cushion defect. The primum portion of the atrial septum, the inlet ventricular septum, atrioventricular valve morphology, ventriculoatrial septal malalignment, and ventricular outflow tract obstruction can be accurately assessed (Fig. 19.88). The four-chamber view generally yields the most diagnostic information on this entity (Fig. 19.89). Importantly, the presence and size of the atrial and ventricular septal defects can be determined and the anatomy of the atrioventricular valves can be assessed. Because the valve leaflets move freely within the defect, accurate assessment of these features requires real-time imaging. During systole, the atrioventricular valve assumes a basal position, obscuring the primum atrial septal defect but permitting assessment of the size of the inlet ventricular septal defect and the presence of atrioventricular valve regurgitation. As the valve opens in diastole, the atrial portion of the defect can be examined. Chordal attachments and the presence of straddling (Fig. 19.90) can also be determined. Although atrioventricular valve regurgitation can be detected from the four-chamber view (Fig. 19.91), the presence of a cleft anterior mitral valve leaflet is better recorded from the parasternal short-axis view (Fig. 19.92). The short-axis view also permits visualization of both the atrial and the ventricular septal defects. In the four-chamber view, the presence of left ventricle-to-right atrial shunting can be detected by using color flow imaging.
Because of the broad spectrum of anomalies that may occur in the setting of an endocardial cushion defect, echocardiography plays a major role in determining the feasibility of surgical repair. Specifically, the relative size of the ventricles, the presence of septal malalignment, and the extent of the atrial and ventricular communications should be established. The morphology of the atrioventricular valves is also critical in planning reparative surgery. Echocardiography allows the anatomy of the valves and their chordal insertions to be determined. The presence of a straddling or overriding valve and the degree of valvular regurgitation can also be assessed. During surgery, the use of transesophageal echocardiography permits assessment of the
adequacy of repair. Most importantly, the presence and severity of residual atrioventricular valve regurgitation can be determined.

**ABNORMAL VASCULAR CONNECTIONS AND STRUCTURES**

**Patent Ductus Arteriosus**

The ductus arteriosus is the normal fetal vascular channel that connects the descending aorta and the main pulmonary artery, providing a conduit for blood from the right ventricle to the thoracic aorta. Failure of the ductus to close shortly after birth is abnormal, giving rise to the term patent ductus arteriosus. This persistent patency of the ductus may be desirable or undesirable, depending on the presence of other associated anomalies. For example, in the presence of pulmonary atresia, the persistent patency of the ductus may be the only source of pulmonary blood flow. Expedient and accurate detection of this vascular channel has profound implications for the critically ill newborn. Later in life, patent ductus arteriosus is one of the important causes of a left-to-right shunting and volume overload of the left ventricle. The functional significance of a patent ductus arteriosus depends on the size of the channel, the pulmonary vascular resistance, and the presence and degree of left ventricular dysfunction. Most patent ducti are identified in early childhood and promptly closed, so are not commonly seen in the adult population.
FIGURE 19.78. With proper beam alignment, the pressure gradient across a ventricular septal defect can be measured. These examples demonstrate both high (A, B) and low (C) jet velocities, suggesting either normal or elevated right ventricular pressure, respectively. C: Low-velocity flow through the defect is consistent with only a 25 mm Hg systolic pressure difference between the left and right ventricles. This was recorded from a patient with Eisenmenger syndrome. See text for details.
FIGURE 19.79. Two examples of muscular ventricular septal defects are recorded with transthoracic echocardiography. A: Color Doppler of a small outlet defect demonstrates left-to-right shunting. B: With continuous-wave Doppler, a high-velocity jet indicates a 100 mm Hg gradient across the septum, suggesting low right ventricular systolic pressure. C: From a different patient, the parasternal long-axis view reveals a larger defect. The right ventricle is dilated and hypokinetic. D: Spectral Doppler imaging reveals that the shunt through the defect is low velocity and predominantly right-to-left due to markedly elevated right heart pressure.

Video 19-79a
FIGURE 19.80. A: A large outlet ventricular septal defect is demonstrated, resulting in Eisenmenger syndrome. B: High-velocity tricuspid regurgitation confirms markedly elevated right ventricular systolic pressure. C: Pulsed Doppler recording of pulmonary valve flow is consistent with pulmonary hypertension.
FIGURE 19.81. Short-axis views of a perimembranous ventricular septal defect that had partially closed by adhesions from the septal leaflet of the tricuspid valve. In A and B, two systolic images show the appearance of the highly mobile windsock (arrows) that has partially sealed the defect. In C, color Doppler shows some residual shunting into the right ventricle. Video 19-81 coming soon
FIGURE 19.82. A small perimembranous ventricular septal defect has partially closed forming a windsock structure on the right side of the septum (A, arrows). In B, color Doppler demonstrates a very narrow jet indicating a small residual left-to-right shunt.

Video 19-82a

coming soon
FIGURE 19.83. An unusual type of defect involves direct communication between the left ventricle and the right atrium. **A:** The presence of a defect is suggested from this subcostal view (arrow). **B:** Color Doppler imaging confirms left-to-right shunting from the left ventricle into both the right atrium and the right ventricle. The images are inverted, as is customary in many pediatric echocardiography laboratories.
FIGURE 19.84. A: A supracristal ventricular septal defect (arrow) is detected using color Doppler imaging. B: Associated aortic regurgitation is demonstrated (arrows). C: Doppler imaging confirms a high-velocity jet through the defect, suggesting an 80 mm Hg transseptal pressure gradient. D: Continuous-wave Doppler imaging of the aortic regurgitation jet.

FIGURE 19.85. Following repair of a perimembranous defect, this patient
presented with dyspnea. In the parasternal long-axis view (A), reduced systolic function was noted and the patch is indicated by the arrow. In B, with color Doppler, mild aortic regurgitation is noted, but no evidence of residual shunting is present.

Both echocardiography and Doppler imaging are crucial in the assessment of patients with patent ductus arteriosus. The first step in imaging a ductus is knowing where to look for it. The pulmonary arterial end of the ductus is located to the left of the pulmonary trunk and adjacent to the left pulmonary artery. The aortic insertion is opposite to and just beyond the origin of the left subclavian artery. The aortic orifice of the channel is usually larger than the pulmonary end, giving the ductus a funnel shape. For direct visualization, the
suprasternal and high parasternal short-axis views are used. In the parasternal short-axis view, angling the imaging plane in a leftward and superior direction allows visualization of the bifurcation of the pulmonary artery (Fig. 19.93). Clockwise rotation permits recording of a greater length of the descending aorta so that the entire ductus may be visualized. From the suprasternal window, the ductus is seen as a narrow channel extending from the inferior border of the aorta to the pulmonary trunk. Unfortunately, this view has significant limitations, particularly in adults. The ductus can be recorded directly in only a few patients and care must be taken to avoid mistaking the left pulmonary artery for a large ductal channel. In addition, the ductus is often aligned such that it is parallel to the ultrasound beam and is therefore subject to the limitations of lateral resolution.

**FIGURE 19.86.** A large perimembranous ventricular septal defect was repaired surgically. In A, a large patch (arrows) can be seen from the parasternal long-axis view. From the short-axis view (B), the patch is again visualized (arrows) and color Doppler demonstrates a residual left-to-right shunt. Continuous-wave Doppler (C) shows that the peak velocity of the jet is 3.5 m/s, although alignment is suboptimal and maximal velocity may be underestimated.
Video 19-86a

coming soon

Video 19-86b

coming soon
Doppler imaging improves the diagnostic sensitivity by directly visualizing left-to-right flow through the channel. In ducts too small to be detected with two-dimensional echocardiographic imaging, a narrow jet of turbulent flow in the proximal pulmonary artery may be the first indication of a patent ductus arteriosus. This flow is usually best seen from the high parasternal short-axis view as a retrograde mosaic jet entering the distal pulmonary artery from the posterolateral direction (Figs. 19.94 and 19.95). The orientation of the jet within the pulmonary artery varies, and distinguishing it from normal pulmonary flow or pulmonary regurgitation
may require slow-motion and freeze-frame analysis.

**FIGURE 19.88.** A complete atrioventricular canal is shown. In systole (A), a common AV valve floats within a large defect involving both the inlet ventricular septum and the primum atrial septum. In B (diastole), when the AV valve opens, the extent of the atrioventricular defect is apparent.

**Video 19-88**

**FIGURE 19.89.** In this patient with an atrioventricular canal defect, (A)
demonstrates spontaneous closure of the ventricular portion of the defect (arrow) via adhesions from the tricuspid valve. No residual ventricular level shunt was present. In B, the primum atrial septal defect remains with color Doppler showing a left-to-right shunt.

Video 19-89

FIGURE 19.90. Complete atrioventricular canal may be associated with a straddling atrioventricular valve. A: Recorded during transesophageal imaging. B: A transthoracic four-chamber view. In both studies, chordae can be seen crossing the inlet defect.
FIGURE 19.91. Atrioventricular valve regurgitation is demonstrated using color flow imaging in this patient with complete atrioventricular canal. Note how the regurgitant jets (arrows) appear to originate from both sides of the atrioventricular valve in a crisscross fashion.
In addition to its role in diagnosis, echocardiography is also used to estimate the magnitude of the shunt and the degree of pulmonary artery hypertension. The left-to-right shunt associated with a patent ductus results in volume overload of the left ventricle. The degree of left atrial and left ventricular dilation is a useful marker of the magnitude of shunting. A dilated and hyperdynamic left side of the heart is an indication of volume overload and, in the absence of other causes, suggests the presence of a significant left-to-right shunt. Doppler imaging also plays a role in this area. In most cases, high-velocity turbulent flow occurs continuously in a left-to-right direction, reaching a peak in late systole (Fig. 19.96). With Doppler imaging, the peak pressure gradient can be calculated by using the modified Bernoulli equation. This method permits a quantitative estimate of pulmonary artery pressure. If the ductus is relatively long (>7 mm), however, the simplified Bernoulli equation may be inaccurate. Bidirectional shunting always implies elevated pulmonary vascular resistance. In this situation, flow occurs from right to left in early systole and from left to right in late systole and diastole. As pulmonary pressure increases, the duration and extent of right-to-left shunt flow in diastole increase.

FIGURE 19.92. From a patient with a primum atrial septal defect, a cleft mitral valve is demonstrated. In the long axis (A), color Doppler demonstrates a mitral regurgitation jet that originates more anterior than usual. This is typical of a cleft valve. In the short axis (B), the cleft is indicated by the arrows.
FIGURE 19.93. A patent ductus arteriosus is recorded from a transthoracic basal short-axis view. The larger arrow indicates the origin of the jet, where the ductus connects to the pulmonary artery. The smaller arrow indicates a separate jet of mild pulmonic regurgitation.
ABNORMAL SYSTEMIC VENOUS CONNECTIONS

A persistent left superior vena cava is the most common congenital anomaly involving the systemic veins. It occurs in approximately 0.5% of the general population and in 3% to 10% of patients with CHD. In most cases, the left superior vena cava drains into the right atrium via the coronary sinus. As such, it has no physiologic consequences (aside from a predisposition to arrhythmias and heart block) and venous return is essentially normal. Less often, it drains into the left atrium or a pulmonary vein, resulting in a right-to-left shunt. Associated lesions, especially defects of the atrial septum, are common. Diagnosis of a persistent left superior vena cava frequently occurs after a dilated coronary sinus is detected with echocardiography. Coronary sinus dilation is usually the result of anomalous drainage to the sinus, either from a persistent left superior vena cava or an anomalous pulmonary vein. Occasionally, the degree of coronary sinus enlargement is so great that the structure is mistaken for something else, such as a pericardial effusion, pulmonary vein, or descending aorta.
FIGURE 19.94. Two examples of patent ductus arteriosus visualized with color flow imaging are demonstrated from the basal short-axis view. A: Note how the jet hugs the lateral wall of the main pulmonary artery (MPA). B: The arrow indicates the entrance of the ductus into the pulmonary artery. DA, descending aorta.

FIGURE 19.95. A patient with a patent ductus arteriosus before (A) and after (B) coil occlusion. After closure of the defect, the shunt is no longer present and only trivial pulmonary regurgitation is apparent. DA, descending aorta; MPA, main pulmonary artery. ⚒️

Video 19-95
FIGURE 19.96. A patent ductus arteriosus is best detected from a high parasternal window using color flow imaging. **A:** Turbulent, high-velocity flow in the proximal pulmonary artery toward the transducer (white arrows) is consistent with a ductus. **B:** Continuous-wave Doppler imaging can then be used to assess the velocity of the jet, an indication of the gradient between the descending aorta and the pulmonary artery.

The coronary sinus is best visualized in the parasternal long-axis view as a circular structure in the posterior atrioventricular groove (Fig. 19.97). Its location anterior to the pericardium distinguishes it from other venous and arterial structures, especially the descending aorta. In the parasternal short-axis view, the coronary sinus can be recorded as a tubular, crescent-shaped structure lying within the atrioventricular groove and communicating with the right atrium. From the four-chamber view, posterior angulation of the beam will demonstrate the coronary sinus in long axis, coursing behind the left atrium and emptying into the right atrium (Fig. 19.98). Occasionally, a Chiari network is seen where the coronary sinus empties into the posterior right atrium.
FIGURE 19.97. A dilated coronary sinus (*asterisk*) is shown in this parasternal long-axis view.
FIGURE 19.98. A dilated coronary sinus is recorded from the apical view. A: The four-chamber view reveals only mild dilation of the right-sided chambers. By directing the ultrasound beam steeper relative to the chest wall (B), the coronary sinus is recorded (asterisk).

FIGURE 19.99. A persistent left superior vena cava is demonstrated from the suprasternal view. A: The vessel is seen just to the left of the aortic arch (AA) and appears connected to the left atrium (arrow). B: Color Doppler imaging demonstrates low-velocity flow directed interiorly into the left atrium. RPA, right pulmonary artery.

Direct visualization of a left superior vena cava is easier in children than in adults. The vessel can be seen from the suprasternal window as a vertical structure to the left of the aortic arch (Fig. 19.99). This view is particularly helpful in determining whether both vena cavae are present, to assess their relative size, and to detect an innominate vein. The connections between the cavae and the atria should also be examined using a combination of two-dimensional and color Doppler imaging. In this example (Fig. 19.99), the drainage of the left superior vena cava into the left atrium is clearly visualized using color flow imaging. Color Doppler imaging may be used to
distinguish higher-velocity arterial flow (which, at usual gain settings, appears as red or blue laminar flow in systole) from venous flow (which is often not detected with color flow imaging). Pulsed Doppler imaging can be used to confirm venous flow by recording low-velocity, phasic flow in a superior-to-inferior direction.

Saline contrast-enhanced echocardiography is of great value in the differential diagnosis of a dilated coronary sinus and to assess abnormal venous caval connections (Fig. 19.100). If injection into the left arm results in opacification of the coronary sinus before the right atrium and ventricle, the diagnosis of a persistent left superior vena cava is established. If the same injection leads to left atrial opacification, abnormal drainage of the vena cava (either left or common) is present. This pattern of drainage is unusual and typically associated with other cardiac lesions. Injection into the right arm should then be performed. In the presence of a left superior vena cava (draining into either the left or the right atrium), this injection should lead to the normal sequence of opacification (i.e., no opacification of the coronary sinus).

**ABNORMALITIES OF THE CORONARY CIRCULATION**

The most important congenital abnormalities involving the coronary circulation include anomalous origin of the coronary arteries and coronary artery fistulae. Coronary artery aneurysms, which may be congenital but more commonly are the result of Kawasaki disease, are also discussed in this section. Anomalous origin of a coronary artery is present in approximately 1% of patients undergoing cardiac catheterization. Origin of the left circumflex artery from the right coronary sinus and origin of the right coronary artery from the left sinus are the most frequently encountered variants. These anomalies are of particular relevance when the course of the aberrant artery passes between the aorta and the pulmonary trunk.
FIGURE 19.100. After contrast injection into a left arm vein, this sequence demonstrates evidence of a persistent left superior vena cava draining into the coronary sinus. A: A dilated coronary sinus is evident (arrow). B: Contrast is seen within the coronary sinus (arrow) before opacification of the right ventricle. C: Bubbles are visualized within the right ventricle (arrow) a few beats later. See text for details.
Although CT angiography is currently the gold standard for the noninvasive imaging of coronary arteries, echocardiography is a common screening test in young patients and may provide the first indication of an anomalous vessel. The ostia and proximal coronary arteries can be imaged with echocardiography from the parasternal short-axis view at the base. This view permits determination of the size and initial course of the arteries. In adults, transesophageal echocardiography generally provides higher-quality
images of the proximal coronary arteries, and anomalous vessels can be identified with a high degree of accuracy. An inability to record the origin of the coronary artery from this view raises the possibility of an aberrant vessel.

Coronary artery anatomy may be especially important in certain forms of complex CHD, such as tetralogy of Fallot and transposition of the great arteries. Here, assessment of coronary artery anomalies and vessel diameter has implications for prognosis and surgical repair. Anomalous origin of the left coronary artery from the pulmonary trunk is one of the causes of heart failure in the neonate. In such patients, the right coronary artery is dilated and the left coronary ostium is absent from the aortic root. The left coronary artery may be visualized but does not connect with the aorta. By using a high parasternal view of the pulmonary trunk (similar to that used to evaluate a patent ductus arteriosus), the vessel can be seen arising from the posterior wall of the pulmonary trunk (Fig. 19.101). Searching for coronary arteries is often easiest using color Doppler imaging.

A coronary artery fistula is a rare anomaly that results from the abnormal connection between a coronary artery and another vessel or chamber (either a coronary vein, pulmonary artery, or the right ventricle). This connection results in a left-to-right shunt and a continuous murmur, which is often confused with a patent ductus arteriosus. Two-dimensional echocardiography reveals dilation of the involved coronary artery that is uniform and often severe. In children, the course of the dilated vessel can be followed by the use of multiple imaging planes and simultaneous color flow imaging. The fistula itself may be difficult to image. Color Doppler imaging and/or contrast-enhanced echocardiography are useful when attempting to follow the path of the vessel (Fig. 19.102). Detection of turbulent flow within the right ventricle or pulmonary artery may identify the site of the fistulous connection (Fig. 19.103). If the left-to-right shunt is large, chamber dilation may also be apparent.

Coronary artery aneurysms usually occur in association with Kawasaki disease. These aneurysms appear as localized dilated segments, usually with a fusiform shape. They often are multiple, may occur anywhere along the vessel, and sometimes are lined with thrombus. Detection requires the use of multiple imaging planes to record as much as possible of the distal arteries. In young patients, the entire left main coronary artery and the proximal segments of the right, left circumflex, and left anterior descending arteries
can be seen from the parasternal short-axis view. The parasternal long-axis view of the right ventricular outflow tract may permit recording of the more distal left anterior descending artery, whereas the apical four-chamber view can be used to assess the left circumflex and right coronary arteries. As noted previously, transesophageal echocardiography can also be used effectively to examine the coronary arteries. The diameter of the coronary artery aneurysms should be measured because the size has prognostic implications. The presence of a pericardial effusion should also be sought. Its presence increases the likelihood of coronary artery aneurysms.

**FIGURE 19.102.** An echocardiogram recorded from a patient with multiple coronary artery fistulae, detected using color Doppler imaging. **A:** Parasternal long-axis view demonstrates a fistulous connection between the right coronary artery and the right ventricle (arrow). **B:** From the apical four-chamber view, multiple fistulae (arrows) can be seen entering the left ventricle along the interventricular septum.
FIGURE 19.103. An example of a coronary artery fistula, with connection between the right coronary artery and the proximal pulmonary artery. Color Doppler imaging demonstrates the jet from the right coronary artery entering the proximal pulmonary artery (arrow). Mild pulmonary regurgitation is also demonstrated in this diastolic frame. Video 19-103
Tetralogy of Fallot is the most common form of cyanotic CHD and is one of the few such lesions that may escape diagnosis until later in life. This anomaly has four anatomic features: (1) anterior and rightward displacement of the aortic root; (2) ventricular septal defect; (3) right ventricular outflow tract obstruction; and (4) right ventricular hypertrophy. The echocardiographic evaluation includes de novo diagnosis of the lesion, a determination of the options for surgical intervention, and postoperative assessment of the adequacy of repair.

The critical developmental defect in tetralogy of Fallot is malalignment of the infundibular septum, resulting in a nonrestrictive infundibular (and sometimes perimembranous) septal defect and overriding of the aorta. Both of these fundamental anatomic features are optimally assessed using the parasternal long-axis view, which permits the viewer to determine the presence of the ventricular septal defect and the degree of aortic overriding (Fig. 19.104). Discontinuity between the infundibular septum and the anterior aortic root is readily apparent. Proper transducer position and angulation are necessary to ensure accurate assessment of the degree of aortic overriding. This feature is variable, ranging from minimal to extreme. In the latter case, the aortic valve may appear to arise exclusively from the right ventricle and resembles a double-outlet right ventricle. Most investigators follow the “50% rule” to make this distinction. If more than 50% of the aorta overlies the left ventricle, the proper designation should be tetralogy of Fallot. If more than 50% of the aorta overlies the right ventricle, double-outlet right ventricle is present.
FIGURE 19.104. From a patient with tetralogy of Fallot, the long-axis view (A and B) shows a large inlet ventricular septal defect with an overriding aorta. A systolic and diastolic frame are shown. From the four-chamber view (C), the defect is again seen adjacent to the AV valves and presence of right ventricular hypertrophy is evident.
The short-axis view allows the echocardiographer to determine the extent and size of the septal defect. More importantly, the right ventricular outflow tract can be assessed. Narrowing can occur on multiple levels. In most cases, it is the displacement of the infundibular septum that produces the subvalvular narrowing that is characteristic of tetralogy of Fallot. In general, the greater the aortic overriding is, the more severe the subpulmonary stenosis. Various combinations of infundibular hypoplasia and muscular hypertrophy may be present. Stenosis may also involve the pulmonary annulus and/or valve. Less often, the proximal pulmonary arteries are hypoplastic, resulting in supra valvular stenosis. In the most extreme situation, pulmonary atresia is present and perfusion of the lungs depends on systemic to pulmonary artery collaterals and a patent ductus arteriosus.

By using the parasternal short-axis and subcostal coronal views, each of these potential levels of obstruction must be carefully evaluated. Color Doppler imaging is often helpful in assessing the location of the narrowed, turbulent flow. Continuous-wave Doppler imaging is then used to determine the pressure gradient across the various levels of obstruction. Determining the size of the pulmonary arteries is important in planning any surgical intervention, and it is best accomplished from the short-axis and suprasternal views. The relative sizes of the right and left pulmonary arteries can be compared. In infants, care must be taken to avoid confusing the left pulmonary artery with a patent ductus arteriosus. The diameter of the right pulmonary artery is best assessed as it passes below the aortic arch (as
recorded from the suprasternal long-axis view). Coronary artery anatomy must also be examined preoperatively, and this assessment generally can be accomplished by using two-dimensional echocardiographic techniques. A coronary artery branch crossing the right ventricular outflow tract (either an aberrant left anterior descending or conus branch) has important implications for surgical repair.

After repair of tetralogy of Fallot, echocardiography plays a key role in assessing the surgical results. Figure 19.105 is from an 80-year-old man who had undergone repair 35 years ago. There was no gradient across his right ventricular outflow track and only trivial pulmonary regurgitation. From the parasternal long-axis view, the ventricular septal defect patch is seen as a linear structure passing obliquely from the septum to the anterior aortic root (Fig. 19.106). The oblique course is a consequence of the aortic overriding. Residual shunting may be detected with Doppler imaging, usually at the margins of the patch. Next, right ventricular size and contractility should be assessed. These parameters have important long-term prognostic implications. Finally, the right ventricular outflow tract is interrogated. Evidence of residual stenosis may be recorded with Doppler imaging. The location and severity of any residual obstruction should be ascertained. In most cases, pulmonary regurgitation is also present. The magnitude varies considerably but is sometimes severe (Fig. 19.107). The clinical implications of chronic, severe pulmonary regurgitation after repair of tetralogy of Fallot are significant, and close follow-up and serial assessment with echocardiography is recommended. Some cases of repair involve either prosthetic pulmonary valves or right ventricular-to-pulmonary artery conduits. These types of repair are illustrated in Figure 19.108. In panels A to C, a porcine pulmonary valve is demonstrated, showing mild pulmonic regurgitation and a mean systolic gradient of 24 mm Hg. In panels D and E, a valved conduit is illustrated, from a patient with tetralogy and tricuspid atresia. The conduit had a peak gradient of approximately 60 mm Hg.
FIGURE 19.105. Following repair of tetralogy of Fallot, careful assessment of the right ventricular outflow tract, pulmonary valve, and pulmonary artery is critical. In this basal short-axis view, the outflow tract is widely patent and trivial pulmonic regurgitation is demonstrated by color Doppler. The level of the pulmonary valve is indicated by the arrow.
Transposition of the Great Arteries

The term transposition is used to describe a discordant ventriculoarterial connection in which the aorta arises from the morphologic right ventricle and the pulmonary artery arises from the left ventricle. Transposition can exist with either situs solitus or situs inversus. For simplicity, this section is a discussion of transposition in the presence of situs solitus only. The distinction between D-transposition and L-transposition is important and often is a source of confusion. In D-transposition, there is atrioventricular concordance and the morphologic right ventricle lies to the right of the morphologic left ventricle. In L-transposition, there is ventricular inversion and atrioventricular discordance. Thus, the morphologic right ventricle is to the left of the morphologic left ventricle. In both cases, the great arteries arise from the “incorrect” ventricle.

With normal conotruncal development, the pulmonary artery arises anterior and to the left of the aorta. Its initial course is posterior and then it bifurcates into right and left branches. The aortic valve is more posterior and rightward, and the course of the aorta is oblique with reference to the pulmonary artery. The aorta does not bifurcate but forms an arch as it passes posteriorly and inferiorly. Thus, the outflow tracts and great arteries of the right and left sides of the heart appear to wrap around one another in a spiral fashion. Transposition results in a more parallel alignment of the great arteries. With two-dimensional echocardiography, this positioning has been described as a “double-barrel” appearance rather than the normal “circle and sausage” orientation (Fig. 19.5).

D-Transposition

The echocardiographic diagnosis of D-transposition requires demonstration of a right-sided right ventricle giving rise to an aorta and a left-sided left ventricle giving rise to a pulmonary artery. In children, this anatomic structure is best evaluated from the subcostal four-chamber view, which allows all these features of D-transposition to be displayed. In adults, however, this assessment is technically challenging. More often, the parasternal short-axis and apical four-chamber views provide most of the
diagnostic information. When recording the four-chamber view, care should be taken to ensure proper orientation, with the left ventricle to the right and the right ventricle to the left. In the short-axis view, the aortic valve is usually anterior and to the right of the pulmonary valve, and the great arteries arise in parallel. It should be emphasized that this spatial relationship between the great arteries is not essential for the diagnosis, and the aorta occasionally lies directly anterior or slightly to the left of the pulmonary valve. These arrangements are easily discerned from the short-axis view at the base (Figs. 19.109 and 19.110). Because the semilunar valves occupy different levels (the aortic valve being slightly more cranial), they usually are not seen in the same short-axis plane. In the long-axis view, this parallel relationship of the great arteries can often be recorded in the longitudinal plane. By demonstrating that the anterior vessel arches posteriorly and the posterior vessel bifurcates, the diagnosis of D-transposition is established. Transesophageal echocardiography can be used to identify the great vessels (Fig. 19.111) but is usually not required. Visualization of the ostia of the coronary arteries and the brachiocephalic branch vessels also serves to identify the aorta.
FIGURE 19.106. After repair of tetralogy of Fallot, Doppler imaging demonstrates a residual ventricular septal defect at the margin of the surgically placed patch (arrow). The left-to-right jet is demonstrated in the long-axis (A) and short-axis (B) views. C: The velocity of the jet is recorded with continuous-wave Doppler imaging.

The presence of ventriculoarterial discordance alone will necessarily result in the creation of two parallel circuits and is incompatible with life. Therefore, admixture of arterial and venous blood is a prerequisite for survival and can occur at any level. An atrial septal defect, usually the secundum variety, is present in most patients. The size and direction of the interatrial shunt can be assessed with Doppler techniques. When venous admixing is inadequate, an atrial septostomy is often performed as a palliative
measure. This intervention can be performed under echocardiographic guidance. Echocardiography also plays a vital role in selecting candidates for this procedure and in determining its success (as judged by the size of the resulting defect).

The evaluation of patients after surgical correction of D-transposition relies heavily on echocardiographic techniques. Two distinct surgical procedures have been performed for treatment of this condition. In the past, the most common form of palliation for d-transposition was an intra-atrial baffle (also known as a Mustard, Senning, or atrial switch) procedure. A baffle diverts vena caval flow through the mitral valve to the left ventricle (and hence the pulmonary circuit) while simultaneously directing pulmonary venous blood through the tricuspid valve (and into the systemic circuit). Echocardiographic evaluation relies on direct visualization of the newly created systemic and pulmonary venous atria and careful assessment of right (i.e., systemic) ventricular function (Fig. 19.112). The presence and severity of tricuspid regurgitation (functional “mitral” regurgitation) should also be determined with Doppler imaging. It is essential to carefully evaluate ventricular function, which is usually done from the apical four-chamber and short-axis views (Fig. 19.113). In this case, the anatomic left ventricle (which is in the “left” position) will be the pulmonary ventricle. The right ventricle will be the systemic ventricle and will often appear dilated and hypokinetic.

In the parasternal long-axis view, the baffle is seen as an oblique, linear echo within the anatomic left atrium (Fig. 19.114). The pulmonary venous atrium is superior and posterior while the systemic venous atrium is in communication with the mitral valve. Medial or rightward angulation may permit visualization of the junction of the pulmonary venous atrium with the right ventricle. From the apical and subcostal four-chamber views, most regions of the baffle can be assessed. Shallow angulation of the transducer allows most of the pulmonary venous atrium to be recorded and is useful in detecting obstruction within this region (Fig. 19.115). By tilting the transducer more posteriorly, the junction between the inferior vena cava and the systemic venous atrium (an uncommon site of obstruction) is visible. Obstruction within the superior vena caval limb of the baffle is more common, but it may be difficult to visualize, particularly in adults. The subcostal and suprasternal short-axis views can be used for this purpose.
FIGURE 19.107. A patient with repaired tetralogy of Fallot. A: From the parasternal long-axis view, the right ventricle is dilated and the echogenic region at the superior portion of the interventricular septum represents the synthetic patch (arrows). B: From the apical four-chamber view, marked right ventricular hypertrophy is apparent. C: The right ventricular outflow tract and proximal...
pulmonary artery appear widely patent. The location of the pulmonary valve is indicated by the arrows. **D:** Color flow imaging of the right ventricular outflow tract demonstrates severe pulmonic regurgitation. **E:** Continuous-wave Doppler imaging confirms pulmonic regurgitation without a significant gradient across the pulmonary valve.

Video 19-107
FIGURE 19.108. Two examples of postoperative tetralogy of Fallot are shown. In A–C, the repair involved replacement of the pulmonic valve with a bioprosthesis. In B, color Doppler demonstrates mild pulmonic regurgitation. In C, continuous-wave Doppler reveals a 23 mm Hg mean gradient across the prosthetic valve. In D–F, a valved conduit was used for the surgical repair. In E, color Doppler shows valvular regurgitation within the conduit. In F, a peak systolic gradient of 60 mm
Hg is demonstrated across the conduit valve.  

Video 19-108a

coming soon

Video 19-108b

coming soon
FIGURE 19.109. D-transposition of the great arteries is illustrated. The patient had undergone an intra-atrial baffle procedure many years previously. In A, the parallel relationship of the great arteries can be seen from the basal short-axis view. The four-chamber view (B) demonstrates a dilated, hypokinetic right ventricle, which is the systemic ventricle.
FIGURE 19.110. In patients with D-transposition of the great arteries, the anatomic right ventricle acts as the systemic ventricle. **A:** From the four-chamber view, note that the right ventricle is dilated and hypokinetic. Similar findings are apparent in the short-axis view (**B**). **C:** The transposed great artery relationship is demonstrated.

FIGURE 19.111. In D-transposition of the great arteries, the relationship between the vessels is readily demonstrated using transesophageal echocardiography. **A:**
The parallel course of the great arteries is shown. B: From a short-axis plane, the side-by-side relationship of the semilunar valves is illustrated, with the aortic valve (AV) in a more anterior position.

FIGURE 19.112. In D-transposition, following Mustard repair, the anatomic right ventricle acts as the systemic ventricle. Assessing systemic ventricular function and the presence and severity of tricuspid regurgitation is essential.
Leaks within the baffle can be detected by using contrast echocardiography from the four-chamber view (Fig. 19.116). With this technique, right-to-left baffle leaks can be diagnosed with high sensitivity. Color Doppler imaging also permits these leaks to be identified and localized. Obstruction within the baffle can also be detected with contrast echocardiography or color Doppler imaging. Obstruction within the superior vena cava is assessed from the suprasternal notch. With a normally functioning baffle, color Doppler imaging can be used to follow the undisturbed, low-velocity flow from the vena cava to the systemic venous atrium. Pulsed Doppler imaging can identify obstruction as a continuous, turbulent flow in excess of 1 m/s. Obstruction within the pulmonary venous atrium requires the use of Doppler techniques for detection. First, color Doppler imaging is used to search for turbulence within the conduit. Then, pulsed Doppler imaging can be applied to measure the increased velocity within the structure. A diastolic flow velocity that is greater than 2 m/s suggests significant obstruction. Lower-velocity turbulent flow does not exclude the possibility of obstruction, however. Transesophageal echocardiography has been used to more accurately assess intra-atrial baffles. Use of this technique may be particularly important in adults in whom transthoracic image quality is sometimes a limitation.
L-Transposition
In simplest terms, L-transposition can be thought of as isolated ventricular inversion in which the morphologic right ventricle is to the left of the
morphologic left ventricle. The echocardiographic diagnosis rests on demonstrating abnormal atrioventricular and ventriculoarterial connections. Determining ventricular morphology and establishing the spatial relationships of the two chambers are accomplished as described previously. The discordant connections are detected by using multiple echocardiographic windows. From the four-chamber view, the presence of ventricular inversion usually can be established (Fig. 19.117). Apical displacement of the left-sided tricuspid valve can also be demonstrated. In the long-axis view, direct continuity between the pulmonary valve and the anterior mitral leaflet is apparent. In most cases, the ventricles are oriented in a side-by-side fashion, which creates some unusual and confusing echocardiographic views. For example, the parasternal long-axis plane may be vertical. In the short-axis view, the septum also appears more vertical (i.e., perpendicular to the frontal plane). The great arteries arise in parallel, with the aorta usually positioned leftward, anterior, and superior to the pulmonary valve. This is best appreciated from the basal short-axis view. This relationship contrasts with D-transposition, in which the basal short-axis view. This relationship contrasts with D-transposition, in which the aortic valve is anterior and usually rightward of the pulmonary valve.

![FIGURE 19.114. A Mustard repair is shown in a patient with D-transposition of the great arteries. The intra-atrial baffle is well visualized. A: From the long-axis view, the relationship of the systemic venous atrium (SVA) and pulmonary venous atrium (PVA) is shown. B: Systemic atrioventricular valve regurgitation is seen with color Doppler imaging. C: An apical view demonstrates the PA arising from the posterior left ventricle.](image-url)
FIGURE 19.115. A Mustard repair of transposition of the great arteries. From the apical window, by tilting the transducer at different angles, the various limbs of the baffle can be visualized. **A:** The pulmonary venous atrium (PVA) can be seen in association with the anatomic right atrium. **B:** The systemic venous atrium (SVA) diverts blood through the mitral valve. 

Video 19-115
After Mustard repair of D-transposition, the intra-atrial baffle can be assessed for leaks using agitated saline. In this example, a venous injection demonstrates a large baffle level leak with bubbles immediately filling the systemic ventricle, the equivalent of a large right-to-left atrial shunt.
FIGURE 19.117. L-transposition is defined by the presence of ventricular inversion, with a right-sided left ventricle acting as the pulmonary ventricle and a left-sided right ventricle acting as the systemic ventricle. Video 19-117
FIGURE 19.118. A common sequelae of L-transposition is the development of systemic (right) ventricular dysfunction, with dilation and hypokinesis (A). In B, associated regurgitation of the TV into the left atrium is also common.
FIGURE 19.119. A patient with situs inversus and L-transposition of the great arteries is evaluated with both transthoracic and transesophageal echocardiography. A: From the apical window, the atria are malposed with the left atrium on the right side and the right atrium to the left. There is atrioventricular
discordance. **B:** Doppler imaging demonstrates mild pulmonary valve stenosis and regurgitation. **C:** With transesophageal echocardiography, the relationship between the atria and the ventricles is shown. The systemic (right) ventricle is moderately hypokinetic. **D:** From a long-axis view, subpulmonic stenosis (arrow) is noted within the left ventricular outflow tract. This is further suggested with color flow imaging in E. TR, tricuspid regurgitation.

**FIGURE 19.120.** An example of tricuspid atresia. **A:** The atretic tricuspid valve is indicated by the arrows and the asterisk denotes the ventricular septal defect. A hypoplastic right ventricle is present but not well seen in this view. A large atrial septal defect is evident. **B:** Significant mitral regurgitation is documented using color Doppler imaging.
Associated anomalies are a common and important feature of L-transposition. Structural abnormalities of the left-sided tricuspid valve occur in most patients. Apical displacement of the leaflet insertions (an Ebstein-like deformity) and tricuspid regurgitation may occur (Fig. 19.118). A perimembranous ventricular septal defect is present in approximately 70% of cases. Less often, left ventricular outflow tract obstruction (valvular or subvalvular pulmonary stenosis) is present and can be assessed with Doppler imaging (Fig. 19.119). Finally, right (i.e., systemic) ventricular function is frequently abnormal and should be examined carefully. Gradual deterioration in function of the right side of the heart frequently occurs over time. Echocardiography plays an important role in the detection of this problem and in the assessment of any associated tricuspid regurgitation. In Figure 19.119, a patient with situs inversus and L-transposition is studied using both transthoracic and transesophageal imaging. From the chest wall, systolic function of both ventricles is determined and Doppler imaging demonstrates a gradient across the pulmonic valve as well as regurgitation (Fig. 19.119B). On transesophageal echocardiography, subpulmonic (i.e., in the left ventricle) stenosis is documented (Fig. 19.119D,E).

**Tricuspid Atresia**

This condition is discussed here because the presence of an atretic tricuspid valve invariably leads to some degree of right ventricular hypoplasia. As a consequence, this lesion may be confused with some of the other disorders...
included in this section. Tricuspid atresia is characterized by an imperforate tricuspid valve, hypoplasia of the morphologic right ventricle, an interatrial communication, and a normally developed left ventricle and mitral valve. In contrast to single ventricle, the hypoplastic chamber has an inlet portion (although it is atretic), and therefore it is properly called a ventricle. The interatrial communication is most often a patent foramen ovale and is therefore restrictive. A larger secundum defect is present in approximately 25% of patients. The clinically important variable features of tricuspid atresia include the ventriculoarterial communication (concordant or transposed), the presence and size of a ventricular septal defect, and the presence and magnitude of obstruction to pulmonary blood flow.

The echocardiographic diagnosis of tricuspid atresia is made from the four-chamber view from which the imperforate tricuspid valve can be visualized directly (Fig. 19.120). The presence of severe valvular hypoplasia (rather than atresia) is established by detecting remnants of the tricuspid valve apparatus. In either case, the inlet is imperforate. When the atresia is caused by a membrane, considerable motion in the area of the annulus may be present. Doppler imaging is useful for confirming the absence of flow through the inlet. The size and function of the hypoplastic right ventricle can be determined, and the presence of mitral regurgitation can also be assessed. The parasternal long-axis view is used to examine the septum for defects and to help determine the great artery relationships. Because any form of great artery connections is possible, the exact position of the posterior great vessel relative to the septum must also be noted. By scanning superiorly, the presence or absence of transposition can usually be determined. In the short-axis view, the right ventricular outflow tract and pulmonary valve can be evaluated for the presence of outflow obstruction. Confirming the diagnosis of pulmonary artery atresia, however, requires the use of multiple imaging planes. The subcostal views may be helpful in assessing the size of the interatrial communication. Dilation of the right atrium and bowing of the septum into the left atrium suggest a small, restrictive communication. From the suprasternal notch, the size and continuity of the pulmonary arteries can be assessed.

**The Fontan Procedure**
For lesions such as single ventricle and tricuspid atresia, in which abnormal right ventricular structure or function prevents adequate pulmonary blood flow, the Fontan procedure is frequently used for effective palliation. The Fontan anastomosis is a connection between the systemic atrium and the pulmonary circuit that is designed to increase pulmonary blood flow. The Fontan circuit can be created in a variety of ways. In many cases, a direct anastomosis using pericardial tissue is placed between the right atrial appendage and the pulmonary artery. In other situations, a valved or nonvalved conduit is used. Intra-atrial conduits, connecting the inferior vena cava to the pulmonary artery, are also placed.

Visualization of the Fontan anastomosis is often challenging. Optimal evaluation requires knowledge of the specific type of connection that was created surgically. The course of most of these connections is retrosternal, further complicating their echocardiographic detection. High parasternal and subcostal views are usually most effective (Fig. 19.121). There have been a variety of modifications and improvements in the original Fontan concept. For example, the Fontan connection may instead involve an internal conduit, sometimes called a lateral tunnel Fontan (Fig. 19.122). These conduits are more easily visualized and appear as a circular insert within the right atrium. Once the connection is visualized, Doppler imaging plays an important role in assessing the flow pattern and in determining the presence of conduit dysfunction. Normal pulmonary artery flow after a Fontan procedure is biphasic, with one peak in late systole and a larger peak in late diastole during atrial contraction, assuming normal downstream resistance. Augmentation of flow velocity is normally seen during inspiration. Abnormal systemic ventricular function is suggested by reduced or absent late diastolic flow and diminished respiratory variation in the flow pattern. Transesophageal echocardiography can also be used to assess the Fontan connection.
FIGURE 19.121. A: Short-axis view at the base of the heart in a patient with tricuspid atresia demonstrates a Fontan conduit (C) (arrows) passing anterior and left of the aorta. B: Angulation of the scan plane permits demonstration of the distal anastomosis of the conduit into the PA (arrowheads). C: Color flow imaging in the same plane demonstrates flow within the conduit without significant turbulence, which suggests the absence of significant obstruction within the conduit.
FIGURE 19.122. This type of Fontan employs an internal conduit and is sometimes called a lateral tunnel Fontan. The conduit can be seen in cross section (asterisk) within the right atrium in this patient with tricuspid atresia.
FIGURE 19.123. One modification of the Fontan procedure involves creating a fenestration to allow shunting between the Fontan connection (asterisk) and the pulmonary venous (i.e., left) atrium, a type of right-to-left shunt. This can be assessed using color Doppler imaging (arrow). Continuous-wave Doppler imaging can also be performed to estimate the gradient across the pulmonary circuit.
Fontan connections may also be fenestrated, purposely allowing right-to-left shunting. This is usually done in the setting of increased pulmonary vascular resistance to “decompress” the right atrium when pulmonary vascular resistance is high. Such fenestrations usually are created at the time of surgery in high-risk patients and closed at a later time. The shunt flow can be visualized using color Doppler imaging (Fig. 19.123). The velocity of the shunt flow, assessed with continuous-wave Doppler imaging, reflects the pressure gradient between the Fontan and the left atrium and is therefore a useful indicator of the total pressure gradient across the pulmonary circuit.

**Suggested Readings**

**GENERAL CONCEPTS**


**COMPLEX LESIONS**


**HEMODYNAMICS**


**POSTOPERATIVE EVALUATION**


**SEPTAL DEFECTS**


Kronzon I, Tunick PA, Freedberg RS, Trehan N, Rosenzweig BP, Schwinger ME. Transesophageal


**Valvular Lesions**


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Chapter 20
Diseases of the Aorta

Echocardiographic evaluation of the aorta has risen in importance due to the increasing recognition of multiple clinical syndromes requiring surveillance for aortic pathology. While transthoracic and transesophageal echocardiography remain frontline diagnostic techniques, computed tomography (CT) and cardiac magnetic resonance imaging (MRI) play an increasingly valuable role for complete evaluation of aortic anatomy.

The major benefit of both CT and MRI is the ability to evaluate the entire extent of the aorta from the aortic annulus through the arch, descending aorta, and into the bifurcation into the femoral arteries. This can be accomplished in virtually all patients with a high degree of accuracy, and these techniques afford superb three-dimensional reconstruction for highly detailed evaluation of complex aortic anatomy. Both techniques provide excellent visualization of aortic dissection and aortic aneurysm, and CT may be particularly useful for evaluation of penetrating aortic ulcer. In general, for patients with disease of the aorta, echocardiography may suffice for initial detection and ongoing surveillance, but the majority of patients will require complete evaluation of the full extent of the aorta on at least one occasion with either CT or MRI. Table 20.1 outlines the relative advantages, disadvantages, and limitations of all currently available imaging techniques. A major limitation of CT is the substantial radiation exposure, as well as contrast load which must be taken into consideration, especially when evaluating relatively young individuals on a serial basis, as may be required for hereditary aneurysm syndromes.

There are multiple diseases affecting the aorta, including the Marfan syndrome, bicuspid aortic valve, familial aortic aneurysm syndromes, as well as degenerative disease related to aging, hypertension, and tobacco abuse. For all of these, not only will imaging be required to establish the diagnosis but,
depending on the nature of the disease, will be required for surveillance at variable intervals. Diseases which affect the aorta are outlined in Table 20.2, and selected Appropriate Use Criteria for the utilization of echocardiographic techniques in known or suspected disease of the aorta is presented in Table 20.3.

NORMAL AORTIC ANATOMY

The normal aorta consists of six segments. These are schematized in Figures 20.1 and 20.2 and consist of the annulus, sinuses of Valsalva, sinotubular junction, ascending tubular aorta, the arch, and the descending thoracic aorta. The proximal portion, from the annulus to the proximal ascending aorta, is commonly referred to as the “aortic root.” Because the proximal aorta consists of four separate sections, the term “aortic root” oversimplifies the anatomy, and when discussing dilation or other pathologies, one should specify the precise level rather than simply referring to the “aortic root.” The aortic annulus is defined as the junction of the proximal ascending aorta with the left ventricular outflow tract. It is part of the fibrous skeleton of the heart and is contiguous with the anterior mitral valve leaflet and perimembranous septum. Because the annulus is a fibrous structure, it is relatively resistant to dilation and represents a relatively stable dimension to which the remaining aortic sizes can be indexed. Typically, the aortic annulus measures 13 ± 1.0 mm/m² in diameter. The normal aorta dilates at the level of the sinuses by approximately 6 mm/m² and then tapers to within 2 to 3 mm of annular size at the sinotubular junction (Fig. 20.1). Aortic size is related to height and body surface area. Normally there are three sinuses of Valsalva of equal size. The right and left sinuses contain the ostia of the right and left coronary arteries. The takeoff of the left coronary artery can be visualized in the left sinus where its position is relatively closer to the annulus than is the takeoff of the right coronary artery which tends to be more superior and closer to the sinotubular junction.

When evaluating the aorta with either echocardiography, CT, or MRI, a precise measurement technique is necessary, as relatively small changes in dimension may trigger an indication for surgical intervention. For echocardiographic techniques, several different schemes for measurement of
the aorta have been proposed. In general, the aorta should be measured at the
annulus, maximum dimension of the sinuses of Valsalva, sinotubular
junction, ascending aorta (typically standardized to the level of the right
pulmonary artery), and mid arch (Figs. 20.3 and 20.4). Multiple additional
measurements of the descending aorta can be made with CT, MRI, or
transesophageal echocardiography. Measurements should be made
perpendicular to the long axis of the direction of flow. Because in disease
states the aorta may take on a curvilinear direction, off-angle measurements
are not uncommon which may result in either over- or underestimation of the
true aortic dimension. CT and MRI, as well as three-dimensional
echocardiography, may reduce this tendency.

<table>
<thead>
<tr>
<th>Technique</th>
<th>Extent</th>
<th>Atheroma</th>
<th>Dissection</th>
<th>Associated Disease</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>TTE</td>
<td>AV, proximal aorta and arch</td>
<td>No</td>
<td>Limited</td>
<td>All cardiac anatomy, AI, LV function, PEF</td>
<td>Limited visualization</td>
</tr>
<tr>
<td>TEE</td>
<td>Aortic valve to diaphragm</td>
<td>Yes</td>
<td>Accurate as viewed</td>
<td>All cardiac anatomy, AI, LV function, PEF</td>
<td>Limited to above diaphragm</td>
</tr>
<tr>
<td>CT</td>
<td>Aortic valve to femorals</td>
<td>Yes</td>
<td>Accurate</td>
<td>Some anatomy, PEF, LV function</td>
<td>Dye load, radiation, motion artifact</td>
</tr>
<tr>
<td>MRI</td>
<td>Aortic valve to femorals</td>
<td>Yes</td>
<td>Accurate</td>
<td>Most cardiac anatomy, AI, LV function, PEF</td>
<td>Patient tolerance availability</td>
</tr>
<tr>
<td>Angiography</td>
<td>Aortic valve to femorals</td>
<td>Yes</td>
<td>Accurate</td>
<td>None</td>
<td>Invasive, limited availability</td>
</tr>
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<table>
<thead>
<tr>
<th>Table 20.2</th>
<th>DISEASES AFFECTING THE AORTA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Atherosclerotic</strong></td>
<td>Aneurysm</td>
</tr>
<tr>
<td><strong>Atheroembolic disease</strong></td>
<td></td>
</tr>
<tr>
<td>Dissection</td>
<td></td>
</tr>
<tr>
<td>-----------</td>
<td></td>
</tr>
<tr>
<td><strong>Nonatherosclerotic</strong></td>
<td></td>
</tr>
<tr>
<td>Medial degeneration</td>
<td></td>
</tr>
<tr>
<td>Aneurysm (syndromic)</td>
<td></td>
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<tr>
<td>Aortic dissection</td>
<td></td>
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<tr>
<td>Intramural hematoma</td>
<td></td>
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<tr>
<td>Annuloaortic ectasia</td>
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<tr>
<td><strong>Inflammatory/infectious</strong></td>
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<tr>
<td>Takayasu arteritis</td>
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<tr>
<td>Giant cell arteritis</td>
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<tr>
<td>Endocarditis</td>
<td></td>
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<tr>
<td><strong>Congenital and genetically mediated</strong></td>
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<tr>
<td>Marfan syndrome</td>
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<tr>
<td>Turner syndrome</td>
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<tr>
<td>Ehlers–Danlos syndrome</td>
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<tr>
<td>Familial aneurysm</td>
<td></td>
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<tr>
<td>Bicuspid aortic valve</td>
<td></td>
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<tr>
<td>Aortic coarctation</td>
<td></td>
</tr>
<tr>
<td><strong>Miscellaneous</strong></td>
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<tr>
<td>Trauma</td>
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<tr>
<td>Intraluminal thrombus</td>
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<tr>
<td>Hypertension</td>
<td></td>
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<tr>
<td>Aortic insufficiency/stenosis</td>
<td></td>
</tr>
<tr>
<td>Iatrogenic injury</td>
<td></td>
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</table>
FIGURE 20.1. The thoracic aorta can be characterized as having three major segments. The ascending aorta extends from the annulus to the innominate artery and includes the three sinuses of Valsalva, the three cusps of the aortic valve, the sinotubular junction, the ostia of the coronary arteries, and the proximal ascending aorta. The arch extends from a left innominate to the ligamentum arteriosum and includes the great vessels arising off of the arch. The descending thoracic aorta extends from the ligamentum arteriosum to the level of the diaphragm. The normal dimensions of the aorta are noted in the schematic and vary with location. Dimensions are given both indexed to body surface area (BSA) and as the range anticipated in routine adult echocardiography.

Table 20.3 APPROPRIATENESS CRITERIA FOR THE APPLICATION OF ECHOCARDIOGRAPHY IN KNOWN OR SUSPECTED DISEASE OF THE AORTA

<table>
<thead>
<tr>
<th>Indication</th>
<th>Appropriateness Score (1–9)</th>
</tr>
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<tbody>
<tr>
<td>1. Symptoms or conditions potentially related to suspected cardiac</td>
<td>A (9)</td>
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etiology including but not limited to chest pain, shortness of breath, palpitations, TIA, stroke, or peripheral embolic event

<table>
<thead>
<tr>
<th>2. Prior testing that is concerning for heart disease or structural abnormality including but not limited to chest x-ray, baseline scout images for stress echocardiogram, ECG, or cardiac biomarkers</th>
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<td>A (9)</td>
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<tr>
<th>19. Hypotension or hemodynamic instability of uncertain or suspected cardiac etiology</th>
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<tr>
<td>A (9)</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>63. Evaluation of the ascending aorta in the setting of a known or suspected connective tissue disease or genetic condition that predisposes to aortic aneurysm or dissection (e.g., Marfan syndrome)</th>
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</thead>
<tbody>
<tr>
<td>A (9)</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>64. Reevaluation of known ascending aortic dilation or history of aortic dissection to establish a baseline rate of expansion or when the rate of expansion is excessive</th>
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<tbody>
<tr>
<td>A (9)</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>65. Reevaluation of known ascending aortic dilation or history of aortic dissection with a change in clinical status or cardiac exam or when findings may alter management or therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (9)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>66. Routine reevaluation for surveillance of known ascending aortic dilation or history of aortic dissection without a change in clinical status or cardiac exam when findings would not change management or therapy</th>
</tr>
</thead>
<tbody>
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<td>rA (3)</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>104. Suspected acute aortic pathology including but not limited to dissection/trans-section</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (9)</td>
</tr>
</tbody>
</table>


![Diagram of the aorta and its components](image)

**FIGURE 20.2.** The relative dimensions of the annulus, sinuses of Valsalva, sinotubular junction, and proximal ascending aorta. In the disease-free state, the
sinuses dilate symmetrically so that their greatest dimension exceeds that of the annulus by approximately 6 mm/m² of the body surface area. At the level of the sinotubular junction, the aorta narrows to within 2 to 3 mm of its annular dimension and then gradually tapers throughout its course. Note that the aortic cusps coapt along a 2- to 3-mm coaptation zone and do not meet tip to tip.

FIGURE 20.3. Transthoracic parasternal long-axis view of the normal aorta. This view includes the normal attachment of the anterior mitral valve leaflet to the posterior wall of the aorta and also visualization of the left atrium. Note the similar relationship in size of the anatomically viewed aorta compared with the schematic in Figure 20.2. In the expanded inset at the lower right note the overlap in coaptation of the aortic cusps (arrows).
There are several different protocols for measuring aortic diameter. These include measuring the true luminal dimension of the inner edge to the inner edge of the aorta, measuring the outer dimension to the outer dimension, and exclusive and unique to echocardiography, measuring the leading edge to leading edge. The latter was first recommended when measuring with M-mode and early-generation two-dimensional scanners where “blooming” of the image was not uncommon and could result in underestimating the true diameter. Because many echocardiographic studies and surveillance studies are based on this technique, the current recommendation of the American Society of Echocardiography is to continue measuring leading edge to leading edge. While measuring leading edge to leading edge at end diastole is the recommended echocardiographic method, guidelines for measuring CT and MRI often include measuring the outer edge to outer edge of the aorta to ensure that the maximum size of the aorta, including intraluminal thrombus and atherosclerotic burden, is included. As such, for a given patient, measurements by CT and MRI may be systematically larger than echocardiographic measurements and caution is advised when comparing aortic measurements from multiple modalities. Measurement at the level of the sinuses of Valsalva may be particularly problematic with two-dimensional echocardiography, as there can be significant variability in the maximum dimension within the sinuses depending on which sinuses are measured (Fig. 20.5). Caution is also needed to avoid including the proximal portion of a coronary artery within the aortic measurement.
The geometry of the sinotubular junction is a crucial feature of normal aortic valve coaptation. Insertion of aortic valve cusps is continuous from the level of the annulus up through the sinuses to the sinotubular junction. Dilation of the sinotubular junction can result in splaying of the coaptation line of the aortic cusps, resulting in secondary aortic insufficiency. The ascending aorta terminates at the right innominate artery (brachiocephalic artery), where the aortic arch begins and continues to the left subclavian artery and ligamentum arteriosum. The three major branch vessels of the arch, the right innominate artery, and the left carotid and subclavian arteries can be visualized in most patients from a suprasternal view (Fig. 20.4) as well as from the transesophageal approach. The dimension of the ascending aorta, arch, and descending thoracic aorta are all similar with slight tapering in the descending thoracic aorta.

**FIGURE 20.4.** Suprasternal notch view recorded in a patient without aortic pathology. In the central figure note the curve of the arch and the takeoff of the left carotid artery (LCA) and left subclavian artery (LSCA). Superior to the arch note the brachiocephalic vein (BCV). In the small inset note the normal Doppler velocity of approximately 60 cm/s recorded in the proximal descending aorta.
FIGURE 20.5. Magnetic resonance angiogram of the aorta recorded in a young patient incidentally noted to have aortic insufficiency and a dilated aortic root. The central figure is a lateral view of the aorta demonstrating the sinuses of Valsalva (arrow), ascending aorta arch, and descending aorta. Note the selective dilation of the sinuses of Valsalva with lesser degrees of dilation at the sinotubular junction and normal appearance of the descending thoracic aorta. The inset is a short-axis view at the level of the aortic sinuses demonstrating the range of dimensions of the aortic root at this level of the sinuses depending on the orientation of the plane. Note that all three measurements transect the center of the aorta and the largest measurement of 41.4 mm as compared to the smallest dimension of 34.5 mm.

ECHOCARDIOGRAPHIC EVALUATION

Only the proximal 4 to 8 cm of the ascending aorta is typically visualized from the parasternal long-axis view. The arch and a short segment of the descending thoracic aorta can be visualized from the suprasternal notch. Figure 20.3 is a parasternal long-axis view of the heart with superior angulation that emphasizes visualization of the ascending aorta. Note the relative dimensions of the annulus, sinuses, sinotubular junction, and ascending aorta which can be accurately determined from this transthoracic image. The suprasternal notch transducer position provides visualization of the arch and great vessels of the aorta. Figure 20.4 was recorded in a normal individual in whom the majority of the arch and great vessels can be visualized from the suprasternal notch. Imaging from the suprasternal view often is more feasible in children and adolescents than in adults. The examiner should be aware that placement of the ultrasound probe in the suprasternal notch may result in mild patient discomfort. Finally, transthoracic echocardiography can visualize a limited portion of the descending aorta. In the parasternal long-axis view, the descending thoracic aorta appears as a circular structure behind the left atrium. On occasion, it can be confused with a dilated coronary sinus; however, the proximity of the coronary sinus to the atrioventricular groove as well as the more rigid shape of the aorta should be the accurate discriminating features. The proximal abdominal aorta can also be visualized from a subcostal imaging position (Fig. 20.6).

Transeosophageal echocardiography provides a broader window to aortic anatomy. The aorta can be visualized from the annulus through the ascending
aorta, arch, and descending thoracic aorta to the level of the gastroesophageal junction. Figures 20.7 through 20.10 are transesophageal echocardiographic images recorded in patients with normal thoracic aortas.

Typically, transesophageal echocardiographic imaging of the aorta begins with imaging of the ascending aorta with the probe behind the left atrium. In general, the proximal 5 to 10 cm of the ascending aorta can be visualized by scanning at a 120-degree imaging plane (Fig. 20.7). By rotating the imaging plane to a 40- to 60-degree view, a series of short-axis views of the proximal ascending aorta can be obtained, including a short-axis view of aortic valve closure (Fig. 20.8). The descending thoracic aorta is imaged by inserting the probe deeper toward the gastroesophageal junction, rotating it 180 degrees to face posteriorly and scanning at a 0-degree imaging plane. The probe can then be slowly withdrawn along the length of the aorta and a continuous series of short-axis views of the thoracic aorta obtained (Fig. 20.9). At any point along the course of the aorta, the image can be rotated to a 90-degree plane for a longitudinal view of the aorta. In elderly patients, the aorta becomes tortuous, and rotation of the probe is frequently necessary to maintain a short-axis view of the aorta in the center of the imaging plane. When visualizing the arch, it should be emphasized that the probe will be at a relatively shallow depth (15 to 25 cm from the incisors) which results in a more dramatic curvature of the probe in the oropharynx which may be less well tolerated than deeper probe positions. The arch is best visualized by slowly withdrawing the probe to the level of the left subclavian artery, and as the probe is further withdrawn, it is rotated clockwise to obtain an elongated view of the arch (Fig. 20.10). At the point that the arch is seen at the apex of the scanning plane, the multiplane probe can be rotated to a 90-degree imaging plane and a short-axis view of the apex of the arch can be recorded. By rotating clockwise and counterclockwise, the takeoff of the great vessels can often be visualized from this view.
FIGURE 20.6. Subcostal imaging of the normal descending abdominal aorta (DAo). The image was recorded with color flow Doppler and laminar flow is seen within the aorta. Flow is also noted in the celiac axis (CA). The inset at the lower left is pulsed Doppler recorded within the lumen of the descending aorta revealing a normal flow profile. [Video]
FIGURE 20.7. Transesophageal echocardiogram of the ascending aorta recorded in a normal disease-free individual in a longitudinal (126 degrees) view that provides imaging analogous to that of the transthoracic long-axis view seen in Figure 20.3. Again note the symmetric dilation at the level of the sinuses and the narrowing at the level of the sinotubular junction. The inset recorded in diastole demonstrates closure of the aortic cusps along a 2- to 3-mm length (arrows). A, anulus; S, sinus of valsalva; STJ, sinotubular junction; T, tubular aorta.

Video 20-7
FIGURE 20.8. Transesophageal echocardiogram recorded at 53-degree image rotation at the base of the heart. With this probe orientation, a short-axis view of the aorta is obtained at the level of the sinuses, revealing the left (L), right (R), and non (N) coronary sinuses. The left atrium, right atrium, and proximal PA are well visualized. A: Image recorded in diastole, and three symmetric sinuses are noted as well as three coaptation lines of the cusps. B: Image recorded in systole
and shows the relatively triangular and symmetric opening of all three cusps.

Video 20-8
FIGURE 20.9. Transesophageal echocardiogram of the descending thoracic aorta. **A:** Recorded at 0 degrees and provides a short-axis view of a circular and symmetric normal aorta with little or no atherosclerotic disease. **B:** Recorded with the imaging plane at 90 degrees providing a longitudinal view of the descending
thoracic aorta. Because of the highly reflective nature of the aortic wall, a reverberation artifact mimicking a second aorta behind the real image is frequently encountered.

Video 20-9A

Video 20-9B
FIGURE 20.10. Transesophageal echocardiographic view of the arch of the aorta. A: Recorded with the imaging plane at 0 degrees with marked clockwise rotation of the probe. In occasional patients, even more marked probe angulation can allow visualization of the ascending aorta to a level near to the sinotubular junction. B: Recorded from the same transducer position with the probe at an
angle of 85 degrees providing a short-axis view of the apex of the arch. The takeoff of the left subclavian (LSC) can often be visualized from this view.

Evaluation of the proximal aorta should be done systematically and measurements should be made at the level of the annulus, sinuses, sinotubular junction, and proximal ascending aorta (Fig. 20.2). The echo report should specifically note dimensions at multiple standardized locations of the aorta. As the pathologic process is diffuse and can involve any area of the aorta, and may simultaneously involve multiple areas, most patients will require at least one screening evaluation with an alternative imaging technique (CT or MRI) to evaluate the full extent of the aorta from the sinuses to its bifurcation (Fig.
An additional ultrasound modality that has been used in evaluation of the aorta is intravascular ultrasound (Fig. 20.12). This can be performed with high-frequency (20- to 30-MHz) transducers or more recently with an intracardiac probe operating at 5.5 to 10 MHz. These higher-frequency probes provide a highly detailed, high-resolution view of intra-aortic anatomy including visualization of the intimal and medial layers when using the higher-frequency probes. Intravascular ultrasound has been used in the diagnosis and management of aortic dissection and as a primary imaging tool to monitor therapeutic fenestration performed for acute aortic dissection. Intravascular ultrasound has the advantage of being able to image the entire aorta, from the root to the iliac artery. In dissection, it demonstrates the true and false lumens, the dissection flap, and thrombosed false lumen. It can also demonstrate the origin of abdominal aortic branches (iliac artery, mesenteric branches, renal arteries), detecting whether they arise from true or false lumen. Intimal tear sites can also be imaged. Determination of aortic segment dimensions by this technique correlate precisely with computed tomographic and transesophageal echocardiographic measurements.
FIGURE 20.11. Composite illustration of a CT angiogram performed in a patient with a mildly dilated ascending aorta. The figure depicts multiple display formats which can be derived from the full CT angiogram. At the lower left is a three-dimensional reconstruction of the aorta from the level of the aortic annulus through the descending thoracic aorta. Note the mild asymmetric dilation at the level of the sinuses. There is excellent visualization of the great vessels arising from the arch. At the upper left inset is an expanded planar view of the ascending aorta at the level denoted by the single line on the three-dimensional image. At this level, the aorta is measured to be 35.4 mm in diameter. Superimposed on the three-dimensional image note the line drawn through the center line of the aorta. At the right is an artificially straightened view in a planar image of the aorta from the level of the annulus at the top of the image to the bifurcation at the bottom. The graph at the right displays the diameter of the aorta at each level.
With advancing age and varying degrees of atherosclerosis, the distensibility and pulsatility of the aorta diminish. Several studies have confirmed the ability of echocardiographic imaging either with manual tracing of the aortic contour throughout the cardiac cycle or automatic edge detection contouring to demonstrate changes in aortic distensibility during systole. These changes have been suggested as an early predictor of atherosclerosis and thought to represent end-organ effects of hypertension and atherosclerosis.
Dilation of the aorta can occur at any point along its course. Identification of disease in one portion of the aorta should prompt evaluation of the full extent of the aorta because many diseases affecting one portion of the aorta can also have manifestations in other areas as a part of a generalized aortopathy. Full evaluation generally will require either CT or MRI. The nature of subsequent surveillance can then be tailored to the location(s) of disease. An aneurysm is defined as enlargement to more than 1.5 times the normal dimension for that aspect of the aorta. Dilation of the aorta can either be an isolated condition or associated with other cardiovascular diseases such as hypertension or aortic valve disease. Bicuspid aortic valve is often associated with concurrent primary disease of the aorta (aortopathy). Idiopathic dilation (below the threshold for definition of an aneurysm) and tortuosity have often been referred to as annuloaortic ectasia. It is unclear whether this is a distinct disease entity or related to the effects of aging, hypertension, or unrecognized primary disease of the aorta.

Dilation of the proximal ascending aorta can often be appreciated from the transthoracic parasternal long-axis view (Fig. 20.13). As noted previously, the aortic annulus is a relatively stable structure that can serve as an internal index to the anticipated size of the aorta in individuals of varying body size. In Figures 20.13 through 20.26, note the variable degree of aortic dilation as it extends from the annulus to the ascending aorta. Because the aortic valve cusps insert circumferentially along the sinotubular junction, dilation or effacement at the level of the sinotubular junction may result in malcoaptation and secondary aortic insufficiency (Fig. 20.16).

Aortic aneurysms are typically characterized by their location as either being in the ascending aorta, arch, or descending thoracic or abdominal aorta. Aortic aneurysms can occur in multiple locations and obviously may be contiguous between several segments. Size ranges from small localized focal aneurysms, to diffuse saccular involvement of extensive lengths of the aorta. Transthoracic echocardiography is often sufficient for initial identification and characterization of more proximal ascending aortic aneurysms. Transesophageal echocardiography is accurate for evaluation of aortic anatomy including aneurysms from the ascending aorta to the gastroesophageal junction. Both echocardiographic techniques may be
limited in precise localization and characterization of aneurysms falling outside of typical ultrasound planes, and for this reason CT or MRI are often essential for full characterization.

**FIGURE 20.13.** Parasternal long-axis view recorded in a patient with mild selective dilation of the sinus of Valsalva. In the central figure note the mildly prominent sinuses of Valsalva (*inward-pointing arrows*). At the upper right inset note the three-cusp aortic valve and the three sinuses of Valsalva. At the lower right inset note the expanded view with measurements of the proximal aortic root documenting a measurement of 4.5 cm at the sinuses of Valsalva.
FIGURE 20.14. Parasternal long-axis view recorded in a patient with dilation of the ascending aorta in the ascending tubular portion (AA). Note the measurements which have been obtained at the annulus (A) sinus of Valsalva (S) and sinotubular junction (STJ). Measurements are as noted in the lower left. There is isolated dilation of the ascending aorta (AA) which measures 4.3 cm. The inset at the upper left is recorded with color flow Doppler and confirms absence of aortic insufficiency.
On occasion, the first clue to the presence of an aortic aneurysm is obtained during routine transthoracic scanning for nonrelated purposes. Figures 20.23 through 20.26 were recorded in patients undergoing transthoracic echocardiography for unrelated reasons in whom aortic aneurysms were incidentally discovered. In general, transesophageal echocardiography will be necessary for further characterization of aortic aneurysms. Figures 20.27 through 20.32 are transesophageal echocardiographic images recorded in patients with known aneurysms of the ascending and descending thoracic aorta and demonstrate the broad range of pathology which can be documented with transesophageal echocardiography, including concurrent atheromatous involvement, rupture, and hemorrhage.
FIGURE 20.15. Transthoracic echocardiogram recorded in a patient with an ascending aortic aneurysm. In the central illustration note the marked dilation of the ascending distal to the sinotubular junction (double-headed arrow). The inset at the upper left was recorded from an apical imaging position, from which the dilated ascending aorta is also visualized. Note in this four-chamber orientation that the dilated ascending aorta has altered the imaging plane such that the right atrium is not simultaneously visualized with the left atrium.  

Video 20-15
FIGURE 20.16. Transesophageal echocardiogram recorded in a longitudinal view of the ascending aorta in a patient with proximal aortic dilation. In A, note the normal appearance of the aortic valve cusps in a closed position and the dilated proximal aorta. In B, note the moderate severity aortic insufficiency related to dilation at the level of the sinotubular junction which results in functional aortic insufficiency.
Aortic insufficiency not uncommonly occurs in ascending aortic aneurysms, and typically in stable patients is related to proximal aortic dilation with effacement of the sinotubular junction. This results in noncoaptation of the aortic cusps and functional aortic regurgitation which can range in severity from mild to severe (Fig. 20.16). If the aortic cusps are otherwise normal, surgical correction of the aortic dilation with restitution of normal proximal aortic geometry results in reduction and/or elimination of aortic insufficiency without the need for aortic valve replacement.

For patients with dilation or aneurysm of the ascending aorta, the likelihood of spontaneous rupture or dissection is directly related to the degree of dilation. Currently, for the general population, a threshold of 55 mm is considered an indication for prophylactic aortic surgery in an effort to reduce the likelihood of a catastrophic event such as rupture or dissection. In addition, a rapid change in the degree of dilation, usually defined as more than 5 mm/yr is often used as an indication for surgery. As operation success and outcomes have improved, many centers are using a threshold of 50 mm as an indication for surgery. It is not unreasonable to adjust this threshold based on gender or body size, however, firm guidelines regarding adjustment have not been established.
FIGURE 20.17. Transesophageal echocardiographic image of a longitudinal view of the ascending aorta in a patient with an ascending aortic aneurysm. The dimensions at the annulus (1), sinus of Valsalva (2), sinotubular junction (3), and maximum dimension of the visualized portion of the ascending aorta (4) are measured.

There are several special populations for which the standard thresholds are modified. Most authorities feel that the threshold for prophylactic intervention of the ascending aorta should be reduced for individuals with Marfan syndrome or with a family history of aortic rupture or dissection. Additionally, patients with a Loeys–Dietz syndrome are intervened at a substantially lower threshold of 40 to 45 mm because of the high prevalence of spontaneous aortic dissection and rupture in this population. Patients with Turner syndrome and a bicuspid aortic valve are also at greater likelihood of aortic syndromes and, in view of their intrinsic short stature, may warrant intervention at thresholds lower than that for the general population.

MARFAN SYNDROME
Marfan syndrome is a heritable disorder of connective tissue associated with a mutation of the FBN1 gene which encodes for fibrillin 1. It is associated with multiple cardiovascular abnormalities. Before the advent of corrective surgery, cardiovascular complications, especially aortic dissection and rupture, were the leading causes of mortality and resulted in death at an average age in the fourth or fifth decade. The cardiovascular manifestations of Marfan syndrome include degeneration of the medial layer of the aorta which results in dilation and weakening of the aortic wall. The most frequent area of dilation is in the proximal aorta and dilation may be confined to the aortic sinuses. Figures 20.33 to 20.36 were recorded in patients with characteristic features of Marfan syndrome. Although the sinuses are the most common sites of dilation, it should be recognized that the underlying pathologic process extends throughout the entire aorta, and patients with Marfan syndrome are at risk of aneurysm formation, dissection, and rupture at any point along the course of the aorta. For most patients, initial screening can be undertaken with transthoracic echocardiography.

![Transesophageal echocardiogram](image)

**FIGURE 20.18.** Transesophageal echocardiogram in a longitudinal view of the ascending aorta. Note the relatively normal dimensions of the annulus and sinotubular junction (double-headed arrow) and the marked dilation of the aneurysmal ascending aorta (An). The degree of dilation and distortion of
anatomy is such that the full boundary of the aneurysm was not visualized by transesophageal echocardiography. The small inset is a CT angiogram from the same patient nicely demonstrating the full extent and dimensions of the ascending aorta.

FIGURE 20.19. Transesophageal echocardiogram recorded in a longitudinal view in a patient with a large localized proximal aortic root aneurysm. In the central figure note the boundary of the aneurysm as noted by the downward-pointing
Management of patients with Marfan syndrome involves serial imaging to evaluate aortic size and monitor progression of dilation. Most authorities believe that, at the time of first detection, a patient should undergo an evaluation of the entire extent of the aorta, which can be performed with CT, or MRI (Fig. 20.37). If there is no evidence of distal aortic dilation, follow-up usually can be performed with transthoracic echocardiography because the proximal ascending aorta is the single most likely site to be involved in subsequent dilation. It should be emphasized that follow-up should include serial measurements as noted previously for comparison.
FIGURE 20.20. Transesophageal echocardiogram recorded in a biplane mode in the same patient depicted in Figure 20.19. The left-hand panel is similar to that seen in Figure 20.19, and the right-hand panel is recorded orthogonal to that and reveals the orifice of the bicuspid aortic valve (arrows), as well as the large saccular aneurysm. Video 20-20
FIGURE 20.21. Transesophageal echocardiogram recorded in a longitudinal view of the proximal aorta in a patient with a bicuspid valve and ascending aortic aneurysm. The aneurysm is largely thrombosed. The outer boundary of the aneurysm is noted by the dark arrows and the shorter downward-pointing white arrow. The patent lumen of the aorta is noted by the double-headed arrow. A bicuspid valve with mild thickening is present (leftward-pointing arrow). The inset at the upper right is a short-axis view recorded roughly at the plane of the double-headed arrow in the central figure. Again, note the marked dilation of the ascending aorta and the smaller functional lumen. The remainder of the aortic cavity is filled by thrombus (double-headed arrows).
The need to index aortic size to body size is not firmly established; however, the implications of dilation less than 50 or 55 mm in a small-statured individual are obvious. Aortic dilation associated with clinically relevant aortic insufficiency has been considered an indication for surgery as well (Figs. 20.35 and 20.36). After surgical repair, continued surveillance is crucial because this is a systemic process involving all portions of the aorta. However, after replacement of the ascending aorta in a patient with Marfan syndrome, follow-up may require transesophageal echocardiography, CT, or MRI because additional disease will typically not be in the field of view of transthoracic echocardiography.

The full spectrum of cardiovascular abnormalities in Marfan syndrome includes not only disease of the aorta but also an increased prevalence of myxomatous degeneration of the mitral valve with mitral valve prolapse (Fig. 20.38). When present, it has the same appearance and clinical implications of myxomatous degeneration and prolapse occurring in the non-Marfan patient. Typically, the leaflets are diffusely thickened and redundant and have characteristic buckling or prolapse behind the plane of the mitral annulus. Echocardiographic imaging in clinical management of mitral valve disease is discussed in Chapter 11.
FIGURE 20.22. Transesophageal echocardiogram recorded in a longitudinal view of the ascending aorta in a patient with a highly localized aneurysm. In the central figure note the relatively normal dimension of the majority of the ascending aorta with a discrete outpouching anteriorly (arrows) representing a discrete aneurysm. At the upper right inset is a three-dimensionally reformatted CT recorded in the same patient. The plane of the aortic valve is noted by the two inward-pointing arrows and the discrete aneurysm by the single white arrow.

Video 20-22
FIGURE 20.23. Parasternal long-axis transthoracic echocardiogram shows a dilated descending thoracic aorta (Ao, arrows) posterior to the A-V groove. Occasionally, the transthoracic echocardiogram revealing a dilated descending aorta can be the first clue to the presence of a descending thoracic aneurysm or dissection.

Video 20-23

In many cases, mitral valve prolapse with mitral regurgitation and aortic
insufficiency may both be noted. However, if aortic regurgitation is the predominant lesion, the left ventricle may dilate, resulting in reduction of the echocardiographic appearance of mitral valve prolapse and occasionally in a reduction in the visualized mitral regurgitation. After aortic valve replacement, ventricular size diminishes, at which point mitral valve prolapse again becomes apparent and mitral regurgitation of a clinically relevant degree may again be appreciated. For patients undergoing aortic valve replacement, who have mitral valve anatomy that is suspicious for myxomatous change, or in whom this lesion complex is suspected, intraoperative evaluation of mitral valve prolapse and regurgitation should be undertaken after aortic valve replacement so that a combined aortic and mitral valve procedure can be performed if necessary.

Patients with Marfan syndrome are at an increased risk to develop an acute coronary syndrome secondary to spontaneous dissection of a proximal coronary artery. In female patients, spontaneous coronary dissection may occur in association with pregnancy or in the postpartum period and these patients will present with typical features of acute myocardial infarction. Identification of a regional wall motion abnormality in a patient with Marfan syndrome or a closely related connective tissue disease, who is otherwise not at risk of atherosclerotic coronary artery disease, should heighten the awareness of spontaneous coronary dissection as a possible etiology.
FIGURE 20.24. Apical four-chamber view recorded in a patient with an aneurysm of the descending thoracic aorta. The aneurysm is noted by the nearly circular echolucent space directly behind the left atrium. Measurements are as noted. Video 20-24
FIGURE 20.25. Transthoracic echocardiogram recorded from the suprasternal notch (SSN) revealing an aneurysm of the distal arch and descending aorta which is partially filled by thrombus. (Video) coming soon
Video 20-25

In addition to the Marfan syndrome, there are other heritable disorders of connective tissue which can present with similar aortic pathology. These include connective tissue diseases such as the Ehlers–Danlos syndrome and genetic syndromes including Turner syndrome (karyotype XO). The aortopathy of Turner syndrome has become increasingly appreciated and the syndrome also may be associated with an increased prevalence of bicuspid aortic valve. The combination of bicuspid aortic valve and aortic dilation in Turner syndrome may confer substantial risk of dissection, and patients with Turner syndrome and aortic disease probably warrant surveillance and follow-up similar to that provided for patients with the Marfan syndrome. Because the patient with Turner Syndrome is short statured, the general guidelines for intervention based on aortic size should be put in clinical context.

SINUS OF VALSALVA ANEURYSM

Sinus of Valsalva aneurysms most often arise from the right sinus. They are highly variable in size, and the overall length of a sinus of Valsalva aneurysm can reach 3 to 5 cm. Aneurysms arising from the right Valsalva sinus typically protrude into the right atrium where they may be visualized as a filamentous or “windsock” structure. When imaged in their short axis, they may appear as a mobile, circular structure mimicking a cystic mass. Rarely, sinus of Valsalva aneurysm arising from the noncoronary sinus can dissect inferiorly into the interventricular septum where it is noted as a cystic structure within the myocardium. Less frequently, sinus of Valsalva aneurysms protrude into the left atrium. Figures 20.39 through 20.43 were recorded in patients with sinus of Valsalva aneurysms. Note in Figure 20.41 the Valsalva aneurysm arising from the right sinus which protrudes into the right atrium. With only anatomical imaging, one may only note a mobile, filamentous mass in the right atrium. The addition of color Doppler often provides definitive clues as to the nature of these echoes because the “windsock” anatomy of the aneurysm can be more fully appreciated when it contains the abnormal color flow signal.
The major complication of a sinus of Valsalva aneurysm is rupture. The most common location for a sinus of Valsalva aneurysm to rupture is into the right atrium where it results in instantaneous elevation of right heart pressures, jugular venous distension, and a continuous murmur on physical examination. Other complications of a sinus of Valsalva aneurysm include distortion of normal coronary sinus anatomy which can result in malcoaptation of the aortic valve cusps and subsequent aortic insufficiency. Although a sinus of Valsalva aneurysm can be suspected from transthoracic imaging, transesophageal echocardiography provides a definitive diagnosis and is probably essential in all cases for full characterization of the aneurysm. Rarely, a sinus of Valsalva aneurysm may thrombose and mimic an intracardiac mass (Fig. 20.43).

![Image](image_url)

**FIGURE 20.26.** Subcostal imaging of the proximal portion of the descending aorta demonstrating aneurysmal dilation with atheromatous involvement (arrows). The inset at the upper right is the planar CT of the abdominal aorta also illustrating irregular aneurysmal dilation. The inset at the lower right is the three-dimensionally reformatted CT angiogram of the proximal abdominal aorta demonstrating diffuse irregularities and aneurysmal dilation.
An abnormality closely related to the sinus of Valsalva aneurysm is the fibrosa aneurysm. This is an exceptionally rare entity in which an aneurysm forms in the fibrous skeleton of the heart and communicates with one of the Valsalva sinuses via a relatively narrow neck. These frequently are seen as a cystic space between the aorta and the left atrium. As with the sinus of Valsalva aneurysm, transesophageal echocardiography is probably essential for the definitive diagnosis of this entity. Cardiac CT and MRI also play a major role in establishing the diagnosis.
FIGURE 20.27. Transesophageal echocardiogram of a discrete arch aneurysm. The lumen of the arch is noted by the double-headed arrow. The remaining horizontal and vertical arrows outline the boundary of the discrete aneurysm which is filled with a thrombus (Th). [Video 20-27]
FIGURE 20.28. Transesophageal echocardiogram recorded in a patient with a discrete aneurysm (An) of the aortic arch. The upper panel is recorded at a 0-degree imaging plane and reveals the arch with a saccular aneurysm. The lower panel is recorded in the same imaging plane with color flow Doppler revealing sluggish flow into and out of the saccular aneurysm.
FIGURE 20.29. Transesophageal echocardiograms of descending thoracic aortic aneurysms. 

A: Note the flow containing lumen of the aorta. The black vertical and remaining horizontal white arrows delineate the absolute external boundary of the aorta and the maximal dimensions of the aneurysm which is largely filled with a thrombus and atheroma. 

B: A descending thoracic aortic aneurysm. The double-headed white arrow outlines the dimension of the aortic lumen. The double-headed black arrow denotes a thrombus and atheroma filling an aneurysmal
cavity. The total dimension of the aorta would be the summed length of *black and white arrows.*
FIGURE 20.30. Transesophageal echocardiogram recorded at 0-degree imaging plane in the descending thoracic aorta at 30 cm from the incisors in a patient with severe, complex atheromatous disease of the aorta. Note the aneurysmal dilation of the aorta and the complex, protruding atheroma into the lumen (white arrows). Also note the lucency within the posterior aspect of the atheroma representing fracture of the atheromatous plaque (dark arrow).
Video 20-30
FIGURE 20.31. Transesophageal echocardiogram recorded in a patient with a contained rupture of the aortic arch associated with a previously known aneurysm. Note the marked distortion of aortic anatomy and the complex echoes external to the boundary of the aorta representing hemorrhage into the mediastinum.
AORTIC DISSECTION

Acute aortic dissection occurs with an annual incidence of 10 to 30 per million. It is a syndrome that results in sudden onset of severe chest and/or back pain with a wide range of secondary cardiovascular and physiologic abnormalities. Aortic dissection, intramural hematoma (IMH), atherosclerotic plaque rupture, and aneurysm rupture all have a similar clinical presentation and are often referred to as an “acute aortic syndrome.” Imaging with echocardiography, CT, or MRI is required to distinguish the presentations. Dissection typically occurs in the setting of pre-existing aortic dilation, Marfan syndrome, or hypertension. Currently, it is felt that aortic dilation of more than 55 mm is a definite risk factor for dissection; however, approximately 40% of dissections occur in aortas smaller than this threshold. The aorta can dissect at any point along its length. Aortic dissection is characterized as one of two variants, each of which has a similar presentation with respect to symptoms (Fig. 20.44).
FIGURE 20.32. Transesophageal echocardiogram recorded at the apex of the arch in a patient with a ruptured aneurysm. Note the atheromatous thickening of the aorta and the vague soft tissue density echoes just beneath the apex of the arch representing fresh thrombus related to rupture. Video 20-32
FIGURE 20.33. Parasternal long-axis view of a patient with Marfan syndrome and a dilated ascending aorta. At the upper left inset is an expanded short-axis view, recorded at the level of the sinus of Valsalva. Note the asymmetric dilation of the left and noncoronary sinuses compared to the right coronary sinus. Video 20-33
FIGURE 20.34. Transthoracic images recorded in a patient with Marfan syndrome and a markedly dilated ascending aorta. The upper panel is a parasternal long-axis view revealing marked dilation of the ascending aorta, predominantly at the level of the sinuses. The lower panel is an apical long-axis view in which the dilated ascending aorta can also be visualized. Note that the
dilation of the ascending aorta results in distorted off-axis imaging from this transducer position.

coming soon

Video 20-34A

coming soon

Video 20-34B
FIGURE 20.35. Transesophageal longitudinal view of the ascending aorta in a patient with Marfan syndrome. **A:** Note the dilation of the proximal aorta, confined to the sinuses of Valsalva with relatively normal dimensions of the sinotubular junction. **B:** Color flow Doppler demonstrates mild central aortic regurgitation, which is a result of malcoaptation of the aortic cusps.
FIGURE 20.36. Transesophageal longitudinal view of the ascending aorta recorded in a patient with Marfan syndrome. **A:** Note the marked dilation of the aortic sinuses with some tapering at the level of the sinotubular junction. Note, however, that the sinotubular junction dimension still exceeds the annular dimension by a substantial degree. Note also the malcoaptation of the aortic cusps with the normal position of the left cusp (*long arrow*) and malcoaptation of the noncoronary cusp (*short arrow*). **B:** The malcoaptation results in substantial aortic insufficiency, which is highly eccentric, the initial portion of which is directed posteriorly (top to bottom).
FIGURE 20.37. Cardiac magnetic resonance imaging recorded in a patient with Marfan syndrome and aneurysmal dilation of the proximal aorta. The main illustration on the right shows the ascending aorta, arch and descending aorta to just past the diaphragm. Note the selective dilation of the sinuses and proximal aortic root. The figure at the lower left is a rotated view of the ascending aorta, from which a measurement navigator can be steered through various areas of the aorta for more refined measurements. In this illustration, the navigator has been located at the maximum area of dilation in the mid-ascending aorta and reveals a relatively circular aorta with a diameter of 49.2 mm. The upper left panel is a planar image of the ascending aorta from which additional measurements can be made.

Classic aortic dissection consists of a tear of the intima into the medial layer allowing communication between the pressurized flow lumen and the medial space. This results in propagation of a column of blood which then further dissects the intima from the media. Propagation can be both proximal and distal to the initial intimal tear. Classic aortic dissection typically begins either at the area of the ligamentum arteriosum and propagates distally or starts in the ascending aorta and propagates distally. On occasion, patients may present with a limited intimal tear without dissection. This variant may
be associated with only very subtle abnormalities on transesophageal echocardiography or other imaging techniques.

The second pathophysiology for aortic dissection is IMH which represents 5% to 10% of aortic dissections. The clinical presentation with respect to symptoms is identical to that of typical dissection, and most authorities believe that it requires the same therapy. Hemorrhage within the medial layer dissects proximally or distally to a variable degree, without rupturing into the lumen. Intramural hemorrhage may progress to rupture into the adventitia, resulting in typical aortic dissection in up to 16% of cases. The clinical presentation, prognosis, and forms of therapy for these mechanisms of acute aortic pathology are similar. A more recently recognized variant of acute aortic pathology is the so-called IMH without dissection. In this instance, a relatively limited area of acute hemorrhage occurs in the medial layer but does not propagate.

Aortic dissections are characterized by their location using either the Stanford or DeBakey schemes. Figure 20.45 depicts the two different characterization schemes. The crucial clinical factor in aortic dissection is whether it involves the ascending aorta (Stanford type A or DeBakey I or II). These patients have a greater likelihood of subsequent rupture, pericardial effusion, aortic insufficiency, and coronary involvement, all of which may be lethal. Ascending aortic dissection is considered a surgical emergency for which rapid, accurate diagnosis is essential and in which transesophageal echocardiography plays a crucial role. While urgent or emergent surgery is the treatment of choice for acute type A dissection, dissection isolated to the descending thoracic aorta (Stanford type B or DeBakey III) is typically managed medically unless complications occur.

**Echocardiographic Diagnosis of Aortic Dissection**

Because transthoracic echocardiography visualizes only a limited area of the ascending aorta, it is not considered an adequate diagnostic tool for exclusion of aortic dissection. However, when an intimal flap is confidently detected on transthoracic imaging, the diagnosis of dissection, but not its full extent, can be confidently established. Other imaging techniques such as transesophageal echocardiography, CT, or MRI are necessary to fully characterize its extent. Figures 20.46 and 20.47 are transthoracic echocardiograms recorded in
patients in which the dissection flap can be identified. Additional imaging from the aortic arch (Fig. 20.48) and imaging of the descending thoracic aorta (Fig. 20.49) can supplement these views. The transthoracic echocardiogram can provide additional confirmatory information such as detection of proximal aortic dilation or aortic insufficiency (Fig. 20.46). Pre-existing aortic dilation is usually present in patients with ascending aortic dissection. Identification of normal aortic dimensions and geometry and absence of aortic insufficiency from a transthoracic echocardiogram are circumstantial evidence against the presence of typical aortic dissection in the ascending aorta, but do not fully exclude the diagnosis, nor do they exclude IMH. At present, the widespread availability of CT in, or immediately adjacent to the emergency department, has made it the most common imaging technique for the initial evaluation of known or suspected aortic dissection. Transesophageal echocardiography remains a major diagnostic tool in the detection of aortic dissection, and large series have suggested that it is used in approximately two-thirds of patients with known and suspected acute aortic dissection. It is often used as a second imaging test to evaluate the aortic valve and to determine the mechanisms of aortic insufficiency. It can be performed in critically ill patients in intensive care units, the emergency department, and the operating room and provides a definitive diagnosis. Complications such as pericardial effusion, aortic insufficiency, pseudoaneurysm, adventitial hematoma, and rupture can all be identified.
FIGURE 20.38. Transthoracic parasternal long-axis echocardiogram recorded in a patient with Marfan syndrome and mild dilation of the ascending aorta, predominantly at the level of the sinuses of Valsalva. In the central figure note the aortic dilation and the myxomatous mitral valve with mitral valve prolapse (arrows). In the inset at the lower right note the expanded view of the proximal aorta and the dilated aorta at the level of the sinuses. In the inset at the lower left, recorded in systole note the moderate mitral regurgitation related to the concurrent mitral valve prolapse. [Video 20-38 CFD]
FIGURE 20.39. Parasternal long-axis view recorded in a patient with a small aneurysm of the right sinus of Valsalva. In the central figure note the approximate 1-cm deep aneurysm protruding off the aorta into the right ventricular outflow tract. The inset at the upper left was recorded with color Doppler and confirms the absence of any communication between the aneurysm in the right ventricle. The inset at the upper left is an expanded short-axis view at the level of the sinuses of Valsalva demonstrating the isolated aneurysmal dilation of the right sinus of Valsalva.
FIGURE 20.40. Transesophageal echocardiogram recorded at 134 degrees in a patient with a ruptured sinus of Valsalva aneurysm. In A, note the filamentous structure arising from the right coronary cusp and protruding into the right
ventricular outflow tract (arrows). B is the same image with color Doppler imaging, revealing a highly turbulent jet from the aorta into the right ventricular outflow tract (arrow) which is continuous, consistent with the rupture of a sinus of Valsalva aneurysm into the outflow tract. [Video 29-40A]

coming soon

Video 29-40A

coming soon

Video 20-40B
FIGURE 20.41. Transesophageal echocardiogram of a sinus of Valsalva aneurysm arising from the right coronary sinus. A: Recorded at 43-degree probe rotation. Note the normal size and geometry of the left (L) and non- (N) coronary sinuses and the elongated windsock aneurysm rising off the right coronary sinus (arrows) and protruding into the right atrium. B: Recorded at a 118-degree image plane the aneurysm now appears as a highly mobile, cystic structure in the right atrium (long arrow). Note the position of the tricuspid valve (TV) as well.

Video 20-41

FIGURE 20.42. Transesophageal echocardiogram recorded in a patient with a large sinus of Valsalva aneurysm which has ruptured into the left atrium. In A,
recorded in a longitudinal view (116 degrees), note the large, relatively wide-mouthed aneurysm protruding directly into the left atrium (inward-pointing arrows). 

B was recorded with color Doppler imaging at an expanded view and confirms rupture of the aneurysm into the cavity of the left atrium.

Video 20-42A

Video 20-42B
Video 20-43

When evaluating the ascending aorta, it is not uncommon to encounter artifactual echoes within the aortic lumen. A skilled echocardiographer should not have difficulty in separating these from aortic dissection. Clues to artifact versus true dissection include random mobility of a true dissection flap as opposed to a more rigid and fixed location seen with artifact. Artifacts not infrequently will arise as a side lobe from the sinotubular junction, and their intensity will progressively diminish in the lumen (Fig. 20.50), whereas a true dissection flap will not lose its echo intensity along its course. Color flow imaging can be very useful for demonstrating margination of flow by a true dissection flap (Fig. 20.51), whereas an artifact will not affect the distribution of the color flow signal.
FIGURE 20.44. Schematic representation depicts the forms of acute aortic pathology. **Upper panels:** Depicts classic aortic dissection in which there is a tear of the intima from the media. The column of blood propagates proximally and distally, and there may be multiple communication points between the lumen and the intima media space. **Lower panels:** The spontaneous intramural hematoma variant of aortic dissection in which there is rupture of the vasa vasorum resulting in hematoma in the medial space without communication between the lumen and the hematoma is depicted. The two right-hand schematics depict the same phenomenon in a short-axis view of the aorta.
An additional confusing echo can be a superimposed venous structure coursing adjacent to the aorta. Typically, this represents the left brachiocephalic vein as it courses superior to the aortic arch. The combination of the brachial cephalic vein and aorta creates a tubular echo larger than that of the normal aorta with a linear structure running
longitudinally. This can occasionally be confused with a dilated aorta with a dissection flap. Color flow imaging will reveal a color flow signal on both sides of the linear echo. Careful scrutiny of the color flow signal will demonstrate that the larger lumen contains pulsatile arterial flow, and the smaller lumen contains continuous flow in a typical venous pattern (Fig. 20.52). An additional method for identifying this as a venous structure is to inject agitated saline contrast into a left upper extremity vein, at which point one can see the contrast within the smaller venous structure thereby definitively identifying it as the brachiocephalic vein.
FIGURE 20.46. Transthoracic parasternal long-axis view with and without color flow Doppler imaging in a patient with acute type A dissection. Note the marked dilation of the ascending aorta which is nearly ubiquitous in type A dissection. The rightward-pointing arrows in the left ventricular outflow tract denote the actual aortic valve. The leftward-pointing arrows denote portions of the intimal flap. Note
the significant amount of aortic regurgitation which is due to malcoaptation of the aortic valve. DAo, descending aorta. [0]
FIGURE 20.47. Parasternal long-axis view in systole (A) and diastole (B) in a patient with a type A dissection. Note the remnants of the intimal flap within the lumen of the dilated ascending aorta (arrows). In diastole, the intimal flap prolapses through the aortic valve into the left ventricular outflow tract. This is one of the several mechanisms for developing aortic insufficiency in acute aortic
FIGURE 20.48. Suprasternal notch view of a patient with a dissection of the arch and proximal descending thoracic aorta. In A, note the takeoff of the great vessels (upper arrows) and the linear echo within the lumen of the aorta (leftward-pointing arrows) representing the intimal flap of the dissection. In B, recorded with color Doppler, note the flow confined to the more medial true lumen and the absence of flow in the laterally located false lumen. The inset is continuous-wave Doppler recorded from the suprasternal notch revealing a peak gradient of 2.8 m/s in the true lumen.
FIGURE 20.49. Subcostal imaging of the proximal abdominal aorta in a patient with a type B dissection. In the central figure note the linear echo running the extent of the visualized aorta in this view representing the dissection flap. At the lower left inset note the color Doppler image revealing flow in both lumens with a higher volume and velocity of flow in the more inferior lumen. The side panels at the right are pulsed Doppler profiles from the superior and inferior lumens demonstrating a reduced velocity in the more anterior lumen. [Video 20-49 CFD]
Figures 20.53 through 20.60 were recorded in patients with acute type A aortic dissections. Color flow imaging can be used to identify the communication points between the true and false lumens. It should be emphasized that the earlier concept of entry and exit points, with the dissection extending between these two points is not accurate. Most dissections have multiple communication points between the true and false lumens at areas where the intima has been sheared from the media. It is important to recognize the larger communication points because they have relevance for surgical repair. Echocardiographic imaging of all aspects of the aortic arch may be problematic in some patients. Arch involvement raises the possibility of involvement of the cerebral and upper extremity vasculature. Compromise of these vascular territories should be specifically considered and CT or MRI angiography is often essential. Figures 20.61 through 20.63 were recorded in patients with acute arch dissection. CT can provide valuable additional imaging of the arch (Fig. 20.64).
FIGURE 20.50. Transesophageal echocardiogram recorded in a longitudinal plane of the ascending aorta. This image shows a common artifact that could be confused with a dissection. This is a classic side lobe artifact arising (small arrows) from a rather bright echo at the sinotubular (vertical arrow) resulting in an unnaturally curvilinear echo extending along the direction of the scan plane lines within the lumen of the aorta. Note in the lower panel that with color flow imaging there is no margination of flow by the linear echo, helping to confirm that this is artifact rather than a true dissection flap. [Video 20-50A]
Figures 20.65 through 20.68 were recorded in patients with type B dissections. In the descending thoracic aorta, there is frequently a concurrent atherosclerotic component. It can occasionally be difficult to separate the true from the false lumen. Several clues enable accurate distinction of the two. In the ascending aorta, there is usually little confusion because one can appreciate the outlet of blood through the aortic valve which, by definition, will be into the true lumen. Distinction between the true and false lumens may sometimes be more problematic in a short-axis view or in the descending thoracic aorta. Clues that enable accurate identification of the true lumen include the fact that it will expand with systole as blood is ejected into it. It may have a more regular shape which may be either circular or oval. Often, especially in the descending thoracic aorta, the true lumen is the smaller of the two lumens. The false lumen is often filled with swirling homogeneous echoes, representing stasis of blood or occasionally with frank thrombus. Finally, the shearing of the intima from the media often results in small fibrinous tags of tissue in the false lumen which represent small tissue remnants where the intima has been sheared from the media (Fig. 20.68).
FIGURE 20.51. Transesophageal echocardiogram recorded in a longitudinal view of the ascending aorta in a patient with an acute type A aortic dissection. A: Note the thickened AV and the intimal flap at the level of the sinotubular junction. B: Note the aortic insufficiency (AI) jet which arises centrally and is functional related to dilation at the level of the sinotubular junction. C: A systolic frame in which the blood being ejected from the left ventricular outflow tract is constrained by the aortic dissection flap.

Video 20-51

Video 20-51B
FIGURE 20.52. Venous flow adjacent to the aortic arch, mimicking aortic dissection (arrow) (A). This represents normal venous communication from the superior vena cava with flow toward the heart. It is common to encounter this space which can occasionally be confused for aortic dissection. As the structure contains normal venous flow, Doppler will demonstrate a continuous color signal that should not be confused with flow into a false lumen (B). Asc Ao, ascending
In skilled hands, the accuracy of transesophageal echocardiography for the detection of aortic dissection is exceptionally high and equivalent to that of the competing techniques such as CT and MRI. In actual practice, false positives most commonly occur when using older generation single or biplane probes or when confusion exists between an artifactual echo protruding into the aorta and a true dissection flap (Fig. 20.50). False-negative examinations are exceedingly uncommon but occasionally occur near the proximal portion of the arch which represents a relative blind spot for transesophageal echocardiography. Most aortic dissections, however, extend for a fairly long portion of the aorta, and a dissection localized only to this limited blind spot is quite uncommon. Although three-dimensional scanning of the aorta may provide a unique and different imaging perspective (Fig. 20.68), it has not been consistently shown to provide incremental clinical information.

**Intramural Hematoma**

IMH represents a variant of acute aortic dissection in which hemorrhage occurs into the medial layer which may propagate both circumferentially and longitudinally, but does not rupture into the lumen. It is distinguished from typical aortic dissection in that there is no communication point between the media and the true lumen. Presenting signs and symptoms as well as
management are virtually identical to that for typical aortic dissection.
**FIGURE 20.53.** Transesophageal echocardiogram recorded in a longitudinal plane in two patients with more localized type A dissection. **A:** Note the relatively normal aortic dimensions and the very limited dissection flap (arrow). A single communication point (open arrowhead) can be seen as well. **B:** A similarly localized aortic dissection (white arrow) is revealed. In this instance, however, note the fairly discrete aneurysmal bulge of the anterior wall of the aorta (black arrows). This was subsequently confirmed at the time of surgery to represent a partial rupture of the aortic wall and small aortic pseudoaneurysm. 

**Video 20-53**

**FIGURE 20.54.** Transesophageal echocardiogram recorded in a longitudinal view of the aorta in a patient with an acute type A dissection. In **A**, the outward-pointing arrows denote the aortic valve cusps which are open in systole. The inward-pointing echoes note the margins of the dissection flap which is circumferential in nature. There is a central true lumen (TL) surrounded by a circumferential false lumen. **B** was recorded with color flow Doppler and confirms the presence of systolic flow confined to the true lumen (double-headed arrow).
Video 20-54A

coming soon

Video 20-54B

coming soon
Recent studies have confirmed the interrelationship of IMH and typical aortic dissection with an intimal flap and have demonstrated that up to 15% of lesions initially identified as an IMH may, over a relatively brief period of time, convert to a typical aortic dissection with an intimal flap and entrance points into the lumen.

On imaging, IMH is defined as an area of crescentic thickening of the wall more than 7 mm thick. By definition, there is no active flow within the “lumen” and no communication point with the true lumen will be noted. IMH, if localized, may present with only subtle echocardiographic findings and must be distinguished from an area of uncomplicated, smooth atheroma. Atheroma, typically, will have evidence of intimal thickening as well as possible calcification within the wall. Figures 20.69 and 20.70 were recorded in patients with documented IMH of the aorta. While transesophageal
echocardiography provides a very high-resolution image of the intima, medial, and adventitial layers, techniques relying on purely luminal visualization, such as aortography, may not accurately identify IMH.
FIGURE 20.56. Transesophageal echocardiogram recorded in a short-axis view of the proximal ascending aorta in a patient with a circumferential type A dissection. **A:** Note the circular aorta containing a second circular structure that is the intimal flap which now defines a circular true lumen (TL) surrounded by a completely circumferential false lumen (FL). **B:** Note that color flow in systole is confined to the smaller inner true lumen. [Video 20-56A](coming soon) [Video 20-56B](coming soon)
FIGURE 20.57. Transesophageal echocardiogram recorded in the longitudinal view of the proximal ascending aorta in a patient with acute type A dissection and marked intimal disruption. The aortic valve is noted by the two rightward-pointing short arrows. Within the lumen of the dilated ascending aorta note the multiple complex intimal flaps (arrows) with multiple areas of tear from the media. In the real-time image note the highly mobile serpiginous nature of the complex intimal flap within the aorta. 

Video 20-57
Complications and Natural History of Aortic Dissection

In addition to diagnosing acute and chronic aortic dissection, echocardiography can be used to document the presence of multiple complications. Common complications of aortic dissection include pericardial effusion with or without hemodynamic compromise, complete or
partial rupture of the aorta (Fig. 20.71) with periaortic or adventitial hematoma, compromise of aortic side branches, compromise of coronary arterial circulation, aortic pseudoaneurysm, and aortic insufficiency.

Pericardial effusion seen in the presence of acute type A aortic dissection is an ominous sign and generally implies rupture of the dissection into the pericardial space. These patients may present with severe hemodynamic compromise related to the compressive pericardial effusion. Anecdotal case reports have suggested that pericardiocentesis to reduce the intrapericardial pressure may be counterproductive and simply result in more aggressive bleeding into the pericardial space and patient demise. Detection of a pericardial effusion with hypotension in the presence of an acute type A dissection should prompt emergency corrective surgery. On occasion, smaller effusions are noted which, at the time of direct inspection, are not frankly hemorrhagic and may represent a “sympathetic” secondary passive effusion rather than evidence of frank rupture. Involvement of various arterial side branches occasionally can be documented with transesophageal echocardiography; however, CT or magnetic resonance angiography is superior for this purpose.

FIGURE 20.59. Transesophageal echocardiogram recorded in a short axis of the ascending aorta from the same patient depicted in Figure 20.58 confirming the presence of a bicuspid aortic valve. Note the “fish mouth” opening end systole (A) with the commissures joining the aortic wall at the 2 o’clock and 7 o’clock positions. B: Recorded 2 cm distally in the aorta and reveals the proximal margin of the dissection flap (5 o’clock to 8 o’clock).
FIGURE 20.60. Transesophageal echocardiogram recording a longitudinal view of the ascending aorta in a patient with an acute type A dissection. A: Note the dilation of the aorta at the sinuses, sinotubular junction and its ascending portion. A relatively normal AV is noted with cusps in an open position. Note the thin, convoluted intimal flap (arrows) within the lumen of the aorta, the mobility of which is appreciated in the real-time image. B: Recorded in the same view with color flow Doppler and demonstrates the significant secondary aortic insufficiency present in this instance.
FIGURE 20.61. Transesophageal echocardiogram recorded in a patient with an acute dissection involving the arch of the aorta. A: Recorded at 0 degrees. Note
the convoluted intimal flap \textit{(arrows)} within the lumen of the aorta and the arch. \textbf{B:} Recorded in a short axis of the distal arch and again depicts a convoluted intimal flap \textit{(arrows)} with multiple communication points. The origin of the left subclavian (LSC) artery is also visible. 

\textbf{Video 20-61}

\textbf{Video 20-61a}
FIGURE 20.62. Transesophageal echocardiogram recorded in a patient with a type A dissection with extension into the arch. This view was obtained in a short axis of the arch of the aorta, in which the takeoff of the left subclavian (LSC) artery can be seen. **A:** Note the convoluted and mobile intimal flap (*arrows*) present in the arch and partially obstructing the takeoff of the left subclavian artery. **B:** Recorded with color flow Doppler and depicts the complex flow patterns around the intimal flap. [Video 20-62](coming soon) [Video 20-62c](coming soon)
FIGURE 20.63. Transesophageal echocardiogram recorded in a patient with arch involvement of a dissection that extended from the sinotubular junction through the arch. These images were recorded in a short-axis view of the arch. **A:** Note the total dimension of the arch which is approximately 6 cm. There is a complex dissection present with the appearance of one true lumen (TL) and two false lumens (FLs). **B:** With color flow Doppler imaging, notice that flow is confined only to the central true lumen and is excluded from the more peripheral false lumens.

Aortic insufficiency is a common complication of type A aortic dissection. It is present in up to 70% of the cases but may be clinically recognized in only half. Echocardiography has identified several different mechanisms for aortic insufficiency that have relevance for surgical correction (Fig. 20.72).
Aortic insufficiency can occur when the dissection extends into the sinus of Valsalva and disrupts the base of an aortic cusp (Fig. 20.73). This results in abnormal aortic valve coaptation. More commonly, aortic dissection results in dilation of the sinotubular junction and valve cusp malcoaptation on this basis (Fig. 20.74). In this instance, the aortic valve itself is anatomically normal and aortic insufficiency is functional and related to dilation of the aortic root. This mechanism of aortic insufficiency is usually amenable to valve-sparing surgery in which restoration of the normal sinotubular junction results in correction of aortic insufficiency. A final mechanism that is uniquely identified by transesophageal echocardiography consists of prolapse of an aortic dissection flap through the aortic orifice (Fig. 20.75). The flap then becomes a conduit for insufficiency of the aortic valve.

Therapy for acute type A aortic dissection typically involves immediate surgical correction. The goal of surgery for aortic dissection is to arrest further propagation of the aortic dissection and to resect irreversibly damaged aortic tissue, replacing it with a prosthetic graft. For ascending aorta graft placement, the ostia of the left main coronary and right coronary arteries are resected from the native aorta and sutured to the aortic graft. Therefore, it is important to evaluate left ventricular function in the operating room, looking for wall motion abnormalities after repair. In high-volume centers, the aortic valve is preserved in 75% of aortic dissection repair. In these cases, postoperative transesophageal echocardiography is important to confirm aortic valve competence (Fig. 20.76).
FIGURE 20.64. Computed tomography recorded in a patient with an acute type A dissection involving the arch and extending into the descending thoracic aorta. On the left, the images are obtained at the level of the ascending (Asc Ao) and descending thoracic aorta (DAo) and, in each instance, the intimal flap is clearly visualized and a small true lumen (TL) seen to be more contrast enhancing than the false lumen. The image on the right was recorded through the arch of the aorta and again reveals similar findings with respect to the true and the false lumens.
FIGURE 20.65. Transesophageal short-axis views of aortic dissection from four different patients. A: Note the relatively preserved circular geometry of the aorta (Ao) which is separated into a true lumen (TL) and substantially larger false lumen (FL). Note that the false lumen is filled with stagnant swirling blood. B: Recorded in a patient with a type B dissection. This image was recorded at a site in the aorta not involved by the dissection. Note the normal size circular aortic lumen and the much larger homogeneous mass (black arrowheads) circumferentially surrounding the aorta. This represents a dissecting adventitial hematoma (AH) external to the aorta at this point. C: A type B dissection, in which the true lumen and false lumen are of more equal size, is demonstrated. Note also in this instance the atheromatous involvement of the anterior wall of the aorta. D: A type B dissection with a smaller, upper true lumen and a much larger false lumen. Note that the false lumen again contains stagnant swirling blood with some areas of lucency.
FIGURE 20.66. Transesophageal echocardiogram recorded at the mid-descending thoracic aorta in a patient with an acute type B dissection. In the central figure note the dilated aorta (arrows) with the crescent-shaped true lumen in the lower half of the image (×). In the inset, recorded with color flow Doppler, note the flow within the true lumen (×) and the communication point with flow out of the true lumen into the larger false lumen (arrow). Video 20-66 CFD
Type A dissection involving the ascending aorta may also involve the arch and a varying extent of the descending aorta. The immediate surgical approach for an extensive type A dissection is to approach the ascending aorta which is the most likely cause of hemodynamic instability. From a technical perspective, it is not possible to simultaneously approach the ascending and descending thoracic aortas with a surgical approach. In this situation, temporizing measures such as fenestration and stenting of the descending thoracic aorta to protect compromised organs is often undertaken. Following correction of the ascending portion of an extensive dissection, there will remain evidence of a type B dissection which, depending on its location and characteristics, may include a thrombosed false lumen or continued patency of a false lumen with multiple entrance and exit points. Limited communication points in the descending aorta may still be visualized after surgical repair. In a substantial number of these patients, chronic thrombosis of the false lumen occurs. Figure 20.77 was recorded in a patient with type B dissection which resolved over time. Figure 20.78 was recorded in a patient after surgical correction of acute aortic dissection with prosthetic graft material.
FIGURE 20.67. Transesophageal echocardiogram recorded in the short axis at two different levels in a patient with acute type B aortic dissection. A–D: Note the dilation of the aorta and the relatively larger false lumen (FL) compared with the true lumen (TL). A, B: No communication point is visualized and flow is confined exclusively to the true lumen. There appears to be a partially thrombosed component as well. C, D: Recorded at a different level and reveals an intimal flap with a 1-cm diameter communication point with obvious systolic flow noted in the image with color flow Doppler.
FIGURE 20.68. Transesophageal echocardiogram recorded in a patient with a chronic type B aortic dissection. These images were recorded at approximately 35 cm from the incisors. The central image is a real-time three-dimensional image of the aorta in a transverse view. Note the dilated, aneurysmal aorta. At the lower aspect of the image note the circular true lumen (TL) surrounded by the larger crescent-shaped false lumen (FL). The arrow points to a mobile remnant of the medial tissue in the false lumen. At the upper left inset is the standard two-dimensional image at the same level depicting a nearly circular true lumen within the larger aneurysmally dilated aorta. The inset at the lower left is a color Doppler flow demonstrating robust flow within the true lumen and absence of significant flow in the false lumen. The inset at the upper right was recorded 5 cm more proximal and again demonstrates a small true lumen, and at this level a fully thrombosed false lumen.
Video 20-68

coming soon

Video 20-68CFD
FIGURE 20.69. Transesophageal echocardiogram recorded at 0° and 116° in the descending thoracic aorta in a patient with a spontaneous intramural hematoma. Notice the relatively normal circular aortic geometry and the crescent-shaped filling defect from approximately 2 o’clock to 10 o’clock. With close scrutiny, one can appreciate the intima (arrows) which has lifted off the medial layers with the hematoma within the intima/medial space. There was no evidence of communication between the lumen and intima. The small insets are computed tomographic images from the same patient depicting classic findings of intramural hematoma as well.
AORTIC ATHEROMA

Atherosclerosis of the aorta is frequently encountered during transesophageal echocardiography. Occasionally, it can also be identified from a suprasternal notch view (Fig. 20.79) or subcostal view (Fig. 20.26). It is most common in older patients or in those individuals with a history of tobacco use, hypertension, and elevated cholesterol, and it may be an integral component of atherosclerotic aneurysm. It is also not infrequently encountered in patients in whom a cardiovascular source of embolus is suspected. Atheromas of the aorta are characterized by location and topographic characteristics. They are
most common in the descending thoracic aorta and arch and far less frequently encountered in the ascending aorta.

Atheroma can be characterized as symmetric and crescentic, in which case it creates a smooth homogeneous crescent filling a portion of the aortic lumen, protruding or complex. Symmetric atheroma can be confused for intramural hematoma; however, the former is more likely to have intimal thickening and areas of calcification. Complex atheroma is defined as atherosclerotic disease with pedunculated or mobile components. Typically, a threshold of 4 mm of protrusion into the lumen has been used for this definition. Atherosclerotic disease with protruding and mobile components is more likely to be associated with cardioembolic disease than is smooth, crescentic atherosclerotic involvement. Complications of significant atherosclerosis of the aorta include aneurysm formation and penetrating ulcer of the aorta. Clinically, penetrating ulcer presents in a manner similar to that of aortic dissection. Figures 20.80 to 20.87 depict aortas with varying degrees and types of atherosclerotic involvement. Real-time three-dimensional transesophageal echocardiography often highlights the remarkable complexity of the more severe forms of atheroma.
FIGURE 20.70. Transesophageal echocardiogram in the longitudinal view of the ascending aorta in a patient with a type A intramural hematoma. Note the 1-cm homogeneous thickening of the posterior aspect of the aortic wall (small arrows) representing spontaneous intramural hemorrhage into the medial layer extending from the annulus to past the sinotubular junction.

Video 20-70
FIGURE 20.71. Transesophageal echocardiogram recorded at a 64-degree angle in the ascending aorta in a patient with aortic dissection leading to pseudoaneurysm and subsequent rupture into the RA. A: Note the distorted contour of the Ao and the extravascular space representing the pseudoaneurysm (PA) with a thin margin which is ruptured into the RA (arrow). B: Note the disorganized, complex systolic flow pattern from the PA into the RA. [Video 20-71A]
FIGURE 20.72. Schematic representation of mechanisms of aortic insufficiency in acute aortic dissection and disease of the proximal aorta. Multiple mechanisms can be responsible for aortic insufficiency including effacement of dilation of the sinotubular junction resulting in malcoaptation of the aortic valve (A), aortic dissection in the presence of intrinsic aortic valve disease (B), actual disruption of the insertion of an aortic cusp (C), and prolapse of a portion of the intimal dissection flap through the aortic valve which serves as a conduit for aortic regurgitation (D).
Transesophageal echocardiogram recorded in a patient with an acute type A dissection. This image was recorded in a longitudinal view of the ascending aorta. In the central figure note the nearly circumferential intimal flap within the lumen of the mildly dilated aorta (inward-pointing arrows). Note the prolapse of the noncoronary cusp into the left ventricular outflow tract (downward-pointing arrow). At the upper right inset is a short-axis view of the aortic sinuses. Note the dissection which encompasses the 6 o’clock to 12 o’clock area of the proximal aorta. The proximal aorta is denoted by the bold inward-pointing arrows and the margins of the intimal flap by the smaller outward-pointing arrows. At the lower left inset is a color Doppler image confirming the presence of aortic insufficiency related to disruption of the coronary cusp at the level of the annulus.
FIGURE 20.74. Transesophageal echocardiogram in a longitudinal view of the ascending aorta recorded in a patient with an acute type A dissection. The central illustration is recorded with color Doppler and reveals central aortic insufficiency related to dilation of the sinotubular junction. The panel at the lower right is an expanded view of the ascending aorta again demonstrating dilation of the sinotubular junction (long arrows) and an anatomically unremarkable aortic valve. The smaller arrows denote the dissection flap which arises from the sinotubular junction but does not extend into the level of the sinuses. In this instance, the aortic insufficiency is functional and related to secondary dilation of the sinotubular junction. Of note, this may be either pre-existing, related to ascending aortic aneurysm, or acute related to dilation secondary to the dissection.
FIGURE 20.75. Transesophageal echocardiogram recorded in a patient with acute type A dissection and severe aortic insufficiency. A: Recorded in a longitudinal (113-degree) view of the ascending aorta in diastole. Note the portion of the dissection flap (white arrow) that is prolapsing through the aortic annulus into the left ventricular outflow tract. B: The accompanying color flow image was recorded in diastole. Note the color flow jet that fills the entire left ventricular outflow tract and is flowing through the prolapsing intimal flap. There is a communication point within the intimal flap resulting in flow of blood directly into the left ventricle (white arrows). Note that the amount of blood escaping from the prolapsing flap is substantially less than that confined by the flap in the left ventricular outflow tract.
Video 20-75a

coming soon

Video 20-75b

coming soon
FIGURE 20.76. Transesophageal echocardiograms recorded in a patient with an acute type A dissection and secondary aortic insufficiency who subsequently underwent a valve-sparing repair procedure. **A:** A longitudinal view of the ascending aorta recorded at the time of acute dissection. Note the dilation of the ascending aorta and the mobile intimal flap at the level of the sinotubular junction (arrow). **B:** Note the moderate aortic insufficiency present at the time of acute dissection. **C:** Recorded following a valve-sparing repair. Note the absence of any significant residual aortic insufficiency.
FIGURE 20.77. Transesophageal echocardiogram recorded in the short axis in the descending thoracic aorta in a patient with an acute type B dissection. A: Recorded at the time of acute presentation and reveals a smaller flow containing true lumen (TL) and a larger false lumen (FL) with significant stasis of blood flow. B: Recorded 3 months later at the same level in the descending thoracic aorta (note scale change) and documents resolution of the type B dissection. The intimal flap and wall are now opposed with a substantially smaller, fully thrombosed false lumen.
Both CT angiography and magnetic resonance angiography can be used to characterize atheroma as well. CT can easily identify both simple and complex atheroma and, with three-dimensional reconstruction techniques, clearly delineate its full extent. Contrast-enhanced CT is an accurate method for the detection of penetrating ulcer.

MISCELLANEOUS CONDITIONS

Coarctation of the Aorta
Aortic coarctation and other associated congenital lesions are discussed in
Chapter 19. Coarctation can be screened for using the suprasternal view (Fig. 20.88). Complete imaging of the aortic arch and proximal descending aorta with echocardiography is often problematic, CT or MRI are required in the majority of instances for accurate characterization of aortic coarctation and can be used to delineate the subsequent collateral network and concurrent secondary aneurysmal dilation with precision (Fig. 20.89).
FIGURE 20.78. Transesophageal echocardiogram recorded in a patient following aortic graft repair of an acute aortic dissection. A: Note the graft material extending from the annulus to the ascending aorta and the residual hematoma between the native Ao and graft material. B, C: Recorded more distally in the ascending aorta, at which point the graft material is visualized in its short and long axes within the dilated native aorta. 

Video 20-78a

Video 20-78b
FIGURE 20.79. The central figure is real-time transthoracic three-dimensional imaging of the aortic arch in a patient with significant atherosclerotic involvement. Note the complex atheroma depicted in the three-dimensional image (short arrows). The inset is the two-dimensional image recorded from the same patient at the same level depicting an area of focal atheroma. (○)
FIGURE 20.80. Transesophageal echocardiogram recorded in a longitudinal view in the proximal ascending aorta at the level of the pulmonary artery. Note the localized complex atheroma protruding into the lumen at this level (arrows). The inset is a real-time three-dimensional echocardiogram recorded at the same level again demonstrating the localized atheroma.

Video 20-80
FIGURE 20.81. Transesophageal echocardiogram recorded in the short-axis view of the arch of the aorta in a patient with moderate atheromatous involvement. The central figure is the two-dimensional image in which moderately complex atheroma is noted predominantly between 3 o’clock and 6 o’clock (arrows). The two insets are real-time three-dimensional images of the same area of the aorta more clearly demonstrating the diffuse nature of the complex atheroma with multiple protruding components and the suggestion of an ulcerated atheroma in the inset at the lower left. [Video 20-81]
Video 20-81A

coming soon

Video 20-81B

coming soon
FIGURE 20.82. Transesophageal echocardiogram recorded in a patient with
marked atheromatous involvement of the descending thoracic aorta. In A, note the irregular thickening of the atheromatous involvement (inward-pointing arrows) and the pedunculated component protruding into the lumen (downward-pointing arrow). The inset is a real-time, three-dimensional image recorded at this level of the aorta, depicting the marked irregularity of the aorta intima related to severe atheroma. B was recorded at a slightly different level and depicts an ulceration into the atheroma (downward-pointing arrow). The small image is a real-time three-dimensional image giving an en face view of the ulcer crater.
FIGURE 20.83. Transesophageal echocardiogram recorded in the longitudinal plane of a descending thoracic aorta with aneurysm. The arrows outline the external boundary of the aorta with all space in between representing an aneurysm with complex atheroma. Note the markedly complex atheroma with multiple pedunculated and mobile components filling the dilated lumen. [Video 20-83]
FIGURE 20.84. Transesophageal echocardiogram recorded in the mid-descending thoracic aorta in a patient with complex atheroma. Note the extensive atheroma burden with irregular margins. At approximately the 12 o’clock position note the smaller component of atheroma which can be seen to be highly mobile in the real-time image (arrow). The series of inward-pointing arrows outline the outer boundary of the aneurysmally dilated aorta. 

Video 20-84

coming soon
FIGURE 20.85. Longitudinal view of the aortic arch recorded in a patient with atherosclerotic disease and a fracture of an atherosclerotic plaque. Note the convoluted echo which folds on itself (arrow) and the mobility of the fractured component of the atheroma in the real-time images. [Video 20-85]
FIGURE 20.86. Real-time transesophageal three-dimensional echocardiographic imaging in the short axis of aorta revealing mobile complex atheroma. The inset figure is a three-dimensionally formatted computed tomography of the aorta in the same patient revealing severe diffuse atherosclerotic disease.

coming soon

Video 20-86
FIGURE 20.87. Real-time three-dimensional echocardiogram in a longitudinal view of the descending thoracic aorta revealing highly complex atheroma within the aorta. Video 20-87
FIGURE 20.88. Transthoracic echocardiogram recorded from the suprasternal notch (SSN) of the arch and proximal descending aorta (DA) in a patient with aortic coarctation. **A:** Note the ridge of tissue (arrow) in A representing the actual coarctation in a location immediately distal to the left subclavian artery. **B:** The color flow imaging further demonstrates the constraint of flow at that level and continuous-wave Doppler demonstrates a mild gradient of only 16 mm Hg across the coarctation. [Video 20-88a](#) [Video 20-88b](#)
FIGURE 20.89. Three-dimensionally reformatted CT angiogram of the aorta in an adult patient with previously unrecognized severe aortic coarctation. The long arrow denotes the level of the coarctation which has resulted in near-complete occlusion of the proximal descending aorta. The plane of the AV is also noted. A robust collateral network has developed including intercostal arteries (small arrows) and the internal mammary artery (IMA).
Aortic Pseudoaneurysm

Aortic pseudoaneurysm represents a contained rupture of the aorta and, as with left ventricular pseudoaneurysm, is characterized by an extraluminal aneurysmal sack communicating with the true lumen by a relatively narrow neck. Aortic pseudoaneurysms occur in several situations, including spontaneous rupture of an aortic aneurysm with subsequent sealing off of the hemorrhage or as sequelae of aortic dissection (Figs. 20.90 to 20.94). Pseudoaneurysm may also result from trauma or be the result of iatrogenic injury (Fig. 20.91). Because they are outside the contour of the normal aorta, visualization may be difficult, and CT is often necessary to make a definitive diagnosis (Figs. 20.92 and 20.93).
Aortic Trauma

Aortic transection is a catastrophic sequela of blunt chest injury, typically after a high-speed impact injury such as that experienced by an unrestrained
passenger involved in a motor vehicle accident. The characteristic injury is partial or complete transection of the descending thoracic aorta at the area of the *ligamentum arteriosum*. Complete aortic transection is a nearly instantaneously fatal event for which there is seldom time for diagnostic imaging. Partial transection may allow survival and arrival to an emergency department for evaluation. In most trauma centers, chest CT is the primary diagnostic modality.

**FIGURE 20.91.** Transesophageal echocardiogram of the proximal ascending aorta recorded in a patient with a large pseudoaneurysm following prior aortic surgery. In the left panel, note the dilated ascending aorta and the discontinuity of the aortic wall (*arrow*). In the right panel, recorded with color flow Doppler imaging, note the definite flow into the area of discontinuity suggestive of aortic rupture and/or pseudoaneurysm. Note the apparent limited extent compared to the true extent documented on CT (*Figure 20.92*) and size of the pseudoaneurysm depicted on echocardiography. 📄
coming soon

Video 20-91
Transesophageal echocardiography can be used for the detection of aortic trauma. It should be emphasized that there are several manifestations of aortic trauma, many of which may be subtle. Because most patients with complete
or nearly complete aortic transection do not survive, it is uncommon to
document this fatal complication with echocardiography. For aortic trauma in
which there has been partial disruption through the media into the adventitia,
periadventitial hematoma is often present. Adventitial hematoma may distort
the shape of the aorta so that it is no longer imaged as a circular structure and
may also deviate either the aorta or esophagus out of position so that when
withdrawing the probe to scan from the gastroesophageal junction superiorly,
the aorta moves out of the imaging plane. When examining the lumen of the
aorta itself, varying degrees of dissection and intimal tear may be seen, which
may be subtle and represent a limited intimal tear without actual dissection.
On occasion, a focal area of the aorta is encountered where circular geometry
is transiently lost and a limited ridge may be seen protruding into the aortic
lumen. This is indirect evidence of partial-thickness trauma at that site. On
occasion, limited trauma results in formation of a thrombus within the medial
space or in the lumen of the aorta itself, and if an apparent thrombus is
detected in a relatively young patient after blunt chest trauma, aortic trauma
rather than atheroma should be considered the most likely diagnosis.

Occasionally patients may experience a partial-thickness tear of the aortic
wall that is not immediately fatal. This complication can then lead to the
formation of an acute aortic pseudoaneurysm which can be detected with a
number of imaging techniques, including transesophageal echocardiography
(Figs. 20.94 and 20.95). On occasion aortic trauma, typically from a high-
speed motor vehicle or sporting accident results in disruption of normal aortic
valve coaptation and acute aortic insufficiency which may range from mild to
severe (Fig. 20.96). In these instances it is critical to pay close attention to
subtle evidence of traumatic IMH or localized dissection. Additional imaging
with CT and/or MRI is warranted in most cases.
FIGURE 20.93. Transesophageal echocardiogram recorded at the aortic arch in a patient with a pseudoaneurysm (PA). The upper panel shows distorted anatomy.
of the arch of the Ao with the relatively narrow neck on the right of the image. The lower panel is recorded with color flow Doppler revealing fairly brisk flow into and out of the PA. The inset is a three-dimensionally formatted CT from the same patient more clearly demonstrating the discrete aneurysm.

Intravascular ultrasound has also been used to document the presence of aortic trauma after blunt chest injury (Fig. 20.97). Because of the high resolution of this technique, more limited areas of intimal tear or disruption of aortic wall integrity can be detected which may not be seen with either transesophageal echocardiography or CT. A limitation of this technique is its relatively shallow depth penetration which precludes defining the presence and extent of an adventitial hematoma to the same degree that can be done
with transesophageal echocardiography.

Rarely interventional or other invasive procedures may result in trauma to the aorta. Complications include aortic perforation, iatrogenic dissection, or creation of an IMH between the wall of the aorta and left atrium. Figure 20.98 was recorded in a patient noted to have aortic insufficiency following an interventional procedure and demonstrates iatrogenic injury to the aorta with disruption of the normal proximal aortic geometry and functional aortic insufficiency both of which resolved with conservative management.

**Infections of the Aorta**

Bacterial or fungal infections of the aorta are an uncommon subset of infectious endocarditis (Fig. 20.99). They typically will arise at an area of atherosclerotic involvement or at the area of the ligamentum arteriosum on the aortic side of a persistent ductus arteriosus. They will manifest as a pedunculated mobile mass, for which the differential obviously includes complex mobile atherosclerotic disease. The infectious nature of the mass may be suggested by the overall clinical situation but obviously only proven by direct inspection. Rarely encountered in contemporary practice is syphilitic aortitis which results in inflammatory thickening of the proximal aorta. Positron emission tomography can be used to document aortic (or aortic graft) infection or inflammation (Fig. 20.100).
FIGURE 20.94. Transesophageal echocardiogram recorded in a long-axis view of the aorta just distal to the ligamentum arteriosum. This illustration was recorded in a patient following a high-speed motor vehicle accident with rupture of the descending thoracic aorta and acute pseudoaneurysm formation. In A, note the break in the continuity of the aortic wall noted by the two small arrows and in B the color flow Doppler demonstrating communication between the aorta and pseudoaneurysm cavity.
FIGURE 20.95. Transesophageal echocardiogram recorded in a patient after a high-speed vehicular accident. The patient presented with thoracic and other trauma with hypotension and shock. The recorded images are at approximately 30 cm from the incisors at the area of the ligamentum arteriosum. Color Doppler confirmed flow from the aortic lumen into the periaortic space. The inset at the lower left is a contrast-enhanced CT at the equivalent level of the aorta, also showing disruption of the aortic contour (arrow) and the extensive mediastinal hematoma (H). The inset at the upper left is a longitudinal CT view of the aorta, demonstrating the break in continuity (arrows) and extensive hematoma tracking superiorly.
FIGURE 20.96. Transesophageal echocardiogram recorded in a young patient with a new murmur noted following a high-speed motor vehicle accident. In the central illustration note the relatively normal dimensions of the proximal aorta and the absence of echoes to suggest either rupture or aortic dissection. Note the abnormal coaptation of the aortic cusps (arrow) which is seen in the expanded view at the lower right inset. At the upper right inset note the color Doppler
revealing moderate severity, highly eccentric aortic insufficiency (arrows).
Aortic Thrombus

In rare instances, a bland mobile thrombus can form within the aortic lumen. This is more common in the proximal descending thoracic aorta and often has been associated with peripheral embolization. Such thrombi are noted as highly mobile echodense masses within the lumen which frequently appear to be attached to the aortic wall by a fairly thin stalk. Figures 20.101 and 20.102 were recorded in a patient with peripheral embolization who underwent transesophageal echocardiography in a search for the source of an embolus. Note the highly mobile tubular echo within the aorta consistent with a thrombus and its origin on an atherosclerotic plaque. Appropriate therapy for intra-aortic thrombus is controversial, and the relative roles of aggressive anticoagulation versus surgical removal have not been fully elucidated. When detected, the patient will require evaluation for a hypercoagulable state or
underlying malignancy.

FIGURE 20.98. Transesophageal echocardiogram in the longitudinal view of the ascending aorta in a patient who developed aortic insufficiency following an interventional procedure. In the central panel note the almost 1-cm thick echo density between the aortic lumen and left atrial cavity representing traumatic hematoma (arrows). This has resulted in distortion of the geometry of the proximal aorta and subsequently in eccentric functional aortic insufficiency as noted in the lower right inset. The upper left inset is a similar view recorded 6 weeks following the initial presentation demonstrating resolution of the hematoma.
FIGURE 20.99. Longitudinal-axis transesophageal echocardiogram recorded in an immunocompromised patient with a fungal infection. In this instance, there has
been involvement of the lung by aspergillosis which has subsequently invaded both the pulmonary artery (PA) and the Ao. Note the irregular intraluminal echoes in both the pseudoaneurysm and the aorta which represent direct extension of the infection into the vascular structures. The long leftward-pointing arrow denotes the area of pulmonary consolidation due to this infection.

FIGURE 20.100. Positron emission tomography (PET) recorded in a patient with inflammatory aortitis. In this view, the left ventricle and ascending aorta are visualized. Notice the increased metabolic activity in the ascending aorta (arrows) represented by the pink coloration, which denotes enhanced metabolic activity.
FIGURE 20.101. Transesophageal echocardiogram recorded in a patient with lower extremity embolic events. This image is a longitudinal view of the thoracic aorta just distal to the arch. The margins of the aorta itself are as noted by the longer arrows. Within the lumen note the elongated tubular thrombus (Th) which has several mobile irregular protrusions (small arrows). The inset at the lower right is a short-axis view of the aorta through the mid portion of the thrombus. The tubular nature of the thrombus (arrows) is easily appreciated. In the real-time image note the significant mobility.  

Video 20-101Lx
FIGURE 20.102. The central image is a real-time three-dimensional image of the aortic thrombus depicted in Figure 20.101. The bulk of the more distal thrombus is noted by the two longer inward-pointing arrows. Note its origin from the wall of the aorta in an area of apparent atherosclerotic plaque (two small arrows). The inset at the lower left is a contrast-enhanced CT angiogram of the aorta from the same patient revealing a large tubular thrombus burden within the distal arch and proximal descending thoracic aorta (arrow).
FIGURE 20.103. Parasternal long-axis transthoracic echocardiogram recorded in
a patient with Takayasu arteritis. Note the abnormally bright echo within the anterior and posterior wall of the aorta in a young female patient in whom atherosclerotic disease would not be expected.

Video 20-103

Takayasu Arteritis

Takayasu arteritis is an inflammatory disease of the aorta and its proximal branches. By definition, it occurs in patients younger than 40 years. It results in marked, irregular intimal thickening and accumulation of inflammatory tissue in the proximal aorta and ostia of major branches including the coronary arteries. Echocardiographically, its appearance is similar to that of atherosclerotic disease (Fig. 20.103). On very rare occasions, other forms of arteritis, such as giant cell arteritis, can involve the aorta.

Suggested Readings

GENERAL


Goldstein SA, Evangelista A, Abbara S, et al. Multimodality imaging of diseases of the thoracic aorta in adults from the American Society of Echocardiography and the European Association of


**Aortic Dissection**


**Atheroma and Aneurysm**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
Chapter 21
Masses, Tumors, and Source of Embolus

Cardiac masses represent a diverse and challenging group of pathologies that include thrombi, benign and malignant neoplasms, congenital anomalies, and vegetations. In addition, normal variants, such as eustachian valves, may mimic these abnormalities and must be distinguished from pathologic masses. Identifying and fully characterizing these conditions may require more than one type of imaging modality. In a minority of cases, even multimodality imaging will fail to provide a definite diagnosis, and either biopsy or surgery may be necessary.

NORMAL VARIANTS AND ARTIFACTS: SOURCES OF FALSE-POSITIVE FINDINGS

The echocardiographic evaluation of cardiac masses is critically dependent on the ability to distinguish normal from abnormal findings. Ultrasound artifacts are common, even in high-quality studies, and may be mistaken for pathologic conditions. Near-field clutter and reverberations are examples of artifacts often confused with pathology (e.g., apical thrombi) on two-dimensional echocardiography (Fig. 21.1). Such artifacts, which are also covered in Chapter 2, must be avoided whenever possible and correctly identified when present. Proper transducer selection and the use of multiple acoustic windows are among the strategies that can be used to avoid potential misinterpretations.

Anatomic variants are ubiquitous, may involve any chamber or valve structure, and are potentially confused with pathologic structures. A list of
commonly encountered normal structures that are often misinterpreted as pathologic is provided in Table 21.1. The right atrium is the chamber that is most often a source of anatomic variants leading to inaccurate interpretation. The Chiari network, eustachian valve, and crista terminalis are examples of structures normally found in the right atrium that, due to individual variation, are frequently confused with pathologic entities. Fatty infiltration in the atrioventricular groove, especially around the tricuspid valve, is a common source of confusion. A benign condition, this fatty deposit is frequently mistaken for tumor or fluid. False tendons in the left ventricular apex are common and occasionally misinterpreted as thrombi (Fig. 21.2). In this example, the diagnosis of a false tendon is relatively straightforward. In some cases, the tendon can be mistaken for the surface of an apical thrombus. Color flow imaging or contrast echocardiography, by demonstrating flow on either side of the linear structure, can be helpful to make this distinction. Additional sources of confusion can be iatrogenic. For example, the suture line in the posterior atrial wall after cardiac transplantation and indwelling pacemaker leads or catheters are examples of “normal” structures that may be misinterpreted as pathologic. Figure 21.3 is an example of a right ventricular moderator band, another normal cardiac structure that can be confused with abnormal masses, such as thrombi.

**FIGURE 21.1.** An apical four-chamber view demonstrates a reverberation (arrows) due to a strong reflector in the near field, either on the transducer or surface or within the chest wall. In real time, it did not move with the motion of the heart. In **A,** it is seen in the left ventricle and in **B,** in the left atrium.
### Table 21.1  
NORMAL VARIANTS AND BENIGN CONDITIONS OFTEN MISINTERPRETED AS PATHOLOGIC

<table>
<thead>
<tr>
<th>Condition</th>
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<tbody>
<tr>
<td>Right atrium</td>
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<tr>
<td>Chiari network</td>
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<tr>
<td>Eustachian valve</td>
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<tr>
<td>Crista terminalis</td>
</tr>
<tr>
<td>Catheters/pacemaker leads</td>
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<tr>
<td>Lipomatous hypertrophy of interatrial septum</td>
</tr>
<tr>
<td>Pectinate muscles</td>
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<tr>
<td>Fatty material (surrounding the tricuspid annulus)</td>
</tr>
<tr>
<td>Location</td>
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<tr>
<td>-----------------</td>
</tr>
<tr>
<td><strong>Left atrium</strong></td>
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<tr>
<td><strong>Right ventricle</strong></td>
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<td></td>
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<td></td>
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<tr>
<td></td>
</tr>
<tr>
<td><strong>Left ventricle</strong></td>
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<tr>
<td></td>
</tr>
<tr>
<td><strong>Aorta</strong></td>
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</tbody>
</table>

LAA, left atrial appendage; LUPV, left upper pulmonary vein.
FIGURE 21.2. A false tendon (arrows) is seen in the body of the left ventricle.
FIGURE 21.3. A moderator band (arrow) in the apex of the right ventricle is often mistaken for a pathologic structure such as a thrombus.

Recognition of such normal variants depends on image quality and technique as well as experience. The use of multiple imaging windows and transducers of different frequency are additional strategies to ensure an accurate diagnosis. The availability of clinical information (such as whether the patient has a pacemaker) can be extremely valuable in avoiding errors.

️ ROLE OF ECHOCARDIOGRAPHY
Echocardiography remains the primary imaging modality for the initial assessment of suspected cardiac masses. Table 21.2 lists the Appropriate Use Criteria for the evaluation of patients with known or suspected cardiac masses or source of embolus. More recently, updated guidelines for the use of echocardiography in evaluating a cardiac source of embolus have been published. This document underscores the versatility of echocardiography and provides specific guidelines for the role of imaging in each category of embolic sources. Assessing cardiac anatomy and identifying abnormal structures are tasks well suited to echocardiography. For many patients, the ability to confidently exclude an intracardiac mass or potential source of embolus is often echocardiography’s most important contribution. When an anatomic abnormality is present, echocardiography is often able to detect it with high sensitivity; characterize its extent, location, and size; and distinguish it from artifact or normal variants. Through a careful anatomic assessment, echocardiography frequently provides important diagnostic information regarding the etiology of the mass and helps guide subsequent therapy.

A limitation of echocardiography, however, is its inability to provide tissue or histologic diagnosis. Distinguishing a benign tumor from a malignancy, or a thrombus from a vegetation, is often impossible on the basis of ultrasound alone. For this reason, other modalities, especially cardiac MRI, have emerged to play an important role in the comprehensive assessment of cardiac masses. In particular, MRI has the ability to determine if a structure contains specific types of tissue, such as lipomatous material, vascularity, or scar. Figure 21.4 illustrates a patient with a pancreatic neuroendocrine tumor and masses within the right atrium, adjacent to the tricuspid valve. MRI was able to characterize the masses as being isointense and avascular, most consistent with thrombus.

<table>
<thead>
<tr>
<th>Table 21.2</th>
<th>APPROPRIATE USE CRITERIA FOR CARDIAC MASSES AND SOURCE OF EMBOLUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indication</td>
<td>Suspected Cardiac Etiology—TTE</td>
</tr>
<tr>
<td>------------</td>
<td>---------------------------------------------------------------</td>
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<tr>
<td></td>
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<tr>
<td>1</td>
<td>Symptoms or conditions potentially related to suspected cardiac etiology, including TIA, stroke, or peripheral embolic event</td>
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<tr>
<td><strong>Pulmonary embolism with TTE</strong></td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>Suspected pulmonary embolism in order to establish a diagnosis</td>
</tr>
<tr>
<td>29</td>
<td>Known acute pulmonary embolism to guide therapy</td>
</tr>
<tr>
<td>31</td>
<td>Reevaluation of known pulmonary embolism after therapy, for assessment of RV function and/or pulmonary artery pressure</td>
</tr>
<tr>
<td><strong>TTE for intracardiac and extracardiac structures</strong></td>
<td></td>
</tr>
<tr>
<td>57</td>
<td>Suspected cardiac mass</td>
</tr>
<tr>
<td>58</td>
<td>Suspected cardiovascular source of embolus</td>
</tr>
<tr>
<td><strong>TEE as initial or supplemental test—embolic event</strong></td>
<td></td>
</tr>
<tr>
<td>109</td>
<td>Evaluation of cardiovascular source of embolus with no identified noncardiac source</td>
</tr>
<tr>
<td>110</td>
<td>Evaluation of cardiovascular source of embolus with a previously identified noncardiac source</td>
</tr>
<tr>
<td>111</td>
<td>Evaluation of cardiovascular source of embolus with a known cardiovascular source in which TEE would not change management</td>
</tr>
<tr>
<td>112</td>
<td>Evaluation to facilitate clinical decision making with regard to anticoagulation, cardioversion, and/or radiofrequency ablation</td>
</tr>
</tbody>
</table>


**CARDIAC TUMORS**

Cardiac tumors are uncommon and primary tumors of the heart are especially rare (see Table 21.3). Metastatic disease to the heart is 20 to 30 times more common than primary cardiac tumors. Among primary tumors, approximately 75% are benign, and most of these are myxomas or fibroelastomas. Among malignant primary tumors, sarcomas account for up to 90%. Lymphomas are less common, often occur in the setting of HIV, and can be either primary or metastatic. Among metastatic tumors, lung, breast, and GI malignancies most frequently affect the heart. However, melanomas are less common than these tumors but more likely to metastasize to the heart.

**Primary Benign Tumors**
Echocardiography is useful to identify conditions in which masses may develop, is an accurate technique to detect and characterize masses once they occur, and provides a noninvasive means for surveillance after treatment or removal. As stated above, primary tumors can be either benign or malignant and occur in all age groups. The most common primary cardiac tumors are listed in Table 21.4. Of these, benign tumors outnumber malignant ones by a ratio of approximately 3 to 1.

**FIGURE 21.4.** Recorded from a patient with a neuroendocrine tumor, the echocardiogram (A) shows two oval-shaped masses in the right atrium (arrows). With cardiac MRI (B), these structures (arrowheads) were found to be avascular and isointense, suggestive of thrombus.

<table>
<thead>
<tr>
<th>Primary</th>
<th>Malignant</th>
<th>Metastatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign</td>
<td>Malignant</td>
<td>Metastatic</td>
</tr>
<tr>
<td>Myxoma</td>
<td>Sarcoma</td>
<td>Lung</td>
</tr>
<tr>
<td>Lipoma</td>
<td>Angiosarcoma</td>
<td>Breast</td>
</tr>
<tr>
<td>Pericardial cyst</td>
<td>Rhabdomyosarcoma</td>
<td>Renal</td>
</tr>
<tr>
<td>Papillary fibroelastoma</td>
<td>Fibrosarcoma</td>
<td>Adenocarcinoma</td>
</tr>
<tr>
<td>Rhabdomyoma</td>
<td>Leiomyosarcoma</td>
<td>Gastrointestinal</td>
</tr>
<tr>
<td>Teratoma(^a)</td>
<td>Lymphoma</td>
<td>Melanoma</td>
</tr>
<tr>
<td>Fibroma(^a)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(^a\)Mostly occur in children.
A common benign primary tumor of the heart is the myxoma, accounting for 30% to 40% of all primary cardiac tumors. Myxomas are usually single and occur in the left atrium in 75% of cases where they most often arise from the area of the fossa ovalis. Their size, shape, and texture can be quite varied. Myxomas may be smooth surfaced but are more often irregularly shaped with filamentous fronds or have the appearance of a “cluster of grapes.” They are typically nonhomogeneous in texture with lucent centers or areas of calcification. Myxomas can be quite large, occupying most of the left atrium and resulting in obstruction to left ventricular filling. They may be attached to the endocardial surface by a stalk. A large atrial myxoma is shown in Figure 21.5. In this patient, the tumor nearly occludes the mitral orifice during diastole. The most important clue to the diagnosis is their location in the left atrium and origin from the midportion of the atrial septum. Given a typical presentation, echocardiography is virtually diagnostic of myxoma. In some cases, however, a definitive diagnosis may be more difficult. Figure 21.6 is from a patient scheduled to undergo coronary bypass surgery. The mass detected on echocardiography is in an unusual location for a myxoma. However, the absence of mitral valve disease or atrial fibrillation makes thrombus unlikely. The mass was surgically resected at the time of his coronary surgery and pathologic evaluation confirmed it to be a myxoma.

<table>
<thead>
<tr>
<th>Type</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign</td>
<td></td>
</tr>
<tr>
<td>Myxoma</td>
<td>30</td>
</tr>
<tr>
<td>Lipoma</td>
<td>10</td>
</tr>
<tr>
<td>Papillary fibroelastoma</td>
<td>8</td>
</tr>
<tr>
<td>Rhabdomyoma</td>
<td>6</td>
</tr>
<tr>
<td>Fibroma</td>
<td>3</td>
</tr>
<tr>
<td>Hemangioma</td>
<td>2</td>
</tr>
<tr>
<td>Teratoma</td>
<td>1</td>
</tr>
<tr>
<td>Malignant</td>
<td></td>
</tr>
<tr>
<td>Angiosarcoma</td>
<td>8</td>
</tr>
<tr>
<td>Rhabdomyosarcoma</td>
<td>5</td>
</tr>
<tr>
<td>Mesothelioma</td>
<td>3</td>
</tr>
<tr>
<td>Lymphoma</td>
<td>2</td>
</tr>
<tr>
<td>Leiomyosarcoma</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 21.4 RELATIVE FREQUENCY OF PRIMARY CARDIAC TUMORS
FIGURE 21.5. A: A myxoma (arrows) is seen in the left atrium on transesophageal imaging. The mass is attached to the fossa ovalis. B: A four-chamber view demonstrates a large myxoma (arrows) within the left atrium partially obstructs the mitral orifice during diastole.

Video 21-5

Transthoracic imaging is usually sufficient, although small tumors or those that involve the right side of the heart may require transesophageal echocardiography for diagnosis. Three-dimensional echocardiography has also been used to more fully characterize atrial myxomas (Fig. 21.7). Myxomas sometimes involve the right atrium (15%) or the left or right ventricle (5% each) (Figs. 21.8 and 21.9). In the example shown in Figure 21.8, note the mobility of this right atrial myxoma and how it extends through the tricuspid valve in diastole resulting in obstruction to right ventricular inflow. Figure 21.9 is a myxoma originating from the roof of the right atrium. Figure 21.10 is a large right ventricular myxoma that partially obstructs tricuspid valve flow. In 5% of cases, myxomas are multiple. They are most often confused with thrombi, although their characteristic location and attachment site is generally helpful in the differential diagnosis. After surgical excision, myxomas can recur. Therefore, surveillance echocardiograms should be obtained annually for several years to guard against this possibility.
FIGURE 21.6. A left atrial myxoma is shown (arrows). The mass can be seen in the long-axis (A) and short-axis views (B). The location of the tumor, attached to anterior wall of the chamber, below the aortic valve, is unusual.  

Video 21-6a

Video 21-6b
FIGURE 21.7. A large left atrial myxoma is demonstrated using three-dimensional imaging. Two off-axis parasternal views (A, B) are provided. The advantages of this modality are best appreciated when viewed in a cine loop format. Video 21-7
FIGURE 21.8. A large mass is seen within the left atrium during systole (A). In diastole (B), note how the mobile mass protrudes through the tricuspid valve creating obstruction to right ventricular inflow. In C, the degree of obstruction is demonstrated with pulsed Doppler, mean gradient = 9 mm Hg. The location, motion, and attachment site are consistent with right atrial myxoma. (continued)
FIGURE 21.9. A large right atrial myxoma is seen on transesophageal echocardiography. In the four-chamber view (A), it can be seen in the superior portion of the chamber (arrow). In the short axis (B), it appears to fill the right atrium (arrow), but an attachment site is not visualized. Using MRI, the stalk attaching the tumor to the right atrial superior wall is indicated by the arrow (C).
Video 21-9a

Video 21-9b
FIGURE 21.10. Another example of a large right ventricular myxoma is recorded using echocardiography (A) and cardiac CT (arrow) (B). The mass can be seen prolapsing through the tricuspid valve during systole causing partial obstruction to inflow. On CT, with right atrial contrast, the avascular tumor is clearly outlined. The surgical specimen is shown in C.  

Video 21-10a

Papillary fibroelastomas represent the other frequently encountered primary tumor of the heart. Historically, based largely on autopsy series,
Myxomas were considered 3 to 4 times more common than fibroelastomas. More recent data using echocardiography (Tamin et al., 2015) suggest that papillary fibroelastomas may be as prevalent, or even more common, than myxomas. These are usually found in older patients and usually arise from either the aortic or mitral valve (Fig. 21.11), although they occasionally arise from nonvalvular endothelium. Because tumors arising from the heart valves are rare and often asymptomatic, establishing a diagnosis can be challenging and often relies on echocardiography. Among tumors that affect the valves, papillary fibroelastomas are by far the most common, accounting for more than 85% of valve-associated tumors. Myxomas and fibromas account for the remainder, whereas malignant tumors involving the valves are very rare.

**FIGURE 21.11.** A transesophageal echocardiogram of the four-chamber (A) and long-axis (B) views show a papillary fibroelastoma (arrows) of the mitral valve. The tumor was attached by a small pedicle to the anterior leaflet and was highly mobile.
FIGURE 21.12. A small papillary fibroelastoma is seen in a patient who had a stroke. The mass (arrow) is seen on the posterior leaflet in diastole (A) and systole (B).

Video 21-12

FIGURE 21.13. A large papillary fibroelastoma is depicted attached to the mitral valve (arrow). This is demonstrated from the long-axis (A) and four-chamber (B) views.
FIGURE 21.14. A papillary fibroelastoma was the cause of this patient’s anterior MI. Using transesophageal echocardiography, the small, highly mobile mass (arrows) can be seen attached to the downstream side of the aortic valve in the long-axis (A) and short-axis (B) views. On coronary angiography (C), several branches of the left anterior descending and left circumflex arteries are abruptly occluded (small arrows). [Video 21-14b]
FIGURE 21.15. Although most papillary fibroelastomas attach to a valve, this example shows a large tumor in the left ventricular outflow tract (arrow), attached to the septal endocardium.
Papillary fibroelastomas are small, generally 0.5 to 2.0 cm in diameter, and are often confused with vegetations. Making this distinction is difficult because of the similarity in the echocardiographic appearance. A correct diagnosis therefore depends on the clinical setting, that is, the presence or absence of signs of infection. These tumors usually attach to the downstream side of the valve by a small pedicle and are irregularly shaped with delicate frond-like surfaces (Figs. 21.12 and 21.13). Significant valvular regurgitation is rare. Mobility is common and generally considered a risk factor for embolization. Figure 21.14 is an example of an aortic valve fibroelastoma that embolized to the left coronary artery of a young woman, resulting in a large anterior myocardial infarction. Although most papillary fibroelastomas are attached to valves, as many as 20% occur in nonvalvular locations. Figure 21.15 is an example of a tumor attached to the left ventricular outflow tract endothelium, detected during an evaluation of aortic stenosis. The diagnosis of papillary fibroelastoma was confirmed when the tumor was excised at the time of valve surgery.

There is some confusion as to whether fibroelastomas are distinct from Lambl excrescences, which are smaller and frequently seen on otherwise normal valves in elderly patients (Fig. 21.16). Whether the two represent the distinct entities remains controversial. Fibroelastomas are also confused with blood cysts, which are unusual blood-containing cystic structures that develop within mitral leaflets (Fig. 21.17). Blood cysts have a broader base, are sessile, and are less mobile than fibroelastomas.
Lipomas are uncommon benign tumors involving the heart. Lipomatous hypertrophy of the atrial septum is one presentation. In this condition, the
atrial septum is infiltrated by lipomatous material that results in dramatic thickening and increased echogenicity of its inferior and superior portions with sparing of the fossa ovalis (Fig. 21.18). The fatty infiltrate is highly echogenic and results in a “dumbbell-shaped” appearance on two-dimensional echocardiography. The condition is thought to be benign and rarely associated with clinical manifestations. Occasionally, the lipomatous material may infiltrate the right and left atrial walls and become so massive that it is confused with thrombus or tumor. Figure 21.19 was recorded from a patient with palpitations. Extensive lipomatous hypertrophy was demonstrated on transesophageal echocardiography. In addition, there appears to be extension of the mass into the right atrium. Further evaluation using MRI confirmed that the entire mass was lipomatous.

Rhabdomyomas are among the most common benign pediatric tumors (Fig. 21.20). They occur either within a cavity, sometimes as a pedunculated mass, or embedded within the myocardium. Such tumors can grow quite large and can obstruct blood flow within the heart. Fibromas are uncommon benign tumors, most often seen in children, and usually involve the left ventricular free wall (Fig. 21.21). On echocardiography, they appear as distinct, highly echogenic, and well-demarcated masses that often extend into the cavity of the ventricle. Although benign, they occasionally result in obstruction to left ventricular filling and have been associated with ventricular arrhythmias. A rare condition that can be confused with a fibroma (or a thrombus) is endocardial fibroelastosis. This disease is usually seen in young children and is characterized by fibrous thickening of the left ventricular endothelium, probably as a nonspecific response to inflammation or infection. An example of endocardial fibroelastosis is provided in Figure 21.22. Unlike fibromas, the mass is endocardial rather than intramyocardial.
FIGURE 21.17. A blood cyst (arrow) within the anterior mitral leaflet. The cyst is relatively immobile and the attachment is broad based. The mass is seen during diastole (A) and systole (B).

Video 21-17

FIGURE 21.18. Lipomatous hypertrophy of the atrial septum. A: A mild degree of
accumulation of lipomatous material is present (arrows). The fossa ovalis is characteristically spared. B: A more extreme form of lipomatous hypertrophy (arrows).  Video 21-18
FIGURE 21.19. An example of an extreme form of lipomatous hypertrophy is shown from a young woman with palpitations. Transesophageal echocardiography demonstrates extensive hypertrophy of the septum, with typical sparing of the fossa ovalis (A). In addition, mobile round structures were seen (arrows) attached to the lipomatous material (A and B). On MRI, the appearance confirmed that the atrial masses were globular lipomatous material extending from the interatrial septum (C).
FIGURE 21.20. Rhabdomyoma is a common pediatric tumor. In this 12-year-old patient, multiple tumors are seen within the left and right ventricle (asterisks) and interventricular septum (arrows).
Primary Malignant Tumors

Malignant primary tumors of the heart are quite rare and include the various types of sarcomas, often undifferentiated, and lymphomas (which can be either primary or metastatic). Such masses often remain asymptomatic and undetected until they become large enough to affect cardiac function or obstruct blood flow. Occasionally malignant tumors cause chest discomfort, arrhythmias, or pericardial tamponade. Figure 21.23 is an example of a fibrosarcoma that occupies the right ventricular outflow tract. Its size and location combine to produce a significant outflow tract gradient, as evidenced by the Doppler recording. Such tumors tend to invade or replace myocardial tissue and thereby dramatically alter the appearance and/or function of the heart. A pleomorphic sarcoma that originated from the left upper pulmonary vein and extended into, and filled, the left atrium is shown in Figure 21.24. As opposed to the well-circumscribed appearance of benign tumors, cardiac malignancies appear to infiltrate the tissues, disrupting normal anatomic planes, and invade or obliterate contiguous structures. The heart often appears tethered and relatively immobile, without its normal translational motion (Fig. 21.25). Although most primary cardiac malignancies involve the myocardium, they can occasionally involve valvular apparatus. Figure 21.26 is taken from a young man with a synovial sarcoma arising from a papillary muscle and extending into the left ventricular outflow tract. At the time of surgery, the tumor was found to be associated with the chordae. Although it was successfully excised, it recurred within a year.
FIGURE 21.21. A 1-day-old male with a large fibroma is imaged with transthoracic echocardiography. In the inverted four-chamber view (A), the extent of the mass is indicated by the arrows. In B, the long axis is medially angulated to demonstrate the extensive involvement of the right ventricular free wall (arrows) and the small ventricular cavity. [Video 21-21]
FIGURE 21.22. An example of endocardial fibroelastosis. Endocardial thickening in the left ventricular apex is present. Thrombus overlies the thickened endocardium (arrows).
FIGURE 21.23. A primary fibrosarcoma is demonstrated in the right side of the heart. **A:** The tumor involves the right ventricular outflow tract and pulmonary artery (arrows). **B:** Narrowing of the right ventricular outflow tract is indicated by the arrows. **C:** Doppler imaging demonstrates a right ventricular outflow tract gradient of approximately 50 mm Hg.
Contrast perfusion imaging may have a role in further characterizing intracardiac masses and distinguishing tumors from thrombi. Enhancement of the mass after contrast injection correlates with the degree of vascularity. Thus, malignant tumors and other vascular structures often demonstrate hyperenhancement while thrombi and other avascular masses, such as myxomas, show less contrast uptake. Alternatively, cardiac MRI may be helpful to depict the extent and infiltrative nature of the mass, as well as the tissue characterization, including the presence or absence of vascularity (see Fig. 21.27).

**FIGURE 21.24.** This pleomorphic sarcoma originated from a pulmonary vein and extended into the left atrium. It is seen protruding through the mitral valve during diastole in the long-axis (A) and four-chamber views (B). This results in a mild gradient across the mitral valve (C).
FIGURE 21.25. A, B: An example of angiosarcoma. The mass had infiltrated the lateral wall of the left atrium and left ventricle and invaded the mitral valve. Obstruction to mitral inflow was present. In real time, the heart appeared fixed due to infiltration by the malignancy. A small pericardial effusion is also present.

Video 21-25

The echocardiographic assessment of these patients has several components. Because primary cardiac malignancy is so much less common than metastatic involvement, the echocardiographic demonstration of an invasive cardiac tumor should suggest the possibility of metastatic disease. In addition, the exact location and extent of a cardiac malignancy must be thoroughly assessed to determine whether resection might be possible. Some malignancies are likely to affect a given chamber or location within the heart. Angiosarcomas, for example, usually involve the right atrium, whereas rhabdomyosarcomas may occur anywhere. Associated pericardial effusion is common, sometimes leading to tamponade.

Metastatic Tumors to the Heart

Echocardiography is often performed in patients with known or suspected malignancy. Among patients with cardiac symptoms, looking for evidence of metastatic spread has therapeutic and prognostic implications. Cardiac function helps determine whether a given patient may be a candidate for particular therapies, such as doxorubicin. In patients who have already received cancer therapy, echocardiography is useful to evaluate for side effects. Adriamycin, for example, can cause cardiomyopathy. Chest
irradiation can result in constrictive pericarditis or scarring and fibrosis of the epicardial coronary arteries. In unstable or critically ill patients, the portability and noninvasive nature of ultrasound represents a significant advantage.

**FIGURE 21.26.** A synovial sarcoma in a 19-year-old male who presented with syncope is evaluated with transesophageal echocardiography. The mass is attached to the mitral chordae and can be seen obstructing the left ventricular outflow tract in diastole (A) and then prolapsing through the aortic valve during systole (B).

Video 21-26
FIGURE 21.27. Subcostal four-chamber view from a patient with a sarcoma (arrows) involving the right atrium and tricuspid valve (A). A pericardial effusion is also present (asterisk). With cardiac MRI, the location and extent of the tumor is similar to what is seen with echocardiography (B). In C, the vascular nature of the tumor is demonstrated (arrows). [Video 21-27a]
The heart is affected relatively less often by metastatic disease compared with other organs. Some investigators speculate that blood-borne malignant cells are destroyed by the contraction of the heart before they become established. Malignant tumors can spread to the heart through direct invasion from adjacent tumors, including lung and esophagus, from propagation through the venous system, or by hematogenous spread (Table 21.5). Melanoma, for example, has a high propensity for metastasizing to the pericardium and/or myocardium, involving the heart in more than 50% of cases. When it does, it can take on a variety of appearances. Intracardiac masses are frequently seen as a manifestation of malignant melanoma. Figure 21.28 is an example of a melanoma that has metastasized to the left ventricular apex. The presence of a mass is suggested on the transthoracic study but is best visualized after injection of a contrast agent. Although the appearance of the mass is similar to that of a thrombus, preserved apical contractility makes a thrombus unlikely and should suggest the possibility of alternative diagnoses. Figure 21.29 is taken from another patient with melanoma, metastatic to the right ventricular apex. Figure 21.30 illustrates metastatic melanoma in a patient who presented with ventricular arrhythmias. The metastatic lesion was intramyocardial, confined to the ventricular septum. Another example of metastatic melanoma is provided in Figure 21.31. In this case, extensive involvement of the interatrial septum is demonstrated. Although masses attached to this area are typically myxomas, the invasive nature of the mass and the shaggy, ill-defined surface of the tumor strongly suggests malignancy. Some leukemias also have a similarly high rate of cardiac spread.

<table>
<thead>
<tr>
<th>Original Source</th>
<th>Cardiac Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung</td>
<td>Direct extension, often via pulmonary veins; effusion common</td>
</tr>
<tr>
<td>Breast</td>
<td>Hematogenous or lymphatic spread; effusion common</td>
</tr>
<tr>
<td>Lymphoma</td>
<td>Lymphatic spread, varied manifestations</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Variable manifestations</td>
</tr>
<tr>
<td>Cancer Type</td>
<td>Description</td>
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<tr>
<td>---------------------</td>
<td>-----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Melanoma</td>
<td>Intracardiac or myocardial involvement</td>
</tr>
<tr>
<td>Renal cell carcinoma</td>
<td>IVC to RA to RV; confused with thrombus</td>
</tr>
<tr>
<td>Carcinoid</td>
<td>Tricuspid and pulmonic valve thickening</td>
</tr>
</tbody>
</table>

IVC, inferior vena cava; RA, right atrium; RV, right ventricle.

**FIGURE 21.28.** Metastatic melanoma often involves the heart. **A:** Image quality prevents visualization of the apical mass. **B:** After contrast injection, the outline of the apical mass (arrows) is apparent.

**Video 21-28**

Figure 21.32 is from a patient with non-Hodgkin lymphoma, with extranodal involvement, including the heart. Following chemotherapy, resolution of cardiac involvement is apparent. However, more common
malignancies, such as breast or lung cancer, account for the greatest percentage of nonprimary cardiac tumors. A patient with metastatic squamous cell carcinoma is illustrated in Figure 21.33. The large mass invades the interventricular septum and extends into the left ventricular cavity. Figure 21.34 is from a patient with advanced lung cancer. In this case, direct extension of the tumor through the left upper pulmonary vein is demonstrated. The mass was so large that it protruded through the mitral valve during diastole. Another example of metastatic lung cancer is shown in Figure 21.35. Unlike the previous example, the tumor in this patient led to a large pericardial effusion and infiltration into both atria.
FIGURE 21.29. Metastatic melanoma involving the right ventricular apex (arrows).
The location of involvement of metastatic disease is frequently the pericardium, resulting in a pericardial effusion and epicardial involvement (Figs. 21.35 and 21.36). The usual signs and symptoms of pericarditis are often absent. In patients with known malignancies, the detection of a pericardial effusion should raise concern about cardiac metastases. However, it is almost impossible, based on echocardiographic findings alone, to establish the cause of a pericardial effusion. Patients with cancer may develop pericardial effusion for any of several reasons. For example, particular chemotherapies can cause pericardial effusion. In most cases, confirming that the effusion is malignant often has therapeutic implications. Pericardiocentesis, usually with biopsy, is generally appropriate but only diagnostic in approximately 50% of cases. When the pericardial involvement is due to metastatic disease, the prognosis is uniformly poor. Figure 21.37 is a case of metastatic disease involving the posterior left ventricular wall and pericardium. Over a period of several weeks, the tumor eroded through the myocardium, resulting in formation of a pseudoaneurysm that gradually increased in size until the time of the patient’s death. Intramyocardial involvement is less common than pericardial metastases and usually occurs secondary to lymphoma or melanoma. Heart failure, obstruction to flow, and arrhythmias may develop as a result. Cardiac involvement is often established at autopsy as an incidental finding in patients with widely metastatic disease. Figure 21.38 is taken from a patient undergoing treatment of a B-cell lymphoma. The tumor had spread to the heart and can be seen filling the right
atrium and extending into the left atrium. Figure 21.39 is an example of a pericardial mesothelioma. The mass is huge and grossly distorts the right side of the heart. Figure 21.40 shows a patient with lymphoma, before and after chemotherapy. The tumor involved the aortic root and posterior wall of the heart, including the area of the coronary sinus. After successful chemotherapy, normal anatomy is restored. In this case, serial echocardiography was critical to follow the progress of therapy and the reduction in tumor burden.

**FIGURE 21.30.** Metastatic melanoma is seen (arrow) as an oval-shaped hypoechoic mass within the interventricular septum.
FIGURE 21.31. A large mobile mass invading the interatrial septum and extending into the right atrium is shown. In the four-chamber view (A), the mass could be confused with a myxoma or a straddling thrombus (arrows). In the short-axis view (B), the bulky, irregular shape of the mass (arrows) and its distortion of the normal interatrial septal shape suggest malignancy. The mass is further characterized with MRI (C, arrow). At surgery, this was found to be metastatic melanoma.
FIGURE 21.32. A very bulky mass, later identified as lymphoma, is recorded from the four-chamber (A) view. The mass fills the right atrium and protrudes into the right ventricle and left atrium (arrow). Following chemotherapy (B), no residual
evidence of cardiac involvement is evident.  

Video 21-32a

Video 21-32b
FIGURE 21.33. From a patient with squamous cell lung cancer, a large metastatic mass is seen from the four-chamber (A) and long-axis (B) views. The tumor invaded the septum and protruded into the left ventricle (arrows).
FIGURE 21.34. From a patient with advanced lung cancer, transesophageal echocardiography reveals a mass arising from the left upper pulmonary vein and extending into the left atrium. During systole (A), the mass can be seen within the upper portion of the atrium. In diastole (B), it extends through the mitral valve.

Video 21-34

FIGURE 21.35. Metastatic lung cancer involving the right heart is shown from the four-chamber (A) and short-axis (B) views. The tumor (arrows) can be seen filling the right atrium and extending into the right ventricle. A pericardial effusion is also present.
FIGURE 21.36. A malignant pericardial effusion (asterisks) demonstrated in a patient with bronchogenic carcinoma. [coming soon]
Intravascular extension of tumor is a common manifestation of renal cell carcinoma (Figs. 21.41 and 21.42). Extension of the cancer into the inferior vena cava (IVC) can lead to right atrial involvement. Pulmonary embolization can occur and occasionally can be recorded with echocardiography. In some cases, the initial diagnosis of this tumor is made after detection of a right atrial mass on echocardiography. Distinguishing tumor from thrombi or other etiologies depends on demonstration of extension into the IVC, retrograde to the kidneys. Figure 21.43 is from a patient with uterine leiomyosarcoma that eventually spread to the right atrium via the IVC. In this case, cardiac MRI (Fig. 21.44) was helpful to fully characterize the extent of disease and to demonstrate that the IVC mass seen on echocardiography was tumor rather than thrombus.

Carcinoid tumors secrete a variety of vasoactive substances, such as serotonin, into the venous system that are usually inactivated by the liver and the lung. When metastatic disease allows these tumor products to reach the right side of the heart, they produce characteristic abnormalities that affect the tricuspid and pulmonary valves. The valve pathology involves fibrosis, smooth muscle proliferation, and endocardial thickening. On echocardiography, the valves appear thickened, retracted, and immobile. A typical but advanced case of carcinoid heart disease is provided in Figure 21.45. The right side of the heart is markedly dilated and the tricuspid valve is thickened and rigid. It appears nearly fixed in a position midway between open and closed. As a result, severe tricuspid regurgitation is present. In most patients with carcinoid heart disease, the tricuspid valve is the predominant site of involvement. Although some degree of stenosis may be present, the main hemodynamic abnormality is usually regurgitation and is often severe. In contrast, when the pulmonary valve is affected, stenosis tends to predominate. An example of this is shown in Figure 21.46. Involvement of the left-sided valves occurs in less than 10% of cases and suggests the possibility of a patent foramen ovale (PFO) with right-to-left shunting.
Baseline

Two months later

FIGURE 21.37. Progression of disease over time in a patient with metastatic melanoma. A to C: Long-axis views. D to F: Four-chamber views. On the initial echocardiogram, a large cystic mass (arrows) was present posterior and lateral to the left side of the heart. Two months later, the mass had increased in size and color Doppler imaging demonstrated flow communication between this structure and the left ventricle. This was due to free wall rupture and pseudoaneurysm formation. Note how the pseudoaneurysm compresses the left side of the heart.

Video 21-37
FIGURE 21.38. A large mass due to a B-cell lymphoma straddles the atrial septum and extends into the left atrium (A, arrow) near the anterior leaflet of the mitral valve. A short-axis view (B) shows the mass filling the right atrium and extending through the tricuspid valve (arrow).
INTRACARDIAC THROMBI

Left Ventricular Thrombi

Patients at risk of the development of a left ventricular mural thrombus are readily identified with echocardiography. Predisposing factors include recent myocardial infarction, left ventricular aneurysm, and dilated cardiomyopathy. Thrombi are most often located in the apex of the left ventricle, usually in the presence of akinesis or dyskinesis. Infarcts that do not result in an apical wall motion abnormality are less likely to be associated with thrombus formation. Although myocardial infarction is the most common predisposing cause of left ventricular thrombi, they can develop in any situation in which low flow and blood stasis occur, such as a chronic left ventricular aneurysm. In patients with dilated cardiomyopathy, low-velocity swirling of blood within the left ventricle also predisposes to the development of a thrombus. With color flow imaging from the apical four-chamber view, a slow, counterclockwise flow of blood during diastole may be present.

Left ventricular thrombi are best detected using transthoracic echocardiography, in part because transesophageal imaging is sometimes limited in its ability to completely record the apex. Using the transthoracic approach, apical views that position the left ventricular apex in the near field are optimal for this purpose. To enhance sensitivity, a high-frequency transducer with a short focal length is optimal. Thrombi are typically amorphous, echogenic structures with variable shape and are adherent to the endocardium (Fig. 21.47). Thrombi may be multiple and mobile and may protrude into the left ventricular cavity. In most cases, they have a texture and appearance that are distinct from the adjacent myocardium. An echolucent center may be present and suggests that the thrombus is relatively new and actively growing (Fig. 21.48). In some patients, differentiating between thrombus and myocardium may be difficult. In Figure 21.49, a large thrombus can be seen within an apical aneurysm. Despite its size, the thrombus is immobile and does not extend into the cavity of the left ventricle. Figure 21.50 demonstrates a smaller thrombus but one that exhibits mobility and protrusion.
FIGURE 21.39. Pericardial involvement of a mesothelioma. A: A large mass (arrows) completely obscures the right side of the heart and encroaches on the left atrium. B: Subcostal image demonstrates the extent of the malignancy (arrows) and the mass effect that it creates on the left side of the heart.

Video 21-39
FIGURE 21.40. A, B: A lymphoma invading the heart and great vessels. The tumor can be seen encasing the aortic root and the posterior atrioventricular groove (arrows). After successful chemotherapy, the echocardiogram appears essentially normal (C, D). Video 21-40
FIGURE 21.41. Renal cell carcinoma often affects the right side of the heart. On this transesophageal echocardiogram, tumors fill the right ventricle (left) and right atrium (right). This is the result of the extension of the malignancy from the kidneys through the inferior vena cava. Video 21-41
FIGURE 21.42. A patient with renal cell carcinoma is evaluated with transesophageal echocardiography. The tumor is shown within the IVC (arrows).

FIGURE 21.43. A large mass filling the right atrium (A, arrows) and inferior vena cava (B, asterisks) is shown from a patient with a uterine leiomyosarcoma.
The sensitivity of transthoracic echocardiography for detecting left ventricular thrombi is between 75% and 95%. Small, laminar thrombi that do not protrude into the cavity are most likely to be missed. Poor image quality greatly affects accuracy and may produce both false-negative and false-positive results. To avoid false-negative results, appropriate transducer selection is critical. A high-frequency (e.g., 5 MHz), short-focus transducer is optimal in most cases. In addition, the use of modified apical transducer positions allows a thorough interrogation and improves accuracy. Large, protruding thrombi are readily seen from the apical window (Figs. 21.49 and 21.50). Figure 21.51 illustrates a relatively large apical thrombus that was not apparent using “standard” apical views. Only when tangential or off-axis
views were obtained was the mass evident. Thrombi may involve more than one cardiac chamber. Figure 21.52 is taken from a patient with dilated cardiomyopathy and atrial fibrillation. Thrombi were detected in both left and right ventricular apices as well as the right atrium. Multiple thrombi in the left ventricle can also occur. Figure 21.53, from a patient with ischemic cardiomyopathy, illustrates thrombi in the left ventricular apex and inferobasal region.
FIGURE 21.44. Cardiac MRI from the same patient as shown in Figure 21.43. The arrows indicate the multilobed structure of the tumor within the right heart.

Both contrast and three-dimensional echocardiography have been used to improve the accuracy for the detection of apical thrombi. Contrast is particularly helpful in patients with poor image quality. Figure 21.54
demonstrates an apical thrombus that could not be visualized on routine transthoracic imaging. After administration of contrast, the apical mass is clearly recorded. The role of three-dimensional imaging is less well established for this purpose. Figure 21.55 is an example of multiple left ventricular thrombi visualized using transthoracic three-dimensional echocardiography.

False-positive results also occur, most often as a result of improper imaging technique leading to foreshortening of the true apex. In most cases, the diagnosis can be made on the basis of the presence or absence of an apical wall motion abnormality. Muscular trabeculations within the left ventricular apex can also lead to false-positive findings. Apical hypertrophy is occasionally misdiagnosed as a mural thrombus. Figure 21.22 is an example of endocardial fibroelastosis, which is a rare condition that can mimic an apical thrombus. Other left ventricular conditions that may be confused with thrombi include hypereosinophilic syndrome (Fig. 21.56). This produces dense endocardial fibrosis that has a characteristic echogenicity or brightness on the echocardiogram. In the example shown, note the bright appearance of both the apical mass and the underlying myocardium. This is likely due to fibrosis and infiltration within the tissue. Mural thrombi often form over the thickened endocardium; thus distinguishing a thrombus from fibrosis may be difficult.

Myocardial noncompaction is a rare congenital form of cardiomyopathy in which the apical portion of the left (and sometimes right) ventricle is involved (Fig. 21.57). Because of failure of normal “compaction” in utero, the involved myocardium is characterized by a spongy appearance with prominent trabeculations and deep intertrabecular recesses. In some cases, color flow imaging will demonstrate flow within these spongiform recesses, creating a “Swiss cheese-like” appearance.
FIGURE 21.45. An example of carcinoid heart disease. A: The right side of the heart is dilated and the tricuspid valve is thickened, fibrotic, and immobile. The tricuspid leaflets are fixed (B) and do not coapt in systole (C). D: Color Doppler imaging demonstrates severe tricuspid regurgitation.
FIGURE 21.46. Carcinoid can also affect the pulmonary valve. **A:** The valve appears thickened and restricted. **B:** The peak gradient across the pulmonary valve is 56 mm Hg. Color Doppler imaging demonstrates severe pulmonary regurgitation (C).
FIGURE 21.47. An example of a left ventricular mural thrombus (arrows) visualized in the long-axis (A) and short-axis (B) views.

FIGURE 21.48. An apical thrombus (arrows) with an echolucent center, from a patient with recent anterior infarction, is shown. The mass is seen from both long-axis (A) and four-chamber (B) views.
FIGURE 21.49. A large apical left ventricular thrombus (arrows) is seen filling an apical aneurysm. In real time, the thrombus demonstrated little mobility.
FIGURE 21.50. A left ventricular apical thrombus (arrows). From the apical two-chamber view (A), the thrombus protrudes into the cavity and demonstrates mobility on real-time imaging (B).
FIGURE 21.51. Standard apical four- (A) and two-chamber (B) views, respectively. From this window, the apex appears free of thrombi. C, D: Off-axis imaging demonstrates a large, circular mass (arrow) consistent with a thrombus.
FIGURE 21.52. From a patient with severe heart failure due to dilated cardiomyopathy, multiple thrombi are recorded. **A:** A left ventricular apical thrombus and a large right atrial thrombus are indicated by the arrows. **B:** A modified apical view demonstrates thrombi in both the left and right ventricles (arrows). 

**Video 21-51**
FIGURE 21.53. From a patient with ischemic cardiomyopathy, multiple left ventricular thrombi (arrows) are shown from the two-chamber (A and B) and subcostal four-chamber (C) views.
FIGURE 21.54. An apical thrombus from a patient with prior myocardial infarction is shown. In A, without contrast, the apex is not well visualized and a mass is not seen. With contrast (B), an apical thrombus is clearly defined (arrows).
Thrombi rarely form in the absence of apical dyskinesis, so masses seen in the setting of normal wall motion should suggest other possibilities. Figure 21.58 is an example of an apical mass in a patient with normal wall motion. This most likely represents a muscle bundle or trabeculation. Tumors or vegetations may also occur in this location, and the final diagnosis can rarely be made solely on the basis of the echocardiogram. Figure 21.59 is from a patient with end-stage renal disease who suffered an anterior myocardial infarction. An apical thrombus formed. Later, despite the improvement in apical wall that followed percutaneous coronary revascularization, the apical thrombus persisted. Transesophageal echocardiography offers few advantages over transthoracic imaging for assessing the apex and detecting left ventricular thrombi. However, the use of multiplane imaging from the gastric views does permit a thorough evaluation of the apex. This is particularly helpful in the presence of poor transthoracic image quality.

Echocardiography can also identify thrombi that are most likely associated with embolic risk (Fig. 21.50). Risk factors include large size, mobility, and protrusion into the left ventricular cavity. Other less well-established risk factors are hyperkinetic wall motion adjacent to the thrombus and an echolucent center (presumably identifying an actively growing thrombus). Assessment of these various characteristics may be helpful in guiding the use of anticoagulation in some patients. Echocardiography can also be used to follow known ventricular thrombi, particularly after myocardial infarction, to detect changes over time and ultimate resolution.
FIGURE 21.55. A, B: These are two cases of multiple left ventricular thrombi recorded using three-dimensional echocardiography. From the apical four-chamber view, multiple thrombi (arrows) are seen within the left ventricular cavity. In real time, both the mobility and the three-dimensional nature of the structures are apparent. (Images courtesy of R. Martin, MD, and M. Vannan, MD.)

Video 21-55
FIGURE 21.56. Endocardial thickening and fibrosis are characteristics of hypereosinophilic syndrome. The highly echogenic mass within the left ventricular apex is the result of this process.
FIGURE 21.57. An example of noncompaction of the left ventricular myocardium. Baseline (A) and contrast-enhanced (B) images are provided. The left ventricle apex has a thickened, spongiform appearance (arrows).
Video 21-57a

coming soon

Video 21-57b

coming soon
**FIGURE 21.58.** An echogenic, small apical mass (arrow) is recorded in a patient with normal left ventricular wall motion. The two-chamber view is shown in diastole (A) and systole (B). This likely represents a trabeculation or muscle bundle within the cavity.

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**Video 21-58**

**Left Atrial Thrombi**

Although thrombi may form anywhere within the left atrium, the appendage is by far the most likely site. Any condition leading to stasis of blood within the left atrium predisposes to thrombus formation. These include mitral stenosis, atrial fibrillation, and dilated and restrictive cardiomyopathy. On the other hand, significant mitral regurgitation, by increasing flow velocity within the left atrium during systole, may reduce the risk of thrombus formation. Figure 21.60 demonstrates a very large left atrial thrombus from a patient with rheumatic mitral valve disease and a huge left atrium. In this extreme case, the thrombus most likely originated in the atrial appendage but grew in size and eventually spread to the body of the left atrium. The left atrial appendage is difficult to image using the transthoracic approach. The basal short-axis view can be manipulated to visualize the left atrial appendage just below the pulmonary artery in some patients. In other cases, the apical two-chamber view will permit recording of the appendage (Fig. 21.61). Because this is feasible in only a small minority of patients, however, transthoracic imaging should not be relied on to exclude left atrial thrombi. Transesophageal imaging is necessary to visualize the entire left atrium, including the appendage, and thus to exclude the possibility of a thrombus...
The approach to interrogation of the left atrium using transesophageal echocardiography is discussed in detail in Chapters 4 and 7. It should be emphasized that the appendage is multilobed in as many as 70% of patients and is lined by pectinate muscles, which can be confused with thrombus (Fig. 21.63). Despite this, the sensitivity of transesophageal imaging for the detection of left atrial thrombus is approximately 95% and in some series has been 100%. Specificity is similarly high. Once visualized, thrombi should be assessed for their size and mobility, and whether they extend into the body of the left atrium. Figure 21.64 is an example of a mobile and protuberant appendage thrombus. Figure 21.65 includes two examples of larger thrombi in the left atrial appendage. Figure 21.66 was recorded from a patient referred for cardioversion of atrial fibrillation. Transesophageal echocardiography demonstrated a mobile thrombus in the appendage. As illustrated, both two- and three-dimensional imaging accurately recorded the mass. The advantages of three-dimensional imaging and its ultimate role in this setting continue to evolve. Figure 21.67 is an example of an unusual left atrial thrombus in a patient with atrial fibrillation. It was not associated with the atrial appendage and appears attached to the interatrial septum, suggestive of a myxoma. However, at the time of surgery, it was proven to be a thrombus. Figure 21.68 is from a patient who had undergone percutaneous closure of his left atrial appendage using an occlusion device. Transesophageal echocardiography performed 6 weeks after implantation shows a thin, amorphous layer of thrombus on the surface of the plug.

**FIGURE 21.59.** From a patient with end-stage renal disease, apical thrombi (arrow) develop in the setting of acute myocardial infarction (A). Despite improvement in apical wall motion following revascularization (B), the thrombi persists (arrows). The apical two-chamber views demonstrate multiple mobile
masses (arrows) within the left ventricular apex, but normal wall motion.

Video 21-59a

coming soon

Video 21-59b

coming soon
FIGURE 21.60. In a patient with untreated rheumatic heart disease, a very large left atrial thrombus (arrows) is seen. The right atrium is also severely dilated.
FIGURE 21.61. The left atrial appendage (asterisk) occasionally can be recorded using transthoracic echocardiography from the apical two-chamber view (A). B: A thrombus within the appendage is indicated by the arrow.
FIGURE 21.62. A thrombus (arrow) is seen with transesophageal echocardiography protruding from the left atrial appendage in this patient with atrial fibrillation.
FIGURE 21.63. Transesophageal echocardiography is used to assess the left atrial appendage for thrombus. A: A normal left atrial appendage is demonstrated. B: The arrows indicate small pectinate muscles within the appendage. These are normal structures that are sometimes confused with thrombi. C: A multilobed appendage is illustrated, the different lobes indicated by the arrows.
FIGURE 21.64. This magnified view of the left atrial appendage demonstrates a small mobile thrombus (arrow).

Echocardiography also allows detection of spontaneous echo contrast (SEC) within the left atrium, possibly a precursor to the development of thrombus formation and certainly a risk factor for embolization (this topic is covered later in this chapter). The most direct evidence of embolic risk is visualization of the thrombus with two-dimensional echocardiography. In addition, pulsed Doppler imaging should also be performed to assess flow velocity within the appendage. Low left atrial appendage–emptying velocity (<20 cm/s) has been reported to significantly increase the embolic risk (Fig. 21.69). Once the left atrial appendage is assessed, the atrial septum should also be interrogated as a possible site for thrombus formation in the presence of an atrial septal aneurysm and/or a PFO. These aneurysms are the result of redundancy of atrial septal tissue leading to a “windsock” appearance within which thrombi may form. In rare instances, echocardiography may
demonstrate thrombus crossing a PFO from the right atrium to the left atrium. Figure 21.70 illustrates a thrombus that probably originated in the lower extremity veins and can be seen straddling the atrial septum through a PFO. This patient had presented with dyspnea, the result of recurring pulmonary emboli. Figure 21.71 is another example of a very mobile thrombus that can be seen crossing the atrial septum via a large PFO.

**FIGURE 21.65.** Two examples of left atrial appendage thrombi. **A:** A relatively small, nonmobile thrombus is indicated by the arrows. **B:** A large thrombus is present (arrows) and appears to fill most of the appendage.

**FIGURE 21.66.** A transesophageal echocardiogram of the left atrial appendage is shown from a patient with atrial fibrillation. **A:** Multiple thrombi (arrows) are demonstrated with two-dimensional imaging. **B:** Using three-dimensional imaging, the thrombi are again visualized (arrows).
Right Atrial Thrombi

Although less common, patients with atrial fibrillation may develop thrombi within the right atrium. The right atrial appendage has a different shape compared with its left-sided counterpart (Fig. 21.72), and echocardiographers are generally less adept at visualizing this structure. However, a right atrial thrombus in the setting of atrial fibrillation is well documented and has been associated with the potential for pulmonary embolus. Thrombi have also been recorded within the right atrium “in transit” (Figs. 21.73 and 21.74). In such cases, the detection of mobile thrombi within the body of the right atrium most likely represents a stage in the development of pulmonary embolus in which thrombi have migrated from lower extremity or pelvic veins into the right side of the heart before embolization to the lungs. Finally, a common source of thrombus formation within the right atrium involves the presence of indwelling catheters or pacemaker leads (Fig. 21.75). In such patients, transesophageal echocardiography is most useful for detecting amorphous and irregularly shaped masses attached to catheters. Such thrombi may become infected or lead to right-sided embolic events. Rarely, right atrial thrombi may be quite large and mistaken for myxoma. Figure 21.76 is from a patient with atrial fibrillation and a hypercoaguuable syndrome. The large mass demonstrated on transeophageal imaging was thrombus and resulted in obstruction to tricuspid valve flow.

Spontaneous Echo Contrast
SEC, or “smoke,” is the swirling, hazy echocardiographic appearance associated with stasis of blood. The development of SEC has been attributed to a variety of low-flow states and the associated red blood cell–protein interactions (e.g., rouleaux formation) that characterize such conditions. To occur, therefore, two conditions must be met. First, there must be a location, usually in the left atrium, right atrium, or left ventricular apex, where stasis or low-flow velocity is present. Then, as a result, some interaction between blood cells and plasma proteins, specifically fibrinogen, must occur (Fig. 21.77). Some investigators have considered SEC a prethrombotic condition, although whether SEC actually leads to thrombus formation is not clearly established. Regardless of cause and effect, the presence of SEC has been consistently associated with increased risk of thromboembolism. SEC is difficult to quantify, and its detection is also dependent on instrument settings. A higher frequency transducer and increased gain settings are sometimes necessary to visualize SEC. One final cautionary note is in order. With modern equipment, using higher frequency transducers and tissue harmonics, SEC may occasionally be seen in normal individuals. This is simply a consequence of highly sensitive instrument settings. The distinction between pathologic and artifactual SEC should be obvious from other echocardiographic clues. For example, if SEC is recorded in the absence of left ventricular failure, mitral stenosis, or atrial fibrillation, it is most likely attributable to machine settings.

**FIGURE 21.67.** A left atrial mass (arrow) is seen in a patient who underwent transesophageal echocardiography prior to cardioversion for atrial fibrillation. In the four-chamber view (A), the mass is attached to the interatrial septum, suggesting myxoma. A three-dimensional image is provided in B, showing the attachment site to the left atrial wall. At surgery, the mass was found to be a thrombus. The left atrial appendage was free of thrombi.
Video 21-67a

coming soon

Video 21-67b

coming soon
FIGURE 21.68. This patient had undergone percutaneous closure of the left atrial appendage using an occlusion device. Follow-up transesophageal echocardiography revealed a thin layer of hazy material on the surface of the device, consistent with thrombus (A and B, arrows). Following 6 weeks of anticoagulation, a repeat study revealed no evidence of residual thrombus (C, small arrows).

Video 21-68b
FIGURE 21.69. A: A left atrial appendage (LAA) thrombus (arrow) is recorded with two-dimensional imaging. B: Pulsed Doppler imaging records low (<20 cm/s) atrial appendage-emptying velocity. Spontaneous echo contrast was also present within the left atrium.
FIGURE 21.70.  

A, B: A thrombus is recorded straddling the interatrial septum through a patent foramen ovale and extending into the left atrium (small arrows). The thrombus was highly mobile and likely originated in the lower extremities. Increased mobility of atrial septal tissue is indicated by the large arrow.
FIGURE 21.71. A thrombus (arrows) is visualized using transesophageal echocardiography straddling the atrial septum through a patent foramen ovale.
ROLE OF ECHOCARDIOGRAPHY IN SYSTEMIC EMBOLUS

One of the most frequent reasons to request an echocardiogram involves the search for a potential cardiac source of embolus. In many large laboratories, this is the single most common indication for transesophageal echocardiography. Embolic events, particularly strokes, can be devastating. Because the cause of a stroke can be difficult to establish on clinical grounds and because embolic strokes are often recurrent, an aggressive attempt to identify potential cardiac sources of emboli is understandable.

FIGURE 21.72. With transesophageal echocardiography, the bicaval view can be adjusted to record the right atrial appendage (*asterisks*). With slight changes in the imaging plane (A, B), the appearance of the appendage is altered.
FIGURE 21.73. Thrombi can occasionally be recorded during transit through the right side of the heart. A to D: Small thrombi are recorded at various locations within the right atrium and right ventricle (arrows). These will most likely lead to a pulmonary embolism.
FIGURE 21.74. A lobulated thrombus is visualized within the right atrium from the right ventricular inflow view (A) and the basal short-axis view (B). The patient had recently suffered a pulmonary embolus. In real time, the tubular-shaped coil of thrombus (arrows) could be seen swirling within the chamber. The right ventricle is dilated and hypokinetic.

Video 21-74a
FIGURE 21.75. Two distinct pacemaker leads are recorded extending from the superior vena cava into the right atrium. A large mass is attached to one lead (arrow). This most likely represents thrombus formation.

FIGURE 21.76. From a patient with atrial fibrillation and hypercoaguable state, transesophageal echocardiography demonstrates a large homogeneous mass in the right atrium (A, arrow). The mass was identified as a thrombus. In B, Doppler demonstrates abnormal flow across the tricuspid valve, due to partial obstruction to inflow.
Unfortunately, the proper use of echocardiography in this setting remains controversial. It is estimated that approximately one-fourth of all strokes are due to a cardiac source of embolus, although the rate is significantly higher in younger patients. A list of potential cardiac sources of embolus is provided in Table 21.6. It is apparent that many of these potential cardiac sources can be identified with echocardiography. In most series, the yield of transesophageal echocardiography is significantly higher than that of transthoracic echocardiography (Table 21.7). For example, atrial thrombi are rarely seen by transthoracic echocardiography (see Fig. 21.61), but readily detected using transesophageal techniques. Using the transthoracic method, only approximately 15% of patients with a suspected embolic event have an identifiable cardiac source. This low incidence may be explained in part by the fact that the echocardiogram is performed after the event so that the cause is no longer present within the heart. An exception to this is the occasional demonstration of thrombus straddling a PFO (see Figs. 21.70 and 21.71). This scenario usually occurs in the setting of thrombus originating from a lower extremity vein, embolizing to the right atrium, and getting caught within the PFO. Such patients often experience both pulmonary and systemic embolism as a result. More importantly, many of the potential cardiac sources of emboli are not easily evaluated from the transthoracic approach. If patients
with evidence of cardiovascular disease (by history and physical examination or electrocardiography) are evaluated with transthoracic echocardiography, the yield is higher, approaching 50%. In all published series, however, transesophageal echocardiography identified a higher percentage of patients with a potential source of embolus. It should be emphasized that although a potential source of embolus may be detected, its presence does not establish a cause-and-effect relationship between the echocardiographic abnormality and the clinical event.

<table>
<thead>
<tr>
<th>Actual Source</th>
<th>Echocardiographic Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV thrombus</td>
<td>Apical aneurysm, presence of thrombus, dilated CM</td>
</tr>
<tr>
<td>LA thrombus</td>
<td>Presence of thrombus in LAA, spontaneous echo contrast, LAA emptying velocity, mitral stenosis, interatrial septal low aneurysm</td>
</tr>
<tr>
<td>Pelvic veins or LE thrombus</td>
<td>ASD, atrial septal aneurysm, PFO</td>
</tr>
<tr>
<td>Native valves</td>
<td>Vegetation, tumor, MVP, mitral annular calcification, sclerotic aortic valve</td>
</tr>
<tr>
<td>Prosthetic valves</td>
<td>Thrombus, vegetation</td>
</tr>
<tr>
<td>Cardiac tumor</td>
<td>LA myxoma, papillary fibroelastoma</td>
</tr>
<tr>
<td>Aorta</td>
<td>Complex aortic plaque, atheroma</td>
</tr>
</tbody>
</table>

| ASD, atrial septal defect; CM, cardiomyopathy; LA, left atrium; LAA, left atrial appendage; LE, lower extremity; LV, left ventricle; MVP, mitral valve prolapse; PFO, patent foramen ovale. |

Many cardiac findings are nonspecific, that is, they are seen with similar frequency in patients with and without embolic events. For example, valve excrescences are seen so commonly in normal, asymptomatic elderly individuals that their detection in patients who have suffered an embolic event is of questionable significance. Aortic atheromas are also seen with regularity on transesophageal imaging (Figs. 21.78 to 21.80). Although they can embolize, their mere presence is usually insufficient proof of cause and effect. A PFO is present in approximately one-third of unselected patients. It
can be detected with either transthoracic or transesophageal imaging, using color flow Doppler imaging or injection of agitated saline (Figs. 21.81 and 21.82). The atrial septum often shows increased mobility or redundancy. A PFO is defined (and differentiated from an atrial septal defect) by the demonstration of atrial shunting in the absence of an anatomic defect or gap in the secundum septum. With transesophageal echocardiography, however, some separation between the overlapping primum and the secundum septa may be seen. This is often respiratory cycle dependent. Once a PFO is demonstrated, estimating its size and the magnitude of shunting has practical implications. In general, separation of the overlapping septal planes by more than 2 mm is consistent with a large PFO. With injection of contrast, the presence of more than 10 microbubbles in the left atrium within three cardiac cycles is also consistent with a large PFO, and it has been suggested that this may confer a stronger link to clinical events.

**Table 21.7** COMPARING THE YIELD OF TTE VERSUS TEE FOR IDENTIFYING POSSIBLE SOURCE OF EMBOLUS

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>n</th>
<th>TTE%</th>
<th>TEE%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pop/1990</td>
<td>72</td>
<td>8</td>
<td>15</td>
</tr>
<tr>
<td>Hofman/1990</td>
<td>153</td>
<td>36</td>
<td>58</td>
</tr>
<tr>
<td>Cujec/1991</td>
<td>63</td>
<td>14</td>
<td>41</td>
</tr>
<tr>
<td>Lee/1991</td>
<td>50</td>
<td>0</td>
<td>52</td>
</tr>
<tr>
<td>De Belder/1992</td>
<td>131</td>
<td>55</td>
<td>70</td>
</tr>
<tr>
<td>Comess/1994</td>
<td>145</td>
<td>ND</td>
<td>45</td>
</tr>
</tbody>
</table>

ND, not done; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography.
Although the incidence of PFO may be higher in young patients who have suffered cerebrovascular events, compared with the general population, the frequency of the finding in the unselected population and the difficulty in establishing cause and effect render the presence of a PFO inconclusive in many cases. In contrast, the combination of PFO and atrial septal aneurysm appears to be associated with a significant increase in risk (Fig. 21.83). In a prospective, multicenter study of patients who had had an ischemic stroke (Mas et al., 2001), the rate of recurrence was increased in the presence of both PFO and an atrial septal aneurysm compared with either condition alone.
FIGURE 21.79. A descending aortic mobile atheroma is seen on transesophageal echocardiography. Biplane two-dimensional images (A) show the mass attached to the aortic wall (arrows). With three-dimensional echocardiography (B), the attachment site and mobility of the atheroma is apparent.
FIGURE 21.80. A large atheroma (arrows) is seen in the ascending aorta in a patient with ischemic cardiomyopathy. A shows a biplane imaging of the ascending aorta. The mobility and attachment site are best visualized in B. The location, just above the sinotubular junction, is an unusual place for mobile atheroma to form.
coming soon

Video 21-80a

coming soon

Video 21-80b
Detecting the presence of a patent foramen ovale often relies on color flow imaging. In this example, a small degree of shunting (arrow) between the right and left atrium is present.
FIGURE 21.82. More extensive shunting is present in this example and is demonstrated using injection of agitated saline through a peripheral vein. The interatrial septum shows excessive mobility, and a clear tunnel-like defect (arrow) is present. The degree of shunting can be estimated by virtue of the number of bubbles that appear within the left atrium.
FIGURE 21.83. An example of an atrial septal aneurysm. A: The aneurysm billows into the left atrium (arrows). B: The redundant tissue billows into the right atrium (large arrow). Injection of contrast into the right side of the heart confirms an associated patent foramen ovale by demonstrating right-to-left shunting.

FIGURE 21.84. From a patient with a submassive pulmonary embolus, transthoracic echocardiography reveals a saddle embolus (arrow) at the bifurcation of the main PA. Two slightly different short-axis views are provided (A, B).

Echocardiography, particularly transesophageal, also can be useful in the setting of pulmonary embolism. Figure 21.84 is an example of a saddle embolus demonstrated using transesophageal echocardiography. Although direct visualization of a pulmonary embolus is uncommon (and better assessed using other modalities such as CT), echocardiography is especially useful in assessment of right ventricular function and pulmonary artery pressure in this setting, both during the acute phase and during subsequent follow-up.

An additional difficulty in this area is the challenge of demonstrating that echocardiographic findings alter management after an embolic event. In the Value of Transesophageal Echocardiography Study (Goldman et al., 1994), among the subset of patients who were studied because of a cerebrovascular event, the results of the echocardiogram affected clinical management in 27% and led to a change in drug therapy in 16%. In most cases, the altered management involved the decision to anticoagulate or close a PFO. It is clear, however, that many patients referred for echocardiography after an embolic event will not see their management altered substantially by the results of the imaging study.
Although the potential for overuse of echocardiography in search of a cardiac source of embolus exists, some studies have supported the cost-effectiveness of this approach. In one investigation (McNamara et al., 1997) in which clinical practice was simulated using a Markov decision model, the cost-effectiveness of different strategies, with and without echocardiography, was compared (Fig. 21.85). Using a hypothetical patient in sinus rhythm who suffers a first stroke, several strategies were tested for the likelihood of establishing a diagnosis and affecting the decision to anticoagulate. The different strategies included various combinations of cardiac history, transthoracic echocardiography, and transesophageal echocardiography, performed in different sequences. Assumptions were made about diagnostic yield, risk of recurrence, likelihood of complications, and outcome, and the cost of each strategy was compared with its utility. Cost-effectiveness was expressed as total cost per quality-adjusted life-year ($/QALY). Transthoracic echocardiography was not cost-effective under any circumstances. In contrast, strategies employing transesophageal imaging were found to be most efficient. Specifically, the two most cost-effective approaches were: (1) transesophageal echocardiography performed only in patients with a history of cardiac problems (most cost-effective, at $8,700 per QALY); and (2) transesophageal echocardiography in all patients ($20,000 per QALY). This was largely based on the ability to detect atrial thrombi and to prevent recurrent strokes by selectively initiating anticoagulation in such patients. The authors concluded that transesophageal echocardiography should be performed in all patients with acute stroke.
FIGURE 21.86. An example of a hiatal hernia is provided. **A:** An echo-free space behind the left side of the heart (*arrows*) is noted. **B:** The short-axis view confirms that the structure is below the diaphragm (*arrows*). **C:** The patient is given a carbonated beverage to drink. This produces a contrast effect within the structure, confirming that it is hiatal hernia.
FIGURE 21.87. These transesophageal images were recorded from a patient 2 days after coronary artery bypass surgery. Systolic (A) and diastolic (B) images are provided. The patient had become hypotensive. A large, amorphous mass within the pericardial space can be seen to impinge on the right atrium and right ventricle. This represents a hematoma that compressed the right side of the heart and contributed to the hypotension.

Although formal guidelines for this application of echocardiography do not exist, some general guidelines are provided in Table 21.2 which summarizes Appropriate Use Criteria in the areas of cardiac masses and sources of embolism. Among patients with a strong clinical suspicion of an embolic event, the yield of echocardiography (especially transesophageal imaging) is reasonable and the test should be considered. Echocardiographic imaging is more likely to provide a diagnosis in younger patients (<50 years) or in patients with known risk factors such as congenital heart disease or a PFO. In most instances, the greater diagnostic yield provided by transesophageal imaging compared with transthoracic echocardiography makes this the technique of choice to search for a potential source of embolus. Finally, the use of echocardiography in this complicated setting should be reserved for those instances in which the results are likely to alter management or to affect therapy. In older patients without clinical evidence of predisposing heart disease who are likely to have cerebrovascular disease, the very low yield of echocardiography argues against its use in this setting.

PSEUDOTUMORS AND OTHER CARDIAC MASSES

In addition to the false-positive results described earlier in this chapter that represent normal variants (Table 21.1), extracardiac masses may impinge on
or compress the heart, creating the illusion of a mass effect. These include tumors within the mediastinum, coronary aneurysms, or hiatal hernias. An example of a hiatal hernia is illustrated in Figure 21.86. The mass appears to be within the atrium but is actually a portion of the stomach. The diagnosis can be clarified by having the patient drink a carbonated beverage during transthoracic imaging. After heart surgery, accumulation of blood and hematoma within the mediastinum or pericardial space can result in external cardiac compression and the illusion of a mass (Figs. 21.87 and 21.88). These usually impinge on the right side of the heart and may affect right ventricular filling or pulmonary blood flow. Although the effects may resolve spontaneously, surgical evacuation is sometimes required.

The development of myocardial cysts is an uncommon complication of echinococcal infection. Although echocardiography is an accurate means of diagnosis, the rarity of the disease contributes to frequent misinterpretation. These cysts most often involve the left ventricular free wall and may project into the chamber or the pericardial space. They tend to be large, thin walled, and septated (Fig. 21.89). Such an appearance is considered classic, and, when present, the echocardiographic diagnosis is straightforward. Color Doppler imaging can be used to confirm the lack of blood flow within the cystic spaces. Rupture can occur and have catastrophic consequences. A more benign condition is the pericardial cyst (Fig. 21.90). These cysts are simple, thin-walled, fluid-filled structures that typically are located within the right costophrenic angle. Because they are benign and usually do not produce symptoms, they must be correctly identified and distinguished from other more serious conditions. Unlike echinococcal cysts, they are extramyocardial and their interior is devoid of loculations or septa. These characteristics, in addition to their typical location, help identify them and distinguish them from malignancy.
FIGURE 21.88. This transthoracic echocardiogram was recorded in a patient 1 week after open-heart surgery. A mass (arrows) is present adjacent to the apex and lateral wall of the left ventricle. This likely represents a pericardial hematoma. The patient was clinically stable, and the mass gradually resolved.
FIGURE 21.89. An echinococcal cyst (arrows) within the interventricular septum is demonstrated in a patient who had recently emigrated from the Middle East. The mass is seen in the long-axis (A), modified long-axis (B), and four-chamber (C) views. The large hydatid cyst is typical of cardiac involvement of echinococcal infection.

FIGURE 21.90. A large pericardial cyst (arrows) is demonstrated from the apical four-chamber view. These cysts are typically circular, thin walled, and echo free. They are often located near the right costophrenic angle.
Suggested Readings

**GENERAL CONCEPTS**


**SOURCE OF EMBOLUS**


Rastegar R, Harnick DJ, Weidemann P, et al. Spontaneous echo contrast video density is flow-related and is dependent on the relative concentrations of fibrinogen and red blood cells. *J Am Coll Cardiol* 2003;41:603–610.


**Atrial Fibrillation and Cardioversion**


Leung DY, Black IW, Cranney GB, Hopkins AP, Walsh WF. Prognostic implications of left atrial


**MASSES AND TUMORS**


Rey M, Alfonso F, Torrecilla EG, et al. Diagnostic value of two-dimensional echocardiography in


Chapter 22

Echocardiography in Systemic Disease and Specific Clinical Presentations

ECHOCARDIOGRAPHY AND SYSTEMIC DISEASE

There are many systemic diseases with cardiovascular manifestations for which echocardiography is appropriate and critical for complete clinical evaluation (Tables 22.1 and 22.2). This chapter discusses the integration of clinical and echocardiographic information for the management of patients with a variety of clinical presentations and disease states.

Hypertension

Clinically, echocardiography is used to detect end-organ cardiac damage due to hypertension, including left ventricular hypertrophy (Fig. 22.1), diastolic dysfunction, and in advanced stages both systolic and more advanced diastolic dysfunction (Fig. 22.2). Numerous algorithms have been proposed for the determination of left ventricular mass and for quantifying left ventricular hypertrophy. The M-mode–derived Teichholz or cubed formula, which assumes spherical geometry of the left ventricle, was used in most early hypertension studies. Because the left ventricle does not adhere to spherical geometry, the absolute measurements are often inaccurate due to tangential imaging planes. Nevertheless, assuming no intervening event such as myocardial infarction, this methodology provides a relatively stable estimate of left ventricular mass over time in any given patient and has for this reason been used successfully for tracking left ventricular mass in
Diastolic dysfunction is one of the earliest manifestations of hypertensive heart disease. In advanced cases of severe untreated hypertension, it may progress to being the predominant contributor to congestive heart failure (CHF) symptoms. Methods by which diastolic dysfunction is evaluated in hypertensive patients are the same as for other diseases (Chapter 6). Generally, in early hypertension, there is delayed relaxation of the myocardium because of hypertrophy, fibrosis, and stiffening, which is manifested as a reduced E/A ratio of mitral valve inflow (Fig. 22.1). Doppler tissue imaging or tissue tracking techniques provide a more detailed characterization of myocardial mechanics, and reveal subclinical abnormalities in hypertensive cardiovascular disease not apparent by standard imaging. It should be emphasized that reduced systolic strain, while a sensitive marker for preclinical hypertensive cardiovascular disease, is nonspecific and has also been reported in preclinical infiltrative and hypertrophic cardiomyopathies and is present in a broad range of other disease states as well. As such, utilization of these advanced parameters clearly needs to be put in context of the clinical situation.

If left ventricular hypertrophy remains uncomplicated by concurrent systolic dysfunction, no other changes are anticipated. In severe long-standing hypertension, left ventricular systolic dysfunction may develop. In patients with end-stage hypertensive heart disease there often is evidence of combined systolic and diastolic dysfunction (Fig. 22.3).

Other cardiac anomalies, which have a relatively greater prevalence in the hypertensive population, include left atrial dilation, calcification of the mitral annulus, and mild degrees of aortic valve insufficiency. With long-standing hypertension, there may be secondary dilation of the ascending aorta with effacement of the sinotubular junction. This has the effect of splaying the closure of the aortic cusps and may result in secondary aortic insufficiency. Patients may present in hypertensive crises, in which case, marked hypertension is seen in association with CNS effects including headache, papillary edema, and often CHF. Echocardiographic findings usually include evidence of pre-existing hypertension such as left ventricular hypertrophy and left atrial enlargement. Systolic function may be compromised (Fig. 22.4). If hypertensive crises are seen in the absence of evidence of pre-existing hypertension, pheochromocytoma, or acute drug toxicity should be
considered.

**Diabetes Mellitus**

Diabetes mellitus is associated with primary and secondary cardiovascular abnormalities. For patients with diabetes, the metabolic derangement results in premature coronary artery disease, sometimes in a very aggressive manner. For type 2 diabetes, especially if seen as part of a generalized “metabolic disorder,” there is an increased prevalence of lipid disorders and hypertension. Detection of coronary disease in the population with diabetes is done in a manner identical to that of the population without diabetes, including the use of rest and stress echocardiography. From a clinical standpoint, it should be recognized that because of the autonomic neuropathy associated with diabetes, typical symptoms may not be present. As such, the indications for proceeding with provocative cardiovascular stress testing, and the end points for termination of a cardiovascular stress test, may not be the same as they are in the population of patients without diabetes.

In addition to premature coronary disease, there may be less clinically obvious cardiovascular manifestations. There is a well-recognized tendency to develop diastolic dysfunction even in the absence of “significant” hypertension or coronary artery disease. This is presumed to be due to accumulation of metabolic byproducts within the myocardial interstitium, resulting in stiffening of the myocardium and delayed relaxation. In routine clinical practice, this is manifested as a reduced E/A ratio of mitral valve inflow and reduced annular e’. It is well recognized that the mitral valve E/A ratio diminishes with age; however, in the population with diabetes, the rate at which it diminishes exceeds that in the population without diabetes due to occult diastolic dysfunction. Reductions in strain and strain rate have been demonstrated in preclinical diabetic heart disease as well. The degree to which aggressive control of even borderline hypertension and scrupulous control of blood glucose will mitigate these changes has yet to be determined.

<p>| Table 22.1 | APPROPRIATENESS CRITERIA FOR THE APPLICATION OF ECHOCARDIOGRAPHY IN SYSTEMIC DISEASE AND CLINICAL DECISION |</p>
<table>
<thead>
<tr>
<th>Indication</th>
<th>Appropriateness Score (1–9)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Arrhythmias/syncope/neurologic events</strong></td>
<td></td>
</tr>
<tr>
<td>4. Frequent VPCs or exercise-induced VPCs</td>
<td>A (8)</td>
</tr>
<tr>
<td>5. Sustained or nonsustained atrial fibrillation, SVT, or VT</td>
<td>A (9)</td>
</tr>
<tr>
<td>7. Clinical symptoms or signs consistent with a cardiac diagnosis known to cause lightheadedness/presyncope/syncope (including but not limited to aortic stenosis, hypertrophic cardiomyopathy, or HF)</td>
<td>A (3)</td>
</tr>
<tr>
<td>8. Lightheadedness/presyncope when there are no other symptoms or signs of cardiovascular disease</td>
<td>A (7)</td>
</tr>
<tr>
<td>9. Syncope when there are no other symptoms or signs of cardiovascular disease</td>
<td>A (9)</td>
</tr>
<tr>
<td>58. Suspected cardiovascular source of embolus</td>
<td></td>
</tr>
<tr>
<td><strong>Pulmonary hypertension</strong></td>
<td></td>
</tr>
<tr>
<td>15. Evaluation of suspected pulmonary hypertension including evaluation of right ventricular function and estimated pulmonary artery pressure</td>
<td>A (9)</td>
</tr>
<tr>
<td>17. Routine surveillance (≥1 yr) of known pulmonary hypertension without change in clinical status or cardiac examination</td>
<td>A (9)</td>
</tr>
<tr>
<td><strong>Disease of the aorta</strong></td>
<td></td>
</tr>
<tr>
<td>63. Evaluation of the ascending aorta in the setting of a known or suspected connective tissue disease or genetic condition that predisposes to aortic aneurysm or dissection (e.g., Marfan syndrome)</td>
<td>A (9)</td>
</tr>
<tr>
<td>64. Reevaluation of known ascending aortic dilation or history of aortic dissection to establish a baseline rate of expansion or when the rate of expansion is excessive</td>
<td>A (9)</td>
</tr>
<tr>
<td><strong>Hypertension cardiomyopathy/heart failure</strong></td>
<td></td>
</tr>
<tr>
<td>67. Initial evaluation of suspected hypertensive heart disease</td>
<td>A (8)</td>
</tr>
<tr>
<td>68. Routine evaluation of systemic hypertension without symptoms or signs of hypertensive heart disease</td>
<td>rA (3)</td>
</tr>
<tr>
<td>70. Initial evaluation of known or suspected HF (systolic or diastolic) based on symptoms, signs, or abnormal test results</td>
<td>A (9)</td>
</tr>
<tr>
<td>74. Routine surveillance (&lt;1 yr) of HF (systolic or diastolic) when there is no change in clinical status or cardiac exam</td>
<td>rA (2)</td>
</tr>
<tr>
<td>75. Routine surveillance (≥1 yr) of HF (systolic or diastolic) when there is no change in clinical status or cardiac exam</td>
<td>U (6)</td>
</tr>
<tr>
<td>91. Baseline and serial reevaluation in a patient undergoing therapy with cardiotoxic agents</td>
<td>A (9)</td>
</tr>
<tr>
<td><strong>TEE as initial or supplemental test</strong></td>
<td></td>
</tr>
<tr>
<td>109. Evaluation for cardiovascular source of embolus with no identified noncardiac source</td>
<td>A (7)</td>
</tr>
<tr>
<td>111. Evaluation for cardiovascular source of embolus with a known cardiac source in which a TEE would not change management</td>
<td>rA (1)</td>
</tr>
<tr>
<td>112. Evaluation to facilitate clinical decision making with regard to</td>
<td>A (9)</td>
</tr>
</tbody>
</table>
Management of the patient with diabetes requires guidelines different from those for patients without diabetes. For a patient with diabetes requiring a major noncardiac surgical procedure, such as renal transplantation or vascular surgery, provocative stress testing, most often with dobutamine stress echocardiography, is typically recommended to identify occult coronary artery disease, even in the absence of classic symptoms, and at younger ages than typically recommended. The strength of evidence to support this recommendation is not robust, but rather based on consensus of opinion. Similarly, the frequency with which diagnostic testing should be repeated is greater than it is for the population without diabetes. After coronary artery bypass surgery, guidelines suggest routine postoperative stress testing only after 5 years. The likelihood of rapid progression is substantially greater in patients with diabetes, and many authorities have recommended earlier and more frequent provocative stress testing (including stress echocardiography) in diabetics.

**Thyroid Disease**

Both hyperthyroidism and hypothyroidism result in cardiovascular disease. Hyperthyroidism results in an increase in total blood volume as well as an increase in left ventricular contractility and a decrease in systemic vascular resistance. This results in a high-output state with an increased left ventricular stroke volume. In addition to these hemodynamic effects, hyperthyroidism results in sinus tachycardia and on occasion may trigger atrial fibrillation. In patients with underlying structural heart disease, the increase in heart rate and stroke volume may precipitate heart failure or unmask previously
compensated heart failure or angina. Extreme hyperthyroidism may result in a high-output state sufficient to result in the appearance of a dilated cardiomyopathy. The cardiomyopathy of hyperthyroidism typically reverses after successful treatment of the metabolic disorder. Hypothyroidism results in directionally opposite changes in left ventricular performance and cardiac output. Pericardial effusion occurs frequently, but even when severe, is an uncommon cause of hemodynamic compromise (Fig. 22.5). In most contemporary practices, significant cardiac effects of unrecognized, untreated thyroid disorders are infrequently encountered.

**Chronic Renal Insufficiency**

Chronic renal insufficiency is often the result of hypertension or diabetes, which, as discussed previously, result in premature coronary artery disease and other anatomic and/or physiologic cardiac abnormalities. In addition to the above secondary features, the metabolic derangements in chronic renal insufficiency, including hyperparathyroidism, result in ectopic calcification, predominantly of the fibrous skeleton of the heart. This is most often manifested as calcification of the mitral annulus (Fig. 22.6) (see Chapter 11 for further examples). The degree of annular calcification is related to the magnitude of hyperparathyroidism and can range from small focal deposits to extensive circumferential deposits of calcium in the annulus. In advanced cases, the calcification invades the proximal mitral valve leaflets and may cause functional mitral stenosis. Secondary features of chronic renal insufficiency include left ventricular hypertrophy due to hypertension and an abnormal texture of the hypertrophied myocardium that mimics that seen in cardiac amyloid (Fig. 22.7). Other abnormalities seen in chronic renal insufficiency include pericardial effusion, which may range from small chronic effusions to presentation with cardiac tamponade. Uremia results in inflammatory and occasionally hemorrhagic pericarditis in which there is often evidence of “stranding” on the visceral pericardium (Fig. 22.8).

<p>| Table 22.2 | SYSTEMIC DISEASES AND CLINICAL PRESENTATIONS IN WHICH ECHOCARDIOGRAPHY PLAYS A VALUABLE ROLE |</p>
<table>
<thead>
<tr>
<th>Systemic conditions with cardiovascular manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Pregnancy</td>
</tr>
<tr>
<td>Chronic renal insufficiency</td>
</tr>
<tr>
<td>Chronic liver disease</td>
</tr>
<tr>
<td>Connective tissue disease</td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
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<tr>
<td>Scleroderma</td>
</tr>
<tr>
<td>Marfan syndrome</td>
</tr>
<tr>
<td>Pulmonary arterial hypertension</td>
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<tr>
<td>Miscellaneous diseases</td>
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<tr>
<td>Thyroid disease</td>
</tr>
<tr>
<td>Sarcoidosis</td>
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<tr>
<td>Hemochromatosis</td>
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<tr>
<td>Muscular dystrophies</td>
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<tr>
<td>Friedreich ataxia</td>
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<td>Carcinoid syndrome</td>
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<td>Ergotamine toxicity</td>
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<table>
<thead>
<tr>
<th>Clinical presentations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Dyspnea</td>
</tr>
<tr>
<td>Pulmonary embolus</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>Cardioembolic disease</td>
</tr>
<tr>
<td>Radiation therapy</td>
</tr>
<tr>
<td>Syncope</td>
</tr>
<tr>
<td>Athletic screening</td>
</tr>
<tr>
<td>Pregnancy</td>
</tr>
</tbody>
</table>
FIGURE 22.1. Apical four-chamber view with mitral inflow (upper right), pulmonary vein flow (upper left), and Doppler tissue imaging of the annulus (lower panels) in a patient with long-standing hypertension. Note the reversal of the mitral E/A ratio, which is paralleled by reversal of annular velocities, all consistent with grade 1 diastolic dysfunction in this otherwise healthy 45-year-old patient. [Video 22-1]
FIGURE 22.2. Transthoracic echocardiogram recorded in a patient with long-standing, poorly controlled hypertension. In the parasternal long-axis view, note the symmetric left ventricular hypertrophy with left atrial dilation. The mitral valve inflow pattern reveals a pseudonormal inflow pattern with a mitral valve E/A ratio of 1.6 and a deceleration time of 214 m/s. Note the pathologically reduced annular E wave (e') of 4.5 cm/s, all consistent with grade 2 diastolic dysfunction. 

Video 22-2
FIGURE 22.3. Apical 4-chamber view recorded in a patient with advanced hypertensive heart disease revealing mild left atrial dilation, left ventricular hypertrophy and evidence of diastolic dysfunction. In the real-time image, note the global hypokinesis of the left ventricle.

Video 22-3
FIGURE 22.4. Parasternal long-axis view recorded in a patient presenting with hypertensive crisis with systolic blood pressures greater than 220 mm Hg and evidence of acute congestive heart failure with pulmonary congestion and a BNP of 3,000. In the parasternal long-axis view, note the normal internal dimension of the left ventricle and the hypertrophy of both the septal and posterior walls. In the real-time image, note the global hypokinesis of the left ventricle. Over a 6-month period with adequate blood pressure control, left ventricular systolic function improved to 50%. Video 22-4
FIGURE 22.5. Echocardiogram recorded in a patient with profound hypothyroidism (TSH >300). Note the large PEF with a swinging heart in the real-time image. The patient had no clinical evidence of hemodynamic compromise. Incidental note is made of severe left ventricular hypertrophy, presumably related to long-standing hypertension.
FIGURE 22.6. Parasternal long- and short-axis echocardiograms recorded in a patient with chronic renal insufficiency and calcification of the mitral annulus. A: In the parasternal long-axis view, notice the focal deposits in the posterior annulus (arrow), which have resulted in a side lobe artifact mimicking an associated mass. B: In the short-axis view, notice the crescent of calcium encompassing the posterior mitral annulus (arrows).
FIGURE 22.7. Parasternal long-axis echocardiogram recorded in a patient with end-stage renal disease. Left ventricular hypertrophy with abnormal myocardial texture, as well as a moderate to large PEF, is present. 

Video 22-7

coming soon
On occasion, patients with chronic renal insufficiency develop systolic dysfunction, which cannot be related to uncontrolled hypertension, coronary artery disease, or other identifiable factors. The presumed etiology of the dysfunction is accumulation of metabolic byproducts, including metalloproteinases, in the myocardium. Numerous cases have been reported in which systolic function recovers after institution of more aggressive dialysis or renal transplantation. Figure 22.9 was recorded in a 34-year-old patient with end-stage renal disease related to glomerulonephritis. Note the significant systolic dysfunction in the real-time images and evidence of marked diastolic dysfunction. Figure 22.10 was recorded 6 months after renal transplantation and demonstrates marked reversal of both the systolic and diastolic dysfunction.

**FIGURE 22.8.** Subcostal echocardiogram recorded in a patient with chronic renal insufficiency and a large pericardial effusion localized over the right atrium and right ventricle. Note the stranding between the visceral and parietal pericardium (arrows), implying an inflammatory process.
coming soon

Video 22-8
FIGURE 22.9. Parasternal long- (A) and short-axis (B) echocardiogram recorded in a patient with chronic renal insufficiency (known not to have coronary artery disease). In the real-time images, note the global hypokineses of the ventricle and the mildly abnormal myocardial texture. The Doppler insets demonstrate an elevated mitral E/A ratio with reduced annular e′/a′ ratio implying restrictive physiology.
The metabolic derangements associated with end-stage renal disease may also result in premature degeneration of bioprosthetic valves. Figure 22.11 was recorded in a patient with end-stage renal disease and secondary hyperparathyroidism who developed severe stenosis of a porcine mitral bioprosthesis 14 months after implantation. Parathyroid hormone levels were markedly elevated.

CONNECTIVE TISSUE/AUTOIMMUNE DISEASE

Systemic Lupus Erythematosus

There can be substantial crossover among many of the connective tissue diseases such as systemic lupus erythematosus (SLE), mixed connective tissue disease, Raynaud phenomenon, and scleroderma, all of which may have cardiovascular manifestations. A classic lesion encountered in patients with SLE is noninfectious endocarditis (the Libman–Sacks lesion) (Figs. 22.12 and 22.13). These are most commonly encountered on the atrial aspect of the mitral valve. They tend to be less mobile than infectious vegetations. They may have an inflammatory component that can result in leaflet deformity and variable degrees of valvular regurgitation. When encountered on the aortic valve, they are usually on the arterial side. They may resolve with successful therapy of the underlying connective tissue disease. Additional manifestations of SLE include acute pericarditis and
pulmonary hypertension. There are no characteristic features of the pericarditis or pulmonary hypertension seen in SLE which distinguish them from other etiologies. A rare manifestation of SLE is coronary vasculitis, which can result in regional or global dysfunction and thereby mimic either an acute coronary syndrome or cardiomyopathy.
Antiphospholipid Antibody Syndrome

Antiphospholipid antibody syndrome is closely related to many connective tissue diseases and has been reported as an integral part of SLE. This syndrome results in a variably hypercoagulable state with a tendency for both venous and arterial thrombosis. In addition, patients with the antiphospholipid antibody syndrome develop sterile valvular vegetations similar to those seen in SLE. Although not intrinsically destructive, they may result in valvular regurgitation (Fig. 22.14). From an imaging perspective they appear identical to Libman–Sacks lesions. They may resolve with anticoagulation and successful treatment of the underlying systemic illness. In all likelihood, some individuals previously diagnosed with Libman–Sacks vegetative lesions may have had sterile vegetations related to the antiphospholipid antibody syndrome. Patients with antiphospholipid antibody syndrome may pose unique challenges with respect to anticoagulation. On occasion, the antiphospholipid antibody syndrome and/or associated abnormalities have resulted in valvular disease sufficient to warrant valve replacement. Prosthetic valves in this situation are more prone to thrombotic complications, and on occasion, bioprosthetic valves have been subject to either rapid degeneration or early thrombus formation. Figure 22.15 was
recorded in a patient with SLE and antiphospholipid antibody syndrome who underwent mitral valve replacement with a bioprosthetic valve because of thrombotic complications related to a mechanical prosthesis. The bioprosthetic valve replacement was complicated by early formation of thrombus on both the sewing ring and valve cusps.

FIGURE 22.11. Parasternal long-axis view recorded 14 months after placement of a bioprosthetic mitral valve prosthesis for combined mitral stenosis and insufficiency in a patient with end-stage renal disease and hyperparathyroidism. Note the marked thickening of the mitral valve cusps and, in the inset, the mitral inflow Doppler documenting a mean gradient of 16 mm Hg across the stenotic mitral valve. Of note, there is also a bioprosthetic aortic valve, also demonstrating signs of early leaflet thickening and degeneration.
On rare occasions, a catastrophic antiphospholipid antibody syndrome (CAPAS) develops with acute severe multiorgan system failure related to both arterial and venous microthrombosis. Myocardial necrosis may be a part of this syndrome. From an echocardiographic perspective, it will present as acute vegetative lesions and/or myocardial necrosis. Instances of isolated papillary muscle rupture have been reported (Fig. 22.16).
FIGURE 22.12. Transesophageal echocardiogram recorded in a (arrow) longitudinal view of the aorta revealing a mass on the ventricular aspect of the aortic cusp in a patient with systemic lupus erythematosus, representing a Libman–Sacks vegetation. [Video 22-12](coming soon)
Scleroderma/Raynaud Phenomenon

Many other connective tissue diseases can have cardiovascular manifestations. Diseases closely related to SLE such as mixed connective tissue disease represent a crossover category for which the different manifestations of SLE may be seen. Patients with Raynaud phenomenon or with the full complex of scleroderma have a greater than usual prevalence of pulmonary arterial hypertension. In patients with scleroderma, the echocardiographic manifestations of pulmonary hypertension are identical to those seen in primary pulmonary hypertension (Fig. 22.17). Concurrent pericardial effusion may be more common in scleroderma than in pulmonary hypertension of other etiologies. The manifestations of pulmonary hypertension as a distinct entity are discussed further in this chapter, and the echocardiographic features of right ventricular pressure overload have been discussed in Chapters 8 and 12.

**FIGURE 22.13.** Transesophageal echocardiogram from behind the left atrium with an expanded view of the mitral valve in a young female patient with systemic lupus and Libman–Sacks endocarditis. A: Note the multiple nodular irregularities on the tips of both the anterior and posterior mitral valve leaflets (arrows). The inset is the pathologic specimen obtained at the time of mitral valve replacement revealing diffuse thickening and nodular irregularity of both the anterior and posterior mitral valve leaflets. B is the accompanying color flow Doppler revealing severe mitral regurgitation.
FIGURE 22.14. Parasternal long-axis view in a patient with connective tissue disease and antiphospholipid antibody syndrome. Note the small, immobile masses on the atrial aspect of both the anterior and posterior mitral valve leaflets (arrows) (A) and the moderate mitral regurgitation on color flow Doppler (B).

Video 22-14

FIGURE 22.15. A: Transesophageal echocardiogram recorded in a patient with systemic lupus, antiphospholipid antibody syndrome, and a hypercoagulable state. The patient had undergone bioprosthetic mitral valve replacement and has had early thrombus formation on the cusps, as well as the bioprosthetic stents (arrows). B is a color flow Doppler revealing only mild central mitral regurgitation, suggesting a nondestructive process.
coming soon

Video 22-15
FIGURE 22.16. Transesophageal echocardiogram recorded in a 24-year-old patient with connective tissue disease and catastrophic antiphospholipid antibody syndrome. Note the rupture of the papillary muscle (arrows) (A) and the highly eccentric mitral regurgitation jet related to a flail mitral leaflet (B).
FIGURE 22.17. Transthoracic echocardiogram recorded in a patient with scleroderma and pulmonary hypertension. A: Note the PEF as well as the dilation of the right ventricle and the right ventricular overload pattern on the ventricular septum. B: In the apical four-chamber view, note the marked right heart dilation with tricuspid regurgitation. In the inset, note the elevated tricuspid regurgitation
CHRONIC LIVER DISEASE AND CIRRHOSIS

There are several clinical situations in which cardiac and hepatic diseases intersect. This includes cardiac diseases that either mimic or result in hepatic disease and hepatic diseases that secondarily result in cardiac disease (Table 22.3). Clinical liver disease can occur as a result of cardiovascular disease when either poor cardiac output with malperfusion occurs, or there is long-standing right ventricular dysfunction with elevated systemic venous pressure. Poor perfusion due to low cardiac output may result in multisystem organ dysfunction, and typically the liver is only one of several organs involved. In this instance, there usually will be biochemical evidence of both synthetic dysfunction and reduced clearance of metabolites. In rare occasions, either poor hepatic perfusion or elevated venous pressures with hepatic congestion result in an obstructive biochemical pattern.

**Table 22.3** HEART AND LIVER DISEASE

<table>
<thead>
<tr>
<th>Cardiac disease with an impact on hepatic function</th>
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<tbody>
<tr>
<td>Malperfusion (hypotension/low-output state)</td>
</tr>
<tr>
<td>Passive venous congestion</td>
</tr>
<tr>
<td>Pericardial constriction</td>
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</table>
In patients with chronic right heart failure, systemic venous pressures are chronically elevated, which results in passive hepatic venous congestion. Chronically, this results in the syndrome of “cardiac cirrhosis,” which has distinct histologic features. The actual manifestations of liver involvement are often indistinguishable from multiple forms of primary liver disease. This syndrome should be suspected when there is evidence of chronic hepatic dysfunction in the setting of cardiac disease likely to cause elevation of hepatic venous pressure. Not uncommonly the manifestations of the liver disease predominate and the cardiac disease is overlooked. Cardiovascular diseases that may result in this syndrome are constrictive pericarditis, restrictive cardiomyopathy, primary pulmonary hypertension, and mitral stenosis or dilated cardiomyopathy with secondary pulmonary hypertension. Cardiac cirrhosis occasionally develops in patients with severe tricuspid regurgitation without pressure elevation.

There are also secondary effects of liver disease on the cardiovascular system. Advanced cirrhosis of any etiology is frequently associated with low systemic vascular resistance. This results in a chronic high-output state in which the resting cardiac output may exceed 10 L/min. In this situation, one encounters hyperdynamic left ventricular function with a resting ejection fraction exceeding 65% (Fig. 22.18). For patients with chronic liver disease, the echocardiographer should be cognizant of the anticipated supernormal left ventricular function and the relatively high ejection fraction. A normal or below normal ejection fraction in the presence of clinically relevant chronic liver disease should raise suspicion of an occult cardiomyopathy or concurrent coronary disease.

End-stage liver disease accompanied by cirrhosis of virtually any etiology results in multiple cardiovascular abnormalities including hypothesized
abnormalities of the myocardium which is referred to as a “cirrhotic cardiomyopathy.” While peripheral vascular resistance is reduced, and cardiac output elevated, changes occur within the myocardium resulting in occult diastolic abnormalities. Often the low peripheral vascular resistance masks any overt manifestations of the cirrhotic cardiomyopathy which may subsequently become manifest following liver transplantation or at the time of volume loading.

In addition, because of the elevated flow, pulmonary artery systolic pressures of 35 to 60 mm Hg may be seen with normal pulmonary vascular resistance (Fig. 22.19). This is analogous to the elevation in pulmonary artery systolic pressure seen in a left-to-right shunt, such as atrial septal defect, or in the high-output state of pregnancy. Mild elevation of pulmonary artery systolic pressure in chronic liver disease is not necessarily an indication of abnormalities of the pulmonary vasculature. Pulmonary hypertension with elevated pulmonary vascular resistance (not as a result of high flow) also has been associated with chronic liver disease and is referred to as “portopulmonary hypertension.” The echocardiographic appearance of portopulmonary hypertension is identical to that of pulmonary hypertension of any other etiology. Reversal of moderate portopulmonary hypertension may occur following liver transplant. Marked pulmonary hypertension of any cause confers a nearly prohibitive risk for liver transplantation.

Other anomalies that can be seen in patients with chronic liver disease include pulmonary arteriovenous malformations (AVMs), resulting in the hepatopulmonary syndrome. The size of the shunt ranges from mild and inconsequential (Fig. 22.20) to large, resulting in clinically relevant levels of right-to-left shunting (Fig. 22.21). These can be detected with saline contrast echocardiography. They result in a delayed continuous right-to-left shunt, compared to an early phasic shunt seen with an atrial level shunt. Additional features of a pulmonary AVM include a gradual increase over time in the contrast appearing in the left heart and identification of saline contrast in the pulmonary veins. In the presence of a large pulmonary AVM, saline contrast intensity in the left heart progressively increases over time and may, after a delay, exceed the intensity in the right heart. For patients with chronic liver disease presenting with hypoxia, saline contrast echocardiography should be performed to identify any pathologic right-to-left shunt due to pulmonary AVMs. If the magnitude of shunting is significant, percutaneous closure of
the pulmonary AVM may be beneficial. Identification of such a shunt also assists in clinical management because it may provide an explanation for otherwise unexplained arterial desaturation.
FIGURE 22.18. Parasternal long-axis view recorded in diastole (A) and systole (B) in a patient with end-stage liver disease and a high-output state. Resting cardiac output was 16 L/min in the catheterization laboratory. Note the mild dilation of the left atrium and left ventricle and the hyperdynamic motion of the left ventricle at rest. Incidental note is made of a small pericardial effusion (arrow).

Video 22-18

Patients with chronic liver disease may have abdominal distention due to either an enlarged liver or ascites. The effect of this is to elevate the diaphragm and compress the heart from below, occasionally resulting in the need for atypical imaging windows. If the posterior wall is compressed, “pseudodyskinesis” of the posterior wall may be noted. The genesis of this phenomenon is illustrated in Figure 22.22. In this situation, the posterior wall is compressed superiorly by the diaphragm and results in abnormal short-axis geometry in diastole. With active myocardial contraction, the ventricle reassumes circular geometry resulting in “pseudodyskinesis” of the inferior or posterior wall. Focusing on myocardial thickening rather than endocardial excursion can help avoid confusing this phenomenon for myocardial ischemia.
FIGURE 22.19. Spectral Doppler imaging recorded in the patient presented in
Figure 22.18. Note the peak tricuspid regurgitation velocity of 3.4 m/s and the greater than usual time velocity integral (TVI) of both left ventricular outflow tract and right ventricular outflow tract.

Video 22-19

Video 22-19

FIGURE 22.20. Apical four-chamber view recorded in a patient with end-stage liver disease and right-to-left shunting related to a small pulmonary AVM. In the central figure, notice the complete opacification of the right atrium and ventricle and the small amount of contrast in the left atrium and ventricle. The upper right is
the precontrast injection confirming absence of contrast in any of the four chambers. In the real-time image, note the gradual and continuous flow of contrast into the left atrium and left ventricle consistent with a pulmonary AVM rather than phasic inflow more consistent with an atrial level shunt.

**FIGURE 22.21.** Apical four-chamber view with intravenous saline contrast recorded in a patient with end-stage liver disease and a large pulmonary arterial venous malformation. **A:** Contrast is present in the right atrium and right ventricle but has not yet appeared in the left atrium or left ventricle. The two pulmonary veins are free of contrast (arrows). **B:** Recorded after image A and shows opacification of the left atrium and left ventricle. Note also that the contrast can be clearly seen in the pulmonary veins (arrows), documenting that the level of shunt is not directly at the atrial level but rather due to a pulmonary arteriovenous malformation.
Occasionally, when performing transesophageal echocardiography in a patient with end-stage liver disease, one encounters large cystic vascular structures adjacent to the esophagus (Fig. 22.23). These represent dilated venous collaterals due to portal hypertension.
FIGURE 22.22. Parasternal short-axis view recorded in a patient with end-stage liver disease and significant hepatomegaly, which has elevated the diaphragm. This has resulted in compression of the inferior wall (arrows) resulting in a noncircular geometry of the left ventricle in diastole (A). B: In early systole, with active ventricular contraction, the ventricle reassumes a circular position, giving the appearance of paradoxic motion in the inferior wall. Note that systolic thickening is preserved. A similar pattern of inferior wall pseudodyskinesis can be
seen in any entity that results in sufficient abdominal distention to compress the left ventricle inferiorly including significant hepatomegaly, ascites, or pregnancy.

Video 22-22

coming soon
Finally, patients with chronic liver disease may be evaluated for cardiovascular risk prior to liver transplantation. Although dobutamine stress echocardiography is accurate for separating low- from high-risk patients presenting for most surgical procedures, its ability to identify patients likely to have perioperative complications associated with liver transplantation is less well established. Many cases of cardiovascular compromise after liver transplantation may relate to an underlying cardiomyopathy that was masked by low peripheral vascular resistance and would not be expected to be detected with dobutamine stress echocardiography. Immediately after liver transplantation, there is an acute increase in systemic vascular resistance (to normal or above), commonly in association with substantial volume loading due to massive transfusion. This may precipitate acute left ventricular decompensation and severe CHF in the absence of ischemic heart disease.

**CHRONIC OBSTRUCTIVE PULMONARY DISEASE**

Chronic lung disease, either obstructive or restrictive, can be associated with significant cardiovascular changes, predominantly mediated by hypoxia-
induced elevation in pulmonary arterial pressure. This leads to right ventricular hypertension with secondary right ventricular hypertrophy (*cor pulmonale*). From a cardiac perspective, the appearance is similar to that of any etiology of pulmonary hypertension, and includes variable degrees of tricuspid regurgitation. Patients with chronic obstructive lung disease frequently have limited parasternal and apical windows because of interference with intervening lung tissue and a more vertical and inferior position of the heart. They often can be better imaged from a subcostal transducer position ([Fig. 22.24](#)) from which virtually all cardiac chambers are often visualized in excellent detail.
FIGURE 22.24. Transthoracic echocardiographic images from a patient with chronic obstructive pulmonary disease and cor pulmonale. A: Subcostal/apical four-chamber view. Note the dilation and hypertrophy of the right ventricle. B: An inflow tract view revealing mild tricuspid regurgitation. In the inset, note the tricuspid regurgitation velocity of 4 m/s, which, assuming a right atrial 10 mm Hg, corresponds to a right ventricular systolic pressure of 74 mm Hg.

Video 22-24

PULMONARY HYPERTENSION

Pulmonary hypertension occurs either as a primary pulmonary arterial process or as a consequence of pulmonary disease or primary cardiovascular disease. Table 22.4 outlines primary and secondary causes of pulmonary hypertension. Echocardiography plays a valuable role in identifying cardiac disease that has resulted in elevation in pulmonary arterial pressure (WHO group 2 pulmonary hypertension). Examples include detection of shunt lesions such as atrial septal defect, mitral stenosis, or severe left ventricular systolic or diastolic dysfunction. The echocardiographic sequelae of pulmonary hypertension on the right heart are similar irrespective of the etiology (Figs. 22.25 to 22.28). Any disease that results in a right ventricular volume or pressure overload results in dilation and eventual hypertrophy of the right ventricle. The ventricular septum, because it is a shared wall between the right and left ventricles, reflects the magnitude of hemodynamic derangement whether it is a volume or pressure overload. Chronic elevation of right heart pressures also results in dilation of the coronary sinus (Fig.

and frequently “opening” of a patent foramen ovale (PFO), which may result in a right-to-left shunt detectable with color flow Doppler (Fig. 22.30) or with contrast echocardiography (Fig. 22.31). Secondary dilation of the proximal pulmonary artery with functional pulmonary insufficiency is also common (Fig. 22.32).

When a patient is encountered with pulmonary hypertension, echocardiography plays a crucial role in identifying cardiovascular disease that may have resulted in secondary pulmonary hypertension (group 2). Echocardiography is less valuable for making a definitive diagnosis of primary pulmonary arterial hypertension, which in part is a diagnosis of exclusion. For patients with pulmonary hypertension, the echocardiographic examination should be directed to identify cardiac disease likely to have resulted in secondary pulmonary hypertension, such as an intracardiac shunt (Figs. 22.33 and 22.34) or left-sided valve or myocardial disease. This is typically easily accomplished with transthoracic imaging.

<table>
<thead>
<tr>
<th>Table 22.4</th>
<th>ETIOLOGIES OF PULMONARY HYPERTENSION</th>
</tr>
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<tbody>
<tr>
<td><strong>Pulmonary arterial hypertension (WHO group 1)</strong></td>
<td></td>
</tr>
<tr>
<td>Idiopathic</td>
<td></td>
</tr>
<tr>
<td>Inherited</td>
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<tr>
<td>Toxin induced</td>
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<tr>
<td>Anorexigens</td>
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<tr>
<td>Associated with connective tissue disease</td>
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<tr>
<td>Congenital heart disease</td>
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<tr>
<td><strong>Related to elevated left heart disease (WHO group 2)</strong></td>
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<tr>
<td>Valvular heart disease</td>
<td></td>
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<tr>
<td>Left ventricular systolic dysfunction</td>
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<tr>
<td>Left ventricular diastolic dysfunction</td>
<td></td>
</tr>
<tr>
<td>Pulmonary vein stenosis/thrombosis</td>
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</tr>
<tr>
<td><strong>Related to intrinsic pulmonary disease/hypoxia (WHO group 3)</strong></td>
<td></td>
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<tr>
<td>Obstructive lung disease (COPD)</td>
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<tr>
<td>Restrictive lung disease</td>
<td></td>
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<tr>
<td>Obstructive sleep apnea</td>
<td></td>
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<tr>
<td>High altitude</td>
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<tr>
<td><strong>Pulmonary embolus (WHO group 4)</strong></td>
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<tr>
<td>Chronic thromboembolic pulmonary hypertension</td>
<td></td>
</tr>
<tr>
<td><strong>Unclear and multifactorial etiologies (WHO group 5)</strong></td>
<td></td>
</tr>
<tr>
<td>Hematologic disorders</td>
<td></td>
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</tbody>
</table>

FIGURE 22.25. Parasternal long-axis view recorded in a patient with severe primary pulmonary hypertension. In this end-systolic frame, note the dilated right ventricle and the posteriorly displaced ventricular septum (arrows) representing a right ventricular pressure overload. [Video 22-25]

Video 22-25
FIGURE 22.26. Parasternal short-axis view recorded in the same patient presented in Figure 22.25. Again, note the markedly dilated right ventricle and posteriorly displaced, flattened ventricular septum in systole consistent with a right ventricular pressure overload (downward-pointing arrows). Note the hypertrophy of infundibular muscles near the right ventricular outflow tract (large arrow).
FIGURE 22.27. Right ventricular inflow tract view recorded in the same patient presented in Figures 22.25 and 22.26. Note the marked dilation of the right ventricle and right atrium and the severe tricuspid regurgitation. The inset figure is a continuous-wave Doppler through the tricuspid regurgitation jet revealing a peak gradient of 81.8 mm Hg from which right ventricular systolic pressure can be estimated to be 97 mm Hg based on an assumed right atrial pressure of 15 mm Hg. Video 22-27
FIGURE 22.28. Apical four-chamber view recorded in a patient with severe pulmonary arterial hypertension. Note the marked dilation of the right atrium and right ventricle and the significant right ventricular hypertrophy. The moderator band and other right ventricular trabecular structures hypertrophy as well and can assume a mass-like appearance. The left ventricle is small and underfilled and has been compressed out of view. [Video]
Contrast echocardiography is commonly employed to detect the presence of a right-to-left shunt. In many patients with pulmonary hypertension, right atrial dilation results in stretching of the foramen ovale. Variable degrees of right-to-left shunting are common in severe pulmonary hypertension. Separation of the small secondary right-to-left shunt due to a PFO from a shunt attributable to an atrial septal defect is occasionally problematic (Figs. 22.31 and 22.34). However, if significant pulmonary hypertension is present, and is secondary to an atrial septal defect, the magnitude of the shunt will be substantial and the appearance of contrast in the left atrium will be nearly instantaneous and continuous throughout the cardiac cycle. Conversely, shunting through a small PFO is most often phasic and timed with the respiratory cycle.

FIGURE 22.29. Parasternal long-axis view recorded in a patient with long-standing, severe, primary pulmonary hypertension. Note the circular, echo-free space bordered by the left ventricular posterior wall and the left atrium representing a markedly dilated coronary sinus (CS).
coming soon

Video 22-29
FIGURE 22.30. Apical four-chamber view recorded in the same patient depicted
in Figure 22.29. **A:** In this image, note the marked hypertrophy and dilation of the right ventricle and dilation of the right atrium as well as the small, underfilled left ventricle. Severe tricuspid regurgitation is present. **B:** An expanded view of the atrial septum. Note the distinct color flow jet related to right-to-left flow through a patent foramen ovale (arrows).

**FIGURE 22.31.** Apical four-chamber view recorded in the same patient presented in Figures 22.25 to 22.27. In this four-chamber view, saline contrast has been
utilized to identify right-to-left shunt. Note the aneurysmal atrial septum which is bowing from the right atrium to the left atrium (arrows) and the small amount of contrast appearing in the left ventricular cavity related to a patent foramen ovale. In the presence of severe pulmonary hypertension with a true atrial septal defect, significantly more right-to-left shunting would be anticipated.

Video 22-31
FIGURE 22.32. Parasternal short-axis view at the base of the heart in a patient with severe, long-standing pulmonary hypertension. Notice the pathologic dilation of the proximal pulmonary artery (PA) (arrows) and the mild functional pulmonic insufficiency.
Most patients with significant pulmonary hypertension have right atrial and right ventricular dilation. Right ventricular hypertrophy is common as is hypertrophy of trabeculae and muscle bundles. On occasion, the irregular hypertrophy of trabeculae can be confused for thrombus or other masses. Concurrent tricuspid regurgitation is nearly ubiquitous and may range from mild to severe. Interrogation of the tricuspid regurgitation velocity allows calculation of right ventricular systolic pressures. In the absence of obstruction of right ventricular outflow, this equals systolic pressure in the pulmonary artery (Fig. 22.35). The echocardiographic methodology for the determination of right ventricular pressure is discussed in Chapters 8 and 12. Finally, many patients with significant pulmonary hypertension will have abnormal left ventricular filling (reduced mitral valve E/A ratio), related to effective underfilling of the left ventricle (Fig. 22.36). The mitral inflow pattern may revert to normal with reduction in the pulmonary hypertension.

In a subset of patients with elevated pulmonary artery systolic pressure, the causative pathology is pulmonary venous hypertension. This can be the result of any form of left-sided heart disease including mitral stenosis (more so than regurgitation) or severe diastolic dysfunction. Echocardiography can identify patients likely to have pulmonary venous hypertension on the basis of the mitral valve inflow parameters and from estimation of left atrial pressure from a comparison of mitral inflow and annular tissue velocities. Typically, patients with pulmonary venous hypertension will have evidence of significant diastolic dysfunction, whereas patients with a primary increase in pulmonary arterial resistance or who have pulmonary veno-occlusive disease often have what appears to be grade 1 diastolic dysfunction with a reduced E/A ratio related to effective underfilling of the left ventricle.

On occasion, a patient is encountered who is suspected to have pulmonary hypertension on clinical grounds but in whom the right ventricular systolic pressure is calculated to be normal or only borderline elevated. In these cases, reassessing the pressure during exercise (supine bicycle) may unmask exercise-induced pulmonary hypertension (Fig. 22.37). Elevation of pulmonary artery pressure with exercise is not specific to any type of pulmonary hypertension. It may be seen in patients with left ventricular diastolic dysfunction which functionally worsens at elevated heart rates.
There are several echocardiographic features in patients with pulmonary hypertension that confer a worse prognosis. These include marked right atrial enlargement, pericardial effusion, and greater degrees of left ventricular compression by the right ventricle. Pericardial effusion typically does not result in hemodynamic compromise but is simply a manifestation of more marked elevation of right heart pressures. Patients who have marked reversal
of septal curvature with a small, slit-like left ventricle are also more prone to
develop significant, and occasionally fatal, hypotension if systemic
vasodilators are given.

Multiple echocardiographic parameters have been employed in an effort to
quantify right ventricular systolic function. One method is the right
ventricular myocardial performance index. That is calculated in a manner
identical to that for the left ventricle as was discussed in Chapter 8. An
additional measure of right ventricular function is tricuspid annular plane
systolic excursion (TAPSE) (Fig. 22.38) measured from M-mode
interrogation of the tricuspid annulus. Reduced annular excursion is a marker
of compromised right ventricular function and has been associated with a
worsened prognosis in patients with pulmonary hypertension. Several values
of TAPSE have been associated with a worse prognosis in pulmonary
hypertension, acute pulmonary embolus and in other disease states. Many
studies have suggested that TAPSE <14 mm confers a worsened prognosis in
most commonly encountered clinical situations. The S-wave of annular
velocity provides parallel information to TAPSE but its prognostic value has
been less extensively studied.
FIGURE 22.34. Saline contrast injection recorded in a patient presenting with pulmonary hypertension subsequently demonstrated to be related to an anomalous connection between the superior vena cava and right pulmonary vein. In the still image, note the nearly equivalent degree of contrast in the right ventricle and left ventricle related to right-to-left shunting. In the real-time image, note the nearly simultaneous appearance of contrast in the right and left atria. The inset is a continuous-wave Doppler from the four-chamber view revealing right ventricle to right atrial pressure gradient of 94 mm Hg consistent with severe pulmonary hypertension. [\textsuperscript{9}]
FIGURE 22.35. Apical four-chamber view recorded in a patient with severe pulmonary hypertension. Note the dilated right atrium and right ventricle and the mild tricuspid regurgitation. In the upper left is the continuous-wave Doppler through the tricuspid regurgitation jet from which a right ventricle to right atrial gradient of 70 mm Hg is calculated. With an assumed right atrial pressure of 15 mm Hg based on chamber dilation and appearance of the vena cava, right ventricular systolic pressure is calculated to be 85 mm Hg. 📹

Video 22-35
FIGURE 22.36. Mitral inflow and pulmonary vein flow recorded in a young female with primary pulmonary hypertension and no evidence of left ventricular disease. Note the reduced E/A ratio of 0.5 of the mitral inflow, and in the lower panel preservation of systolic forward flow from the pulmonary veins, all consistent with reduced inflow and effective underloading of the left ventricle rather than intrinsic diastolic dysfunction.

A number of effective therapies have been developed for the treatment of pulmonary hypertension. Serial echocardiography with Doppler interrogation of tricuspid regurgitation velocity can be used to follow the response to
therapy (Fig. 22.39) by monitoring not only tricuspid regurgitation jet velocity (for right ventricular systolic pressure) but also the degree to which left ventricular filling and function are compromised.

**FIGURE 22.37.** Apical four-chamber view recorded in a patient with exertional dyspnea and no apparent etiology. A was recorded at rest and reveals mild tricuspid regurgitation with a right ventricular to right atrial pressure gradient of 33 mm Hg consistent with borderline elevation of right ventricular systolic pressure. B was recorded immediately after 5 minutes of supine bicycle exercise and reveals dilation of the right atrium and right ventricle with moderate tricuspid regurgitation and an increase of the right ventricle to right atrial pressure gradient to 60 mm Hg consistent with exercise-induced pulmonary hypertension.

Video 22-37a
FIGURE 22.38. Illustration of the tricuspid annular plane systolic excursion (TAPSE). Upper panel: Recorded in a normal, disease-free individual in which TAPSE is measured as 16 mm. Lower panel: Recorded in a patient with long-
standing, severe pulmonary hypertension and right ventricular systolic dysfunction. Note the reduced TAPSE of 6 mm.

**MISCELLANEOUS DISEASES**

**Sarcoidosis**

Sarcoidosis is an inflammatory multisystem disease of uncertain etiology. Histologically, the hallmark is noncaseating granulomas in multiple organs. The predominant sites of involvement are the lungs and lymphatic system. The heart has been reported to be involved in up to 40% of advanced cases. Its actual prevalence has probably been underestimated. With more widespread use of CMR and cardiac PET scanning, an increasing number of patients, often presenting with arrhythmias, are being recognized. Cardiac involvement can include the pericardium, conducting system, or myocardium and result in either diffuse microscopic focal infiltrates or larger nodules within the myocardium (Fig. 22.40). Ventricular arrhythmias, including ventricular tachycardia, are common. Involvement predominates in the basal posterior and lateral walls and the proximal septum. Mitral regurgitation is often present. Focal wall motion abnormalities superficially may mimic those of ischemic disease but often occur in a location inconsistent with usual coronary anatomy (Figs. 22.41 and 22.42). Patients with cardiac sarcoidosis may also present with global left ventricular dysfunction, mimicking dilated cardiomyopathy (Fig. 22.43). Therapy for cardiac sarcoidosis includes high-dose steroid therapy and may result in improvement in global systolic function.
There are no specific echocardiographic findings in cardiac sarcoid, and other imaging modalities, such as contrast-enhanced cardiac magnetic resonance imaging (CMR), play a valuable diagnostic role. Cardiac MRI can
be instrumental in establishing the diagnosis of cardiac sarcoidosis. Findings with cardiac sarcoidosis are not infrequently incidentally encountered during a CMR examination being performed in a patient with unexplained arrhythmias. In addition to quantifying left ventricular systolic function and detecting regional wall motion abnormalities in a manner similar to that for echocardiography, gadolinium enhancement often provides valuable clues as to the specific diagnosis of cardiac sarcoidosis. The classic finding reported in cardiac sarcoidosis is an atypical location of late gadolinium enhancement. As opposed to ischemic heart disease where gadolinium enhancement reveals a fairly typical subendocardial or transmural pattern, in sarcoidosis there is often an atypical location of gadolinium enhancement including selective subepicardial enhancement. Myocardial enhancement is also noted as focal areas of enhancement which would not correspond to anticipated coronary anatomy (Fig. 22.44).

**FIGURE 22.40.** Parasternal long-axis (A) and short-axis (B) transthoracic echocardiogram recorded in a patient presenting with ventricular arrhythmias and subsequently documented to have cardiac sarcoidosis. In the parasternal long-axis view note the relatively normal chamber dimensions but the area of echo intensity in the mid anterior ventricular septum (arrows). In the short-axis view note the selective increase in echo intensity of the posterolateral papillary muscle (arrows). Cardiac MRI with gadolinium enhancement in this patient is presented in Figure 22.44 which reveals increased gadolinium enhancement in the areas of echo intensity.
Video 22-40a

Video 22-40b
FIGURE 22.41. Parasternal long-axis view recorded in a patient with documented cardiac sarcoidosis. In the parasternal long-axis view note the area of pathologic thinning in the proximal anterior septum (arrows). The two upper panels are cardiac magnetic resonance images from the same patient. In the panel at the upper left note the area of thinned proximal septum (arrow) corresponding to the area of thinning noted in the echocardiogram. At the upper right note the mid myocardial region of late gadolinium enhancement, characteristic of cardiac sarcoidosis (arrows). 

Video 22-41
Additional techniques which are used to establish the diagnosis of cardiac sarcoidosis include nuclear medicine isotope scans, typically using rubidium assessment of myocardial perfusion and fludeoxyglucose F18 (FDG) positron emission tomography for evaluation of metabolism. A typical pattern seen in patients with sarcoidosis is an area of reduced myocardial perfusion, often in a pattern inconsistent with typical coronary anatomy, corresponding to an area of increased metabolism suggesting an inflammatory response (Fig. 22.45).

**FIGURE 22.42.** Parasternal long-axis view recorded in a patient with documented cardiac sarcoidosis. Note the normal chamber sizes. This image was recorded at end-systole. Note the discrete posterior wall motion abnormality (arrows) inconsistent with the usual territory of an infarct related to coronary artery disease.
**Hemochromatosis**

Hemochromatosis involves the heart in most advanced cases and results in either an infiltrative pattern, similar to that seen with amyloidosis, or more commonly a dilated cardiomyopathy, which is indistinguishable from cardiomyopathy of other etiologies. The diagnosis should be suspected in patients with other manifestations of hemochromatosis such as diabetes and abnormal skin coloring who simultaneously present with a dilated cardiomyopathy. Figure 22.46 was recorded in a patient who had previously undergone cardiac transplantation for end-stage dilated cardiomyopathy due to hemochromatosis and subsequently developed biopsy-proven hemochromatosis in the transplanted heart. Note the thickened ventricular walls with abnormal myocardial texture.

**Tuberous Sclerosis**

Tuberous sclerosis is a heritable neurocutaneous disease presenting in childhood with developmental delay, seizures, and cutaneous abnormalities. Cardiac rhabdomyomas are commonly seen and present as multiple intracardiac tumors resembling cardiac myxoma (Fig. 22.47). Over time, they may regress leaving focal areas of endocardial fibrosis and scar (Fig. 22.48).
FIGURE 22.43. Apical four-chamber view recorded in a patient presenting with dilated cardiomyopathy and global left ventricular systolic dysfunction, subsequently proven to be related to cardiac sarcoidosis.

Video 22-43
Hypereosinophilia

Hypereosinophilia due to eosinophilic leukemia, tropical hypereosinophilia, or idiopathic eosinophilia result in characteristic echocardiographic abnormalities. The classic abnormality is obliteration of the left or right ventricular apex by laminar thrombus (Figs. 22.49 and 22.50). Pathologically, the thrombus is composed of inflammatory tissue, thrombus, and eosinophilic infiltrates. It results in a reduction of ventricular chamber size and increasing stiffness, resulting in a restrictive cardiomyopathic picture. Hypereosinophilic heart disease may also involve the posterior left ventricular wall and mitral apparatus and result in mitral regurgitation.

Carcinoid Syndrome

Carcinoid tumors release metabolites of serotonin and tryptophan that have a toxic effect on the cardiac endothelium (the carcinoid syndrome). These metabolites are deactivated in the lung, and, as such, left-sided involvement is rare unless there are concurrent pulmonary metastases or a right-to-left shunt. The classic abnormality in carcinoid syndrome is diffuse thickening and immobility of the tricuspid and pulmonary valves (Figs. 22.51 and 22.52). This results in a combination of stenosis and regurgitation. In advanced cases, the entire length of the tricuspid valve leaflet is thickened and rigid as opposed to a more domed appearance in rheumatic tricuspid valve disease. Rheumatic involvement can be distinguished from carcinoid syndrome because the vast majority of patients with rheumatic tricuspid valve disease will have concurrent mitral valve disease. See Chapter 12 for further discussion of carcinoid valve disease.
FIGURE 22.45. These images were recorded in the same patient presented in Figures 22.40 and 22.44. A: Myocardial perfusion scan using rubidium presented in long-axis view of the left ventricle. Note the relative absence of myocardial perfusion in the proximal anterior septum (double arrows) as well as the focal areas of absent perfusion in the inferoapical wall (single arrows). B is an FDG PET scan in the same patient in the same view as (A). Note the areas of hyperactivity (arrows) corresponding to areas of diminished perfusion, suggesting hypermetabolic activity in these areas.

FIGURE 22.46. Parasternal short-axis view recorded in a patient with documented cardiac hematochromatosis. Note the increased wall thickness and the abnormal myocardial texture with modest reduction in systolic function.
FIGURE 22.47. Off-axis parasternal view recorded in a young child with tuberous sclerosis and rhabdomyomas in the right ventricular outflow tract and left ventricle (arrows).
FIGURE 22.48. A is an apical four-chamber view in a young adult with a history
of tuberous sclerosis and prior documentation of multiple right and left ventricular rhabdomyomas. Over time, the tumors have spontaneously regressed, leaving bright, fibrotic, nodular areas on the left ventricular myocardium (arrows). B is a right ventricular inflow tract view revealing a similar nodular density in the right ventricle (arrow) at a location known to be a prior rhabdomyoma in childhood.
FIGURE 22.49. Composite imaging recorded in a patient with hypereosinophilia and involvement of the left ventricle. In the central image, note the four-chamber view with mild atrial dilation and the obliterative echoes within the apex of the left ventricle. At the upper right, note the cardiac computed tomographic images again revealing obliteration of the left ventricular apex by a soft tissue density (arrows). At the lower right is a cardiac magnetic resonance image of the same patient again revealing obliteration of the left ventricular apex by soft tissue density echoes with lesser involvement of the right ventricular apex. At the lower left is a positron emission tomographic image of the same patient revealing FDG-avid signals along the border of the mass in the left ventricle consistent with an inflammatory mass.
Sickle Cell Anemia

Sickle cell anemia (hemoglobin SS) has been associated with a number of cardiovascular abnormalities. It should be recognized that any severe, chronic anemia (including thalassemia) results in a high-output state, which in turn may lead to left ventricular dilation and, if severe and long standing, to the appearance of a dilated cardiomyopathy. Sickle cell anemia may also be associated with microinfarction and ventricular dysfunction (Fig. 22.53). Through a presumed similar thrombotic mechanism, patients may also develop pulmonary hypertension.

![Image of echocardiogram](image)

**FIGURE 22.50.** Apical four-chamber view recorded in a patient with milder apical involvement related to the hypereosinophilic syndrome. The *double-headed arrow* represents the actual myocardial thickness. Note the laminar thrombotic-appearing material at the apex (*upward-pointing arrows*) related to hypereosinophilic syndrome.
FIGURE 22.51. Apical four-chamber view recorded in mid systole in a patient with carcinoid syndrome and involvement of the tricuspid valve. Note the marked dilation of the right atrium and right ventricle and the diffusely thickened tricuspid valve leaflets (arrows) which are noted to be immobile in the real-time image.
Video 22-51
FIGURE 22.52. Right ventricular inflow tract views recorded in the same patient.
depicted in Figure 22.51. In A, note the marked dilation of the right atrium and right ventricle and the diffusely thickened immobile tricuspid leaflets (arrows). In B, note the wide open tricuspid orifice and “free” tricuspid regurgitation.

Video 22-52a

Video 22-52b
FIGURE 22.53. A is a parasternal long-axis view recorded in a young, African American male with sickle cell anemia and a chronic hemoglobin less than 8 g/dL. Note the dilated left ventricle and, in the real-time image, the global hypokinesis of the left ventricle. Incidental note is also made of a minimal pericardial effusion (arrow). B is an apical three-chamber view from which longitudinal strain is calculated. Note the reduction of strain in all segments, except the apex in this view and, in the bulls-eye plot, the reduction in strain globally with minor degrees of apical sparing.

Video 22-53

Human Immunodeficiency Virus
Infection with human immunodeficiency virus or acquired immunodeficiency syndrome is associated with a variety of cardiovascular manifestations, none of which are specific to the syndrome. Pericarditis, pulmonary hypertension,
and dilated cardiomyopathy have all been described. The mechanism by which the human immunodeficiency virus results in these manifestations is not fully understood. Because of their immunocompromised state, patients are prone to infections, including endocarditis with atypical organisms.

**Diet–Drug Valvulopathy**

In the late 1990s, a number of patients who had been exposed to anorexigens, especially the combination of fenfluramine and phentermine, developed an unusual form of valvular heart disease. Anatomically, the most obvious lesion was of the mitral valve. In advanced cases, the mitral valve and its chordae appeared encased in a matrix (Fig. 22.54), similar to that seen in the carcinoid syndrome; but without the tricuspid valve involvement. Aortic insufficiency was likewise noted; however, the echocardiographic appearance of the aortic valve was most often unremarkable. Initial reports suggesting an incidence of diet–drug valvulopathy of 16% to 40% were clearly erroneous. Better designed surveillance studies demonstrated an incidence between 3% and 15%, with the more prevalent lesion being aortic rather than mitral insufficiency. There was a definite relationship between duration of exposure to the drugs and the prevalence of valve disease. Most studies suggested that valve involvement was rare with less than 6 months of drug exposure and most valvular lesions were mild. There are no agreed on echocardiographic findings specific to this syndrome. Several follow-up studies have suggested that, in many patients, the valvular regurgitation may regress over time. Because the offending drugs are no longer used it is unlikely that one will encounter new cases of diet–drug valve disease. A similar form of valvulopathy had previously been reported in patients using ergotamine for treatment of migraine. More recently, a similar syndrome of valve disease has been reported in patients following pergolide therapy for Parkinson disease.

**Clinical Presentations and Problem Solving**

There are several common clinical presentations for which echocardiography plays a primary diagnostic role and has a direct and relevant impact on management (Table 22.2). For many of these presentations,
echocardiography carries a class I recommendation as a primary diagnostic tool in the respective American College of Cardiology/American Heart Association guidelines for management of that particular disease.

**FIGURE 22.54.** Apical long-axis view recorded in a patient with previous exposure to anorexigens and diffuse distal leaflet and chordal (arrows) thickening of the mitral valve. This appearance is not specific to diet–drug exposure, and the relationship to drug exposure is only presumptive and made in the absence of any other potential etiology for the leaflet thickening.
FIGURE 22.55. Apical four-chamber view recorded in a patient presenting with new-onset congestive heart failure symptoms related to a previously unrecognized dilated cardiomyopathy. Note the dilation of the left ventricle with the mildly abnormal spherical geometry and in the real-time image the global hypokinesis of the left ventricle.

Video 22-55

Congestive Heart Failure
CHF is one of the most common diagnoses encountered in contemporary practice and a leading cause of hospitalization. The anatomic and physiologic substrate underlying CHF is diverse and includes valvular heart disease, ischemic heart disease, and primary myocardial disease. Between 30% and 50% of patients presenting with CHF have preserved systolic function, and have heart failure based on diastolic dysfunction. This syndrome is referred to as heart failure with preserved ejection fraction (HFpEF). Indices of systolic function such as left ventricular diastolic and systolic volumes and ejection fraction can be determined with echocardiography and are instrumental in stratifying patients into preserved versus reduced systolic function categories (Figs. 22.55 and 22.56). Echocardiography can identify the underlying anatomic substrate in most patients presenting with CHF. Performance of echocardiography is considered a class I indication in the American College of Cardiology/American Heart Association guidelines for management of patients with new or recurrent CHF. In modern practice, all patients initially presenting with CHF, whether acute or chronic, should undergo echocardiography to determine the causative underlying anatomic substrate and to assess both systolic and diastolic function as well as to screen for secondary effects such as functional valve regurgitation and pulmonary hypertension.
FIGURE 22.56. Apical four-chamber view recorded an elderly patient presenting with congestive heart failure manifest as combined pulmonary congestion and peripheral edema. Note the marked enlargement of both the right and left atria but the relatively normal size of the ventricles. In the real-time image, note the preserved left ventricular systolic function. In the left hand panels note the moderate functional tricuspid regurgitation but the relatively normal right ventricular to right atrial pressure gradient in the Doppler tracing. In the panels on the right note the steep deceleration time of mitral inflow in the pathologically reduced E wave velocities suggestive of grade 3 diastolic dysfunction in this patient with restrictive physiology related to long-standing hypertension and age.
On the basis of echocardiographic findings, heart failure can be stratified into diseases requiring surgical management such as valvular heart disease and those requiring medical management such as dilated cardiomyopathy. The complete evaluation of patients with CHF typically can be performed with transthoracic echocardiography. Stress echocardiography can play an
incremental role in identifying an ischemic substrate and viable myocardium in patients with chronic systolic dysfunction. For patients with primary myocardial disease, serial echocardiography can be used to evaluate recovery of function with therapy and screen for complications of heart failure.

There are several echocardiographic features to be noted in patients with heart failure that have prognostic relevance (Table 22.5). There is an inverse relationship between left ventricular systolic function and clinical outcome. Additional features to be noted in patients with CHF include the presence of concurrent mitral or tricuspid regurgitation, right ventricular dysfunction, or secondary pulmonary hypertension, each of which confers a worse prognosis in patients with CHF.

Evaluation of diastolic properties of the heart using Doppler echocardiography also provides important prognostic information. Patients with a high E/A ratio and short deceleration time (the restrictive pattern) have a worse prognosis compared with those with a pattern of delayed relaxation and an E/A ratio less than 1.0. In the setting of systolic dysfunction, the exaggerated E/A ratio represents pathologic stiffening of the ventricle with concurrent elevation of left ventricular diastolic pressures. It generally implies a combination of volume overload and diastolic dysfunction. Data also suggest that the intermediate pattern of pseudonormalization confers a similar prognosis. Evaluation of pulmonary vein flow and Doppler tissue imaging of the mitral annulus can assist in identifying patients with the pseudonormal pattern and hence an adverse prognosis. Multiple studies have confirmed the adverse prognosis associated with diastolic dysfunction as evaluated with Doppler tissue imaging for analysis of mitral annular velocity. In addition, the adverse prognosis associated with left atrial dilation has been well established.

**Acute Pulmonary Embolus**

Acute pulmonary embolus occurs both on a background of major medical illnesses and in previously healthy individuals with a precipitating risk factor such as immobilization. The classic symptoms of a pulmonary embolus include acute pleuritic chest pain and breathlessness. Patients with a pulmonary embolus on the background of another major illness often have atypical presentations or may not be acutely symptomatic. The degree of
hemodynamic compromise is directly related to the embolic burden and ranges from trivial and inconsequential to instantaneously fatal events seen with large or multiple pulmonary emboli. For patients presenting with the acute onset of dyspnea, echocardiography can be a helpful tool, but a normal echocardiogram should not be used to exclude the presence of a pulmonary embolus in a patient whose symptoms otherwise warrant evaluation of that entity.

**Echocardiographic Findings**

Echocardiographic findings in a pulmonary embolus are directly related to the magnitude of the embolus. The degree to which there has been pre-existing cardiovascular disease must also be factored into this analysis. With large hemodynamically significant pulmonary emboli, right heart dilation and systolic dysfunction occur (Fig. 22.57). Assuming a previously normal cardiovascular system with normal pulmonary artery pressures, the right ventricle is not conditioned to generate pressures in excess of 60 to 70 mm Hg. Therefore, if one encounters pressures of 70 mm Hg or more in a suspected pulmonary embolus, the scenario of acute on chronic thromboembolic disease or a pulmonary embolus superimposed on previously existing pulmonary hypertension must be considered. For a patient presenting with acute breathlessness and chest pain, and in whom right ventricular dilation with tricuspid regurgitation and mild elevation of the pulmonary artery pressure is noted, a pulmonary embolus should be one of the initial diagnoses to be considered. Evaluation of left ventricular function is obviously crucial because inferior infarct, complicated by right ventricular infarction, may have a similar echocardiographic appearance but would not be expected to be seen in conjunction with elevated pulmonary artery pressure. In many patients, with pulmonary emboli, only mild right heart dilation and tricuspid regurgitation may be noted and result in subtle nonspecific abnormalities of ventricular septal motion. For small pulmonary emboli, it is not uncommon to see an entirely normal echocardiogram, thus a normal echocardiogram should not be used to exclude the diagnosis of acute pulmonary embolus. Depending on the size of the embolus and magnitude of resultant right ventricular dysfunction, right-sided cardiac output may be compromised and reduce left ventricular filling. This may result in a reduced
E/A ratio of mitral filling but is obviously a nonspecific finding. An echocardiographic sign that has been considered specific for acute pulmonary embolus is preservation of motion of the apical right ventricular segments with basal wall motion abnormalities (McConnell sign) (Fig. 22.58).

**FIGURE 22.57.** Subcostal image recorded in a patient with an acute, large pulmonary embolus. Note the dilation of the right ventricle and in the real-time image, the hypokinesis of the proximal two-thirds of the right ventricular wall. The lower right image was recorded from an apical four-chamber view and reveals mild tricuspid regurgitation with a peak velocity of 3 m/s consistent with an estimated right ventricular systolic pressure of 46 mm Hg (assuming a right atrial pressure of 10 mm Hg).
FIGURE 22.58. Subcostal image recorded in a patient with a massive pulmonary embolus demonstrating dilation of the right ventricle. The presented frame is in end-systole. Note the preserved contractility of the apical segment of the right ventricle with malfunction of the more basal portions (arrows) (McConnell sign).

Video 22-58

On rare occasions, one can directly visualize a pulmonary embolus in a proximal pulmonary artery (Fig. 22.59). This may best be accomplished with transesophageal echocardiography (Fig. 22.60), which is not usually
performed for the routine evaluation of a suspected pulmonary embolus. On occasion, one identifies thromboembolism in transit (TEIT), which represents a large thrombus, typically from a deep venous structure in the lower extremities that has become entangled in the tricuspid valve apparatus. This thrombus takes on a serpiginous and highly mobile appearance on echocardiography and appears to curl on itself. Figures 22.61 and 22.62 were recorded in patients with TEIT. Notice in Figure 22.62 that a portion of the thrombus has protruded through a PFO into the left atrium, hence placing the patient at risk of paradoxical systemic embolization. Treatment of TEIT remains controversial, with most authorities arguing for immediate surgical removal of the thrombus in appropriate candidates and others recommending either lytic therapy or aggressive heparinization. Detection of TEIT represents a high-risk subset of patients with mortality often exceeding 75% if not treated.

FIGURE 22.59. Parasternal short-axis view recorded in a patient with an acute pulmonary embolus. Note the tubular mass at the bifurcation of the pulmonary
artery (small arrows). In the real-time image, note the mobile nature of this mass, which has the classic appearance of a “saddle” embolus. LPA, left pulmonary artery; RPA, right pulmonary artery.

Video 22-59

FIGURE 22.60. Transesophageal echocardiogram recorded in a patient with large proximal pulmonary embolus. In A, visualized at the level of the ascending aorta and right pulmonary artery, note the homogeneous mass filling the right pulmonary artery (RPA). In B, recorded with color Doppler, note the marked obstruction to flow by the thrombotic mass.
FIGURE 22.61. Transthoracic right ventricular inflow tract view recorded in a patient presenting with acute dyspnea due to pulmonary emboli. In the central figure, note the elongated, tubular mass spanning the right atrium and right ventricle (arrows). The two figures in the insets are views of the right atrium recorded from two additional time points for two additional cardiac cycles, revealing the highly variable location and appearance of the serpiginous mass within the right heart. This is better appreciated in the accompanying real-time image.
In patients with suspected pulmonary embolus, attention should be paid to curvature of the interatrial septum. If there is elevation of right heart pressure, the interatrial septum will bow persistently from right to left, rather than having normal phasic variation in both directions (Fig. 22.63). Detection of right-to-left shunting through a PFO with saline contrast echocardiography is circumstantial evidence that right heart pressures are elevated. Several echocardiographic features have been associated with a worsened prognosis in patients with an acute pulmonary embolus and have been suggested as an indication for aggressive therapy with lytic or catheter based therapy. These include evidence of significant right heart dilation and right ventricular systolic dysfunction. Other echocardiographic parameters, which provide prognostic information in acute pulmonary embolus, include the myocardial performance index and tricuspid annular plane systolic excursion (TAPSE).
FIGURE 22.62. Transesophageal echocardiogram recorded in a patient with thromboembolism in transit (TEIT). Note the complex serpiginous mass present within both the right and left atrium (arrows). Because of the serpiginous nature of the mass, which curls on itself, the left atrial component appears as two, small, discrete masses. In the real-time image, the continuity of all components of the thromboembolism in transit can be better appreciated. The inset is the surgeon’s sketch of the findings at the time of surgical excision outlining the elongated thrombus present in the right atrium and traversing the atrial septum into the left atrium.
Atrial Fibrillation

Atrial fibrillation is present in 6% to 10% of patients older than 70 years. It may be encountered in the presence of a structurally normal heart (lone atrial fibrillation) or more commonly in association with underlying cardiovascular disease. There are several classic cardiovascular diseases associated with atrial fibrillation, most notably rheumatic mitral stenosis. However, hypertension and underlying coronary disease are currently the most common risk factors for development of atrial fibrillation. On the basis of clinical and echocardiographic criteria, patients with atrial fibrillation are classified as having valvular versus nonvalvular atrial fibrillation. Echocardiography should be performed in all patients with atrial fibrillation. Detection of a structurally normal heart identifies a subset of patients more likely to have spontaneous conversion to sinus rhythm and, when combined with a relatively young age and absence of clinical risk factors, identifies a subset at relatively low risk of embolic complications. Conversely, detection of previously unsuspected cardiomyopathy or mitral stenosis implies a lower likelihood of spontaneous restoration of sinus rhythm and an increased likelihood of cardioembolic complications. Guidelines for long-term
anticoagulation in patients with chronic atrial fibrillation are based in large part on patient’s age, concurrent hypertension, diabetes, heart failure, and evidence of underlying structural heart disease, which can be easily assessed with transthoracic echocardiography.

Symptoms of atrial fibrillation are highly variable and may be confined to a sensation of palpitations with a rapid, irregular heart rate. More worrisome is development of exercise intolerance and dyspnea, which may relate to CHF, either related to the unmasking of pre-existing disease or development of a rate-related cardiomyopathy. The latter phenomenon is well known to occur in patients with atrial fibrillation with an uncontrolled ventricular response. In this situation, patients may present with an echocardiogram consistent with a dilated cardiomyopathy (Fig. 22.64). Assuming the duration of uncontrolled atrial fibrillation is modest, chamber dilation typically is less impressive than is the systolic dysfunction. There is a high likelihood of recovery of function unless there is concurrent underlying cardiomyopathy (Fig. 22.65).

Probably the most concerning complication of atrial fibrillation is thromboembolism including stroke, which, prior to the advent of modern anticoagulation strategies, caused substantial morbidity and mortality in patients with chronic atrial fibrillation. In patients with atrial fibrillation, a distinction should be made between those with valvular atrial fibrillation and those with nonvalvular atrial fibrillation. Current guidelines define “valvular atrial fibrillation” as that seen in the presence of rheumatic mitral stenosis, or a repaired or replaced mitral valve. The risk of thromboembolic complications is higher and management strategies are different for those with valvular versus nonvalvular atrial fibrillation. This distinction can be made with confidence on the basis of an echocardiogram.
FIGURE 22.64. Transthoracic echocardiograms recorded in a patient presenting with newly recognized atrial fibrillation and symptoms of fatigue and dyspnea. A is an apical four-chamber view revealing modest dilation of the left ventricle and global hypokinesis which can be appreciated in the real-time image. B is an apical four-chamber view, again revealing global hypokinesis of the left ventricle and moderate to severe functional mitral regurgitation.
FIGURE 22.65. Apical four-chamber view recorded in the same patient presented in Figure 22.64. These images were recorded 3 months following cardioversion to normal sinus rhythm. A is an apical four-chamber view confirming a decrease in left ventricular size and normalization of left ventricular systolic function, better appreciated in the real-time image. B is an apical four-chamber view with color Doppler confirming resolution of the secondary mitral regurgitation coincident with improvement in left ventricular systolic function. 

coming soon

Video 22-65a
Thromboembolism occurs in patients with atrial fibrillation because of stasis of blood in the left atrium leading to thrombus formation (Figs. 22.66 and 22.67). More than 90% of thrombi forming in the presence of atrial fibrillation will be located in the left atrial appendage. The prevalence of thrombus has ranged from 6% to 30% in patients with atrial fibrillation. The likelihood of finding thrombus is related to the etiology of the underlying cardiac disease and the duration of atrial fibrillation, which explains the broad range in prevalence. It should be emphasized that detection of dense “smoke” or spontaneous contrast (Figs. 22.68 and 22.69) in the left atrium or left atrial appendage may be associated with a similar risk of thromboembolic events.
FIGURE 22.66. Transesophageal echocardiogram recorded in a patient with paroxysmal atrial fibrillation and a neurologic event. The small black arrows depict the outer border of the left atrial appendage, which is completely filled with thrombus including a smaller component protruding into a side lobe (white arrows).
Both thrombus and stasis are directly related to the integrity of atrial transport, which can be assessed by a number of echocardiographic parameters. The simplest is to assess the entrance and exit velocities of blood from the left atrial appendage by placing a pulsed Doppler sample volume at the mouth of the atrial appendage (Fig. 22.70). In patients with atrial fibrillation, there is tremendous variability in the atrial entrance and exit velocities. Many patients with atrial fibrillation, especially if there is no cardiomyopathy or other significant structural heart disease, have exit velocities equivalent to that seen in patients in sinus rhythm. There is indirect evidence that preservation of exit velocities reduces the risk of stasis and clot formation. Conversely, other patients with atrial fibrillation may have pathologically low velocities (lower panels in Fig. 22.70), a finding that has been correlated with a greater likelihood of spontaneous echo contrast and thrombus formation. Other methods for assessing atrial appendage transport include Doppler tissue imaging of the appendage wall as well as planimetry of the appendage area for calculation of “ejection fraction.”

Considerable research has focused on the potential role of transesophageal echocardiography in guiding the management of patients with atrial fibrillation. Conventional therapy involves 3 to 4 weeks of oral anticoagulation before cardioversion, followed by 3 to 6 months of anticoagulation after restoration of sinus rhythm. The institution of 3 to 4 weeks of anticoagulation before cardioversion will reduce the likelihood of thromboembolism substantially and has been considered standard of care. It has been postulated that, in the absence of echocardiographic evidence of left atrial thrombus, elective cardioversion can proceed with a low embolic risk (provided that patients are adequately anticoagulated at the time of the procedure and anticoagulation maintained for several weeks afterward). This strategy shortens the duration of atrial fibrillation and presumably promotes more rapid recovery of mechanical atrial function (i.e., reduced left atrial appendage stunning).
FIGURE 22.67. Transesophageal echocardiogram recorded in a patient with atrial fibrillation presenting for cardioversion. Note the large thrombus burden within the left atrial appendage including two, prominent, distinct thrombi (arrows). The inset was a repeat transesophageal echocardiogram recorded after 8 weeks of antithrombotic therapy revealing resolution of the previously noted atrial appendage thrombi. [Video]
FIGURE 22.68. Expanded view of the left atrial appendage in a patient with atrial fibrillation. In this example, there is no distinct thrombus but vague swirling smoke-like echoes suggesting stagnant blood. [Video coming soon]
Both the conventional approach and the TEE-guided approach are clinically reasonable. The decision as to which strategy to employ is clinically based and often related to the need to restore sinus rhythm rapidly and/or the perceived risk of the additional 3 to 4 weeks of precardioversion anticoagulation. For either approach, postcardioversion anticoagulation is required for a minimum of 6 weeks, and many authorities recommend a longer duration depending on the clinical scenario.

**FIGURE 22.69.** Transesophageal echocardiogram recorded in a patient with atrial fibrillation with dense spontaneous contrast in the left atrium and left atrial appendage. The upper inset is a pulsed-wave Doppler from the left atrial appendage revealing markedly diminished left atrial appendage entrance and exit velocities of approximately 20 cm/s.
The need for postcardioversion anticoagulation is related to atrial stunning. Following conversion to normal sinus rhythm, spontaneously, pharmacologically, or by direct electrical cardioversion, a phenomenon of atrial stunning may occur. This phenomenon results in an abrupt decrease in atrial appendage function immediately following restitution of normal sinus rhythm and increases stasis in the left atrial appendage and hence the likelihood for thrombus formation. Historically, it was recognized that the likelihood of a thromboembolic complication following cardioversion occurs not instantaneously, but within the ensuing 72 hours. This probably relates to the stunning with delayed thrombus formation and embolization rather than “ejection” of a pre-existing thrombus. Figures 22.71 and 22.72 were recorded in a patient during elective cardioversion from atrial fibrillation. In Figure 22.71, note the near normal atrial appendage entrance and exit velocities while in atrial fibrillation and the abrupt decrease in atrial transport function immediately noted after restoration of sinus rhythm. This is paralleled by the appearance of spontaneous echo contrast immediately following electrical cardioversion as noted in Figure 22.72. Serial echocardiography has demonstrated that several weeks may be required for recovery of atrial mechanical activity. The time over which the propensity to form thrombus diminishes after restoration of sinus rhythm is not well established.
FIGURE 22.71. Atrial appendage velocities recorded in a patient before (upper panel) and after (lower panel) cardioversion of atrial fibrillation. Prior to cardioversion: Note the entrance and exit velocities of 40 to 60 cm/s while in atrial fibrillation and the reduction in velocities to approximately 30 cm/s after restitution of normal sinus rhythm (lower panel).
FIGURE 22.72. Transesophageal echocardiogram recorded before and immediately after cardioversion from atrial fibrillation. A: Note the normal size of the left atrial appendage and the absence of any clot or spontaneous echo contrast. B: Recorded shortly after electrical cardioversion to normal sinus rhythm (arrow) and reveals spontaneous echo contrast in the atrial appendage related to
atrial appendage stunning.

FIGURE 22.73. Expanded view of the left atrial appendage recorded in a patient with a dilated cardiomyopathy being investigated for cardiac source of embolus. The patient is in normal sinus rhythm at the time of the examination and had no history of atrial fibrillation. Notice the dilation of the left atrial appendage, as well as the spontaneous echo contrast within the appendage. The inset is Doppler velocities recorded within the left atrial appendage and confirms normal sinus rhythm but pathologically reduced entrance and exit velocities coincident with the spontaneous echo contrast while in normal sinus rhythm.
On occasion, a similar appearance of reduced atrial transport with spontaneous echo contrast and even thrombus in the atrial appendage can be noted in patients in normal sinus rhythm. In the majority of instances, there is either concurrent dilated or restrictive cardiomyopathy with marked atrial dilation and evidence of diastolic dysfunction. Many of these patients may have paroxysmal atrial fibrillation, and hence, the spontaneous echo contrast and thrombus formation may be more related to that entity. Figure 22.73 was recorded in a patient with hypertensive cardiovascular disease with diastolic dysfunction who was in normal sinus rhythm and not known to have a history of atrial fibrillation. The appearance of spontaneous contrast is identical to that in atrial fibrillation, and Doppler flow velocities within the atrial appendage are pathologically reduced.

Multiple echocardiographic parameters have been evaluated as potential markers for success of either cardioversion or ablation for restoring sinus rhythm. These have included assessment of left atrial size and volume as well as the underlying substrate and left ventricular systolic function. More recently, global strain of the left atrial wall has shown promise for being predictive for success of procedures designed to maintain sinus rhythm (Fig. 22.74). At this time, none of the techniques reliably predict long-term success of maintaining sinus rhythm as to be used independently in clinical decision making.
FIGURE 22.74. Recording of left atrial strain measurement. Note the area of interest in the left atrial wall and the accompanying plot of longitudinal strain in the left atrial myocardium.
FIGURE 22.75. Apical four-chamber view recorded in an elderly female with classic Takotsubo syndrome occurring after marked situational stress. Notice the apical ballooning (arrows) and the preserved function at the base of the heart. The inset is a full-volume, three-dimensional image of the left ventricle. Notice the apical ballooning and hyperdynamic motion at the base of the heart when compared to the “wire frame” contour of the left ventricle in diastole. Video 22-75 3D

coming soon

Video 22-75 3D
Takotsubo Syndrome
The Takotsubo syndrome is a stress-related phenomenon typically encountered in older, female patients after some form of severe emotional stress, such as an adverse medical diagnosis, death of a spouse, etc. The range of emotional stress provoking the Takotsubo syndrome is highly variable. Other names for this syndrome have included the “broken heart syndrome,” “stress cardiomyopathy,” and “apical ballooning syndrome.” Clinically, the patients present with abrupt onset of chest pain which may be accompanied by dyspnea and evidence of CHF. Ventricular arrhythmias have been reported but are rare. The 12-lead electrocardiogram classically reveals deep symmetric T-wave inversion in the anterior precordium, although many patients have more subtle and more nonspecific ST- and T-wave changes. Enzyme elevation is generally mild and well below what would be anticipated on the basis of the wall motion abnormalities.
FIGURE 22.76. Apical five-chamber view recorded in a patient with Takotsubo syndrome presenting with congestive heart failure and typical ECG and enzyme patterns. The patient is known to be free of coronary artery disease. A was recorded in end-systole. Note the hyperdynamic motion at the base of the heart with apical ballooning and the systolic anterior motion of the mitral valve (arrow). B is the color Doppler image recorded in the same patient revealing both turbulence in the left ventricular outflow tract, as well as mitral regurgitation (arrows) which is related to systolic anterior motion and which resolved with resolution of the syndrome.
FIGURE 22.77. Apical long-axis view in a patient with classic Takotsubo syndrome. The image was recorded at end-systole and demonstrates systolic anterior motion of the mitral valve related to the hyperdynamic motion at the base. The inset is the continuous-wave Doppler through the left ventricular outflow tract revealing a classic, late-peaking dynamic outflow tract gradient of 69 mm Hg. Both the wall motion abnormalities and dynamic outflow tract obstruction resolved with resolution of the syndrome.
With echocardiographic imaging, the classic Takotsubo syndrome reveals marked dilation and ballooning of the left ventricular apical segments with preserved or hyperdynamic contractility in the basal segments. In systole, this results in a lightbulb-shaped left ventricle which resembles the Japanese octopus trap, hence the name “Takotsubo.” Because of the apical ballooning and hyperdynamic basal segments, dynamic outflow tract obstruction can occur, as can dynamic mitral regurgitation. The syndrome typically resolves in as short as 3 days, although more prolonged recovery times of up to 6 weeks or more have also been reported. By definition, the patients are free of obstructive coronary artery disease. Figures 22.75 to 22.79 illustrate the range of abnormalities seen in the Takotsubo syndrome, as well as examples of induced and transient mitral regurgitation and/or dynamic outflow tract obstruction.

While the classic Takotsubo syndrome involves the apical segments of the left ventricle, less often encountered variants include isolated posterior or lateral wall motion abnormalities or a reverse Takotsubo in which the apex is preserved and more basal segments are involved. Imaging with cardiac MRI has documented similar wall motion abnormalities, as well as evidence of transient myocardial edema.
Neurogenic Myocardial Stunning
Following an acute neurologic event, classically an intracerebral hemorrhage,
a phenomenon of myocardial neurogenic stunning may occur. The syndrome is characterized by deep symmetrical T-wave inversion in the anterior precordial leads of the electrocardiogram. On echocardiography, these patients have marked apical dyskinesis and dilation, mimicking ischemia or infarction in the left anterior descending coronary artery territory. Typically, elevation of cardiac enzymes is minimal and the wall motion abnormality typically reverts to normal over a 3- to 14-day period. The pathophysiology of this phenomenon is not fully known but appears related to autonomic discharge with catecholamine “surge.” The findings are virtually identical to the abnormalities reported with the apical ballooning or Takotsubo syndrome.

FIGURE 22.79. Apical four-chamber view recorded in a patient with Takotsubo syndrome involving both the left and right ventricles. Note the hyperdynamic motion of the left ventricle at the base (inward-pointing arrows) and the obvious apical ballooning. Also note the hyperdynamic motion of the proximal right ventricular wall (black arrow) and the akinesis and dilation of the more distal right ventricular segments.
**Syncope**

Evaluation of patients with syncope is often problematic, and the incremental yield and overall utility of echocardiographic screening of otherwise healthy individuals with a single episode of syncope is low. There are obvious cardiovascular diseases that can result in syncope such as critical aortic stenosis, hypertrophic cardiomyopathy, and other cardiovascular diseases associated with arrhythmia such as dilated cardiomyopathy. The yield of echocardiographic screening for detection of these abnormalities in a patient with a normal physical examination and a normal resting 12-lead electrocardiogram is relatively low. Current guidelines do recommend echocardiography in patients with or without clinical evidence of structural heart disease who have experienced syncope.

**Cardio-Oncology**

Many chemotherapeutic agents are associated with cardiotoxicity. The most widely appreciated are the anthracycline class of agents typified by doxorubicin and more recent breast cancer drugs, such as trastuzumab (Herceptin). Doxorubicin toxicity results in left ventricular systolic dysfunction, indistinguishable from that seen in cardiomyopathy of other etiologies. There is a less well recognized acute and transient decrease in left ventricular systolic function that is occasionally seen at the time of acute infusion and does not necessarily imply long-term systolic dysfunction. Patients at risk of having pre-existing cardiovascular disease should undergo
surveillance echocardiography to ensure normal left ventricular systolic function before institution of chemotherapy, and those receiving potentially cardiotoxic agents all require baseline and serial assessments of left ventricular function. If, during the course of chemotherapy, a patient develops symptoms suggestive of CHF, repeat echocardiography is clinically indicated to reassess left ventricular function. There are no specific echocardiographic markers that allow identification of patients likely to develop chemotherapy-related cardiotoxicity. Chemotherapy agents other than anthracyclines can also result in acute cardiac decompensation, including high-dose cyclophosphamide (Cytoxan). The frequency with which this occurs is substantially less than that with doxorubicin and the dysfunction is usually transient.

Cardiotoxicity from chemotherapy manifests as reduced systolic function and the ensuing cardiomyopathy is indistinguishable from other forms of nonischemic cardiomyopathy (Figs. 22.80 and 22.81). In advanced forms, multichamber dilation and a globally hypokinetic left ventricle with reduced systolic function are seen. Obviously, identification of patients prior to development of overt cardiomyopathy would have a significant impact on management. Multiple parameters have been proposed as markers of early subclinical chemotherapy toxicity. At this time, the parameter showing the most promise, and which is used on a regular basis in the majority of centers where chemotherapy cardiotoxicity monitoring is undertaken, is global longitudinal strain (GLS). Several studies have demonstrated that GLS declines in advance of any detectable reduction in left ventricular systolic function and that this decline is predictive of subsequent development of cardiomyopathy or CHF. Because of the fairly broad range of normal values for GLS, it is imperative that each patient serves as his/her own control and that a high-quality baseline echocardiogram including determination of GLS is obtained prior to institution of chemotherapy. Depending on the chemotherapeutic agent, monitoring is conducted every 1 to 3 months and continued until completion of therapy. Thresholds at which one should have concern for occult chemotherapy-induced myopathy have not been uniformly established, however, a reduction in GLS by 25% appears to be a threshold at which concern should be raised. After identification of subclinical myocardial depression in patients receiving chemotherapy, a decision to continue, alter, or discontinue the chemotherapy regimen needs to be made based on overall
patient-related features and not exclusively on the basis of the echocardiographic findings.

**FIGURE 22.80.** Apical four-chamber view recorded in a patient receiving chemotherapy. In this apical four-chamber view recorded prior to institution of chemotherapy, note the mild mitral regurgitation and in the real-time image the normal left ventricular systolic function. The upper left inset is the plot of global longitudinal strain which was normal at −22.6%. The lower right image is the continuous-wave Doppler of the mitral regurgitation jet, from which an LV $dP/dt$ of 2,424 mm Hg/s is calculated. The upper right inset is the lateral Doppler tissue profile revealing a systolic velocity of approximately 10 cm/s.
FIGURE 22.81. Imaging recorded in the same patient presented in Figure 22.80 6 months after institution of chemotherapy. In the apical four-chamber view note the now moderate mitral regurgitation and in the real-time image the visually normal left ventricular systolic function. Global longitudinal strain has declined to −17% and LV dP/dt to 932 mm Hg/s. The lateral Doppler tissue systolic velocity is now under 8 cm/s. 🎥
Radiation-Induced Cardiac Disease

Mediastinal radiation is associated with both acute and chronic cardiac pathology. Fortunately, modern techniques of radiation therapy have resulted in more precise targeting, which has reduced the magnitude of this problem. The most common early manifestation of radiation-induced cardiac disease is pericarditis. It may be associated with transient constrictive physiology. It has the characteristics of other forms of inflammatory pericarditis (Fig. 22.82). The time course for resolution of this form of pericarditis may be months. An obvious clinical dilemma when one encounters a new pericardial effusion in a patient who has undergone radiation therapy for malignancy is whether the effusion is related to the malignancy or the radiation therapy. This distinction needs to be made on clinical grounds.

Radiation therapy also affects the heart in a delayed manner and patients may develop manifestations 3 to 15 years after mediastinal radiation. Delayed presentation includes chronic constrictive or effusive constrictive pericarditis, myocardial disease, and valvular abnormalities. Assuming an anterior portal, the right ventricle may be disproportionately affected and may result in the appearance of a restrictive cardiomyopathy. Valvular disease most often involves the aortic valve and anterior mitral valve leaflet (Figs. 22.83 and 22.84). Radiation damage results in a fairly characteristic appearance of the anterior mitral valve leaflet. The proximal portion of the anterior leaflet is selectively thickened and rigid with sparing of the distal aspect. The likelihood of valvular damage from radiation is dose dependent, and there is
usually a 3- to 5-year delay in its appearance. The earliest lesion is aortic regurgitation with mitral and aortic stenoses being a later finding.

**FIGURE 22.82.** Parasternal long-axis view recorded in a patient with esophageal cancer, following radiation therapy. Note the anterior PEF and the nodular densities in the interventricular groove (arrows).
coming soon

Video 22-82
FIGURE 22.83. Parasternal long-axis views recorded in two patients 15 and 20 years following mantle radiation for Hodgkin lymphoma. A: Note the thickening of
the aortic valve and the prominent thickening and rigidity of the proximal half of the anterior mitral leaflet (main illustration and upper left inset). B: Note the pleural effusion with atelectatic lung (arrow) and the apparent thickening of the pericardium (small white arrows). The mitral inflow pattern reveals an E/A ratio of 2.0 with a short deceleration time suggesting either a constrictive or restrictive process. In the real-time image, note the abrupt relaxation pattern of the posterior wall. PI, pleural effusion.
FIGURE 22.84. Parasternal long-axis images recorded in a patient following mantle radiation for Hodgkin disease 20 years prior to this presentation. In A, recorded without color Doppler note the thickening of the aortic valve and the selective "boardlike" thickening of the anterior mitral valve leaflet. Mitral annular calcification and thickening of the posterior mitral leaflet are also noted. In B, recorded with color Doppler note the mild to moderate aortic insufficiency associated with an elevated gradient of approximately 50 mm Hg as noted by the continuous-wave Doppler in the upper panel. Video 22-84a
Screening for Athletic Competition and the Athlete’s Heart

Before competitive athletic activity, potential participants often undergo a general health evaluation. From a standpoint of cardiovascular disease, this generally consists only of recording blood pressure, heart rate, and auscultation of the heart. In an asymptomatic individual with a normal cardiovascular physical examination, and no family history of heritable cardiovascular disorders, the likelihood of finding significant underlying cardiovascular disease that would adversely affect the suitability for competitive sports is low. In this setting, routine evaluation with echocardiography has not been shown to be cost effective and its routine use remains controversial. Individuals for whom an echocardiogram may be indicated include those with a family history of exertional syncope or sudden cardiac death and those who have symptoms. Table 22.6 lists a number of cardiovascular abnormalities that have relevance for competitive sports. Many, such as aortic stenosis, should be detected on physical examination. The combination of a thorough physical examination and a 12-lead electrocardiogram generally suffices for detection of most relevant abnormalities. In individuals in whom surveillance echocardiography is indicated before participation in competitive sports, the examination should be tailored to exclude disease of the proximal aorta that would predispose to dissection or rupture, hypertrophic cardiomyopathy, and occult valvular heart disease. If possible, the origin of both coronary arteries should be identified.
because anomalous origin of a coronary artery has been associated with sudden cardiac death at the time of physical exertion. This rare anomaly obviously will not be detected by a history, physical examination, or a 12-lead electrocardiogram. At this time screening echocardiography is not recommended in the general population in the absence of symptoms, abnormalities on physical examination, or a relevant family history. Its overall prevalence in the population is too low to warrant routine echocardiographic screening solely for that purpose prior to athletic participation.

**Table 22.6**  
ATHLETIC SCREENING: RELEVANT ABNORMALITIES

<table>
<thead>
<tr>
<th>CONFERRING INCREASED RISK FOR PARTICIPATION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Moderate and high risk</strong></td>
</tr>
<tr>
<td>Marfan syndrome</td>
</tr>
<tr>
<td>Other aortic dilation</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Occult-dilated cardiomyopathy</td>
</tr>
<tr>
<td>Valvular aortic stenosis (moderate or worse)</td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
</tr>
<tr>
<td>Anomalous coronary artery origin</td>
</tr>
<tr>
<td><strong>Low risk</strong></td>
</tr>
<tr>
<td>Mitral valve prolapse with ≤ mild regurgitation</td>
</tr>
<tr>
<td>Bicuspid aortic valve with gradient ≤25 mm Hg (peak)</td>
</tr>
<tr>
<td>Mild mitral stenosis (New York Heart Association Class I)</td>
</tr>
<tr>
<td>Uncomplicated atrial septal defect</td>
</tr>
<tr>
<td>Mild pulmonary stenosis</td>
</tr>
<tr>
<td>Small, restrictive ventricular septal defect</td>
</tr>
</tbody>
</table>

Vigorous athletic training results in compensatory changes in cardiac anatomy. The degree of athletic training required to result in the “athlete’s heart” is substantial, and changes are not seen in casual, recreational athletes. The type of athletic activity has an impact on the nature of left ventricular remodeling. Vigorous endurance training such as long-distance running, Nordic skiing or cycling results in mild ventricular hypertrophy with an elevation in left ventricular mass due to chamber enlargement and, to a lesser degree, in wall thickness (Fig. 22.85). Right atrial and ventricular enlargement is also noted. The bradycardia associated with the athletically
conditioned heart is often associated with the appearance of mild visual “global” hypokinesis. It should be recognized that chamber enlargement allows preservation of stroke volume. While resting ejection fraction may be slightly below normal, calculated stroke volume and, hence, cardiac output remains normal. Conversely, intense isotonic training (weight lifting, wrestling, etc.) results in more concentric hypertrophy. Table 22.7 outlines the anticipated changes in left ventricular wall thickness, internal dimension, and mass for different types of highly trained athletes. An additional factor to consider is that most modern athletes train with a combination of resistance and endurance exercise, and, as such, the “pure” categories of athletic heart anatomy are relatively uncommon. Wall thickness rarely exceeds 13 mm in the “athlete’s heart,” and values progressively over 13 mm should raise the consideration of a hypertrophic cardiomyopathy. Additionally, diastolic dysfunction is infrequent in the athletes’ heart and its presence suggests pathologic hypertrophy. The hypertrophy of the athlete’s heart regresses with several months of deconditioning, a feature that reliably separates it from pathologic hypertrophy. The clinician should also be aware of the impact of the illicit use of anabolic steroids on the heart, used in an effort to boost performance. These agents may result in greater degrees of hypertrophy than seen due to a pure training effect and also result in premature coronary artery disease.

| Table 22.7 | CARDIAC STRUCTURE AND FUNCTION IN ENDURANCE-TRAINED ATHLETES, COMBINED ENDURANCE- AND STRENGTH-TRAINED ATHLETES, STRENGTH-TRAINED ATHLETES, AND CONTROL SUBJECTS |
|---|---|---|---|---|---|
| | Endurance-Trained Athletes | Combined Endurance- and Strength-Trained Athletes | Strength-Trained Athletes | Control Subjects | P |
| LVID_d (mm) | 53.7 | 56.2 | 52.1 | 49.6 | <0.001 |
| PWT_d (mm) | 10.3 | 11.0 | 11.0 | 8.8 | <0.001 |
| RWT | 0.389 | 0.398 | 0.442 | 0.356 | <0.001 |
| LVM | 249 | 288 | 267 | 174 | <0.001 |
The Heart in Pregnancy

Pregnancy results in substantial physiologic and hemodynamic changes that have manifestations on the echocardiogram (Table 22.8). By the third trimester of pregnancy, there is an increase in blood volume of 50%, a decline in peripheral vascular resistance, and an increase in cardiac output. These changes reach their maximum at the end of the second trimester. This results in a mild increase in chamber dimensions and the appearance of a high-output state with an increased stroke volume. Typically, the left atrium increases in size by 10% to 15% and the left ventricle by 5% to 10% (Fig. 22.86). Mild dilation of the right atrium and right ventricle is also present. The increased stroke volume manifests as an increased time velocity integral of aortic and pulmonary flow (Fig. 22.87). Mild degrees of tricuspid insufficiency are commonly encountered. Other features of pregnancy include small pericardial effusions, which can be seen in 20% of patients. Effusions resulting in hemodynamic compromise do not occur due to uncomplicated pregnancy, and if there is evidence of hemodynamic compromise, an alternate etiology for the effusion should be considered.
FIGURE 22.85. Parasternal long-axis echocardiogram recorded in a marathon runner (height, 5 ft 10 in; weight, 150 lb; body surface area [BSA] = 1.8 m$^2$). Note the mild left ventricular dilation for a subject of this body size and the wall thickness, which is at the upper normal range. Relative wall thickness (RWT) is preserved at 0.34. Left ventricular mass index is at the upper range of normal. LVID$_d$, left ventricular end-diastolic internal diameter; PW, posterior wall.

The mild left ventricular dilation can secondarily change the appearance of the mitral valve. On occasion, one encounters a female patient with mitral valve prolapse and mitral regurgitation in whom the prolapse becomes less apparent during pregnancy. The mechanism underlying this phenomenon is the more ideal mitral valve coaptation, which occurs as a result of an increase in left ventricular volume and internal dimensions.

In late pregnancy, the enlarged uterus results in compression of thoracic structures, including the heart. This may result in a motion abnormality in the
posterior wall, similar to that seen in chronic liver disease with significant ascites (Fig. 22.22).

Rarely after pregnancy an acute cardiomyopathy develops, referred to as peripartum cardiomyopathy. The echocardiographic appearance of peripartum cardiomyopathy is identical to dilated cardiomyopathy of any etiology, as discussed in Chapter 17. Finally, the peripartum period may represent a period of vascular “laxity,” and both aortic and coronary artery dissections are more common at this time. If a pregnant or peripartum patient presents with acute chest pain, consideration should be given to these entities.

<table>
<thead>
<tr>
<th>Table 22.8</th>
<th>CARDIOVASCULAR AND ECHOCARDIOGRAPHIC CHANGES IN PREGNANCY</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Physiologic Sequelae</strong></td>
<td><strong>Echocardiographic Findings</strong></td>
</tr>
<tr>
<td>Increased blood volume</td>
<td>Dilation of LA, LV</td>
</tr>
<tr>
<td>Decreased systemic vascular resistance</td>
<td>Increased LV stroke volume</td>
</tr>
<tr>
<td>Increased stroke volume and cardiac output</td>
<td>Altered MV coaptation</td>
</tr>
<tr>
<td></td>
<td>Mild tricuspid regurgitation</td>
</tr>
<tr>
<td></td>
<td>Elevated TR velocity (mild)</td>
</tr>
<tr>
<td>Other</td>
<td>PAC, premature atrial contraction; PSVT, paroxysmal supraventricular tachycardia; PVC, premature ventricular contraction; TR, tricuspid regurgitation.</td>
</tr>
<tr>
<td>Pericardial effusion</td>
<td>Increased prevalence of benign arrhythmias (PVCs, PACs, PSVT)</td>
</tr>
</tbody>
</table>
FIGURE 22.86. A, B: Parasternal long-axis view echocardiogram recorded in
diastole (A) and systole (B) in a healthy female patient in the third trimester of pregnancy. Note the mild dilation of the left atrium and the left ventricular systolic function, which is at the upper normal range.

Effects of Advanced Age

With age, there are predictable changes commonly seen in the heart. One of the most common is a progressive angulation between the ascending aorta and left ventricular outflow tract often in conjunction with localized proximal septal hypertrophy (Fig. 22.88). This results in a “sigmoid” shape to the proximal ventricular septum. The hypertrophy may be quite focal and result in a localized area of turbulence in the outflow tract that may be the source of the ejection murmur often heard in elderly patients.

There is a progressive increase in the likelihood of annular calcification with age. With advanced age, even in the absence of sustained hypertension, myocardial stiffness increases. This results in chronic diastolic dysfunction that can be detected with Doppler techniques and that results in left atrial dilation, secondary pulmonary hypertension, and an increased prevalence of atrial fibrillation (Fig. 22.89). In addition, characteristic changes will be seen in the wall of the aorta due to progressive thickening. Mild focal degrees of thickening are common in the aortic and mitral valves as well as the mitral valve chordae. With advanced age combined with long-standing hypertension (especially if poorly controlled), a pattern mimicking hypertrophic cardiomyopathy may develop (Fig. 22.90).
FIGURE 22.87. Spectral Doppler recordings from the same patient presented in Figure 22.86. **A:** Note the mitral inflow pattern with an E/A ratio of 2.2. **B:** The right ventricular outflow tract (RVOT) time velocity integral (TVI) is elevated at 17 cm. **C:** The left ventricular outflow tract tracing reveals an elevated peak velocity of 2 m/s and an increased TVI of 27 cm. Note in Figure 22.86 that there is no evidence of aortic stenosis or other outflow tract obstructions and the elevated velocities are the result of high cardiac output and not obstruction.

FIGURE 22.88. Apical long-axis view recorded in an elderly patient with a systolic ejection murmur. In the central panel note the marked angulation of the proximal anterior septum. This is related to age-related angulation between the proximal aorta and proximal septum. At the upper right is a color Doppler image. Note the atypically oriented flow acceleration (arrows) at the angulation of the ventricular septum suggesting acceleration of flow around the septal bulge. No functional obstruction to flow is present.
FIGURE 22.89. Apical four-chamber view recorded in an elderly patient with a history of atrial fibrillation and mild congestive heart failure manifest predominantly as fluid retention and edema. In the central figure note the marked dilation of the right and left atria but the relatively normal left ventricular size and function. The right ventricle is dilated. In the upper left panel note the moderate tricuspid regurgitation and in the lower right panel the tricuspid regurgitation peak velocity of 3.6 cm corresponding to a peak pressure gradient of 52 mm Hg suggesting the presence of moderate pulmonary hypertension. This combination of biaatrial enlargement with preserved left ventricular systolic function, often in conjunction with functional tricuspid regurgitation and modest pulmonary hypertension as well as atrial fibrillation is an increasingly common constellation of tenderness to encounter in the elderly population.
FIGURE 22.90. Apical four-chamber view recorded in an elderly hypertensive patient with the “hypertensive hypertrophic cardiomyopathy of the elderly.” A: In the apical four-chamber view, note the relatively small cavity size and evidence of left ventricular hypertrophy. B: In systole, note the systolic anterior motion of the...
mitral valve (arrow). The inset is a continuous-wave Doppler image recorded through the left ventricular outflow tract showing a characteristic late peaking velocity consistent with dynamic outflow tract obstruction.

Video 22-90

Suggested Readings

**Atrial Fibrillation**


**Pulmonary Embolism/Pulmonary Hypertension**


Chung T, Emmett L, Mansberg R, Peters M, Kritharides L. Natural history of right ventricular


**Systemic Disease**


**Hypertension, Diabetes, and Obesity**


**Cardio-Oncology**


**MISCELLANEOUS**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
In addition to its widespread use in ambulatory and hospitalized patients with known and suspected cardiac disease, echocardiography plays a valuable role in the management of patients in the emergency department and in medical and surgical intensive care units. In the operating room, transesophageal echocardiography is instrumental in determining the success of valve repair and for identification of surgical complications. The performance of echocardiography in intensive care units, the emergency department, operating room, and catheterization laboratory is often challenging and complicated by a less than ideal imaging environment. This is related to the often critically ill nature of the patients who may require concurrent urgent therapy, are not able to cooperate with breath holding or ideal positioning, and may be undergoing mechanical ventilation. The operator should recognize that limited imaging planes will be common, and that an examination targeted to specific clinical questions may be all that is available. Even within these limitations a skilled echocardiographer/clinician can often answer the majority of clinically relevant questions through a combination of direct imaging and assumptions based on indirect findings.
The use of echocardiography in patients with coronary disease is well established and is discussed in Chapter 15. Echocardiography also plays a valuable role in management of patients in medical intensive care units with a broad range of problems such as hypoxia, sepsis, hypotension, and shock. Surveillance studies have suggested that as many as one-fourth of patients in a medical intensive care unit have an underlying cardiovascular abnormality that may mimic a noncardiac condition and/or complicate therapy. Its use is similar in patients in a postsurgical intensive care unit. Table 23.1 outlines a number of clinical disorders which are encountered in an intensive care unit for which echocardiography plays a role in management. Table 23.2 outlines the areas in which echocardiography is considered an appropriate diagnostic test in these settings. It should be emphasized that in many instances the role of echocardiography will be to exclude cardiovascular disease as a cause of hemodynamic instability and hence allow the clinician to appropriately direct attention to noncardiovascular conditions. There are a number of echocardiographic findings which are of relevance to the likelihood of postoperative complications. These include occult left ventricular systolic dysfunction and pulmonary hypertension, both of which have been associated with worsened outcomes related not necessarily to cardiovascular complications but to organ system failure, sepsis, and other noncardiac issues.

**Hypotension and Shock**

In dealing with patients with hypotension and shock, one must distinguish among a cardiac etiology resulting in primary reduction in cardiac output, a purely noncardiac entity such as sepsis or hemorrhage with hypovolemia, and cardiac entities resulting in hemodynamic instability such as acute valvular insufficiency. It is also important to identify concurrent cardiac abnormalities that may complicate either diagnosis or therapy. Figures 23.1 through 23.6 were recorded in patients in medical or surgical intensive care units with a variety of acute illnesses. Patients with severe infection and sepsis may have acute, severe left ventricular dysfunction in the absence of coronary disease or pre-existing cardiomyopathy. Figure 23.1 was recorded in a patient hospitalized with sepsis, hypotension, and malperfusion. The echocardiogram documented severe left ventricular systolic dysfunction that improved after
Patients with pre-existing pulmonary disease may be hospitalized with acute respiratory compromise related to decompensated pulmonary disease and/or decompensation of concurrent congestive heart failure. When a patient presents in this manner, it can be difficult to ascertain the contribution of underlying cardiac disease from that secondary to pulmonary disease. Figure 23.4 was recorded in a patient presenting with multilobar pneumonia requiring ventilatory support who had clinical right heart failure. Notice the marked enlargement of the right ventricle and right atrium with secondary tricuspid regurgitation and evidence of right ventricular pressure overload. Pulmonary hypertension confers a worsened prognosis for patients with acute severe medical illness. Severe pulmonary hypertension may result in a syndrome in which overall cardiac output is limited by right heart flow. In advanced cases, this may compromise left ventricular filling and make patients susceptible to significant hypotension in the presence of vasodilation related to either medical therapy or sepsis.

<table>
<thead>
<tr>
<th>Table 23.1 USE OF ECHOCARDIOGRAPHY IN THE INTENSIVE CARE UNIT</th>
</tr>
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<tbody>
<tr>
<td>Surveillance</td>
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<tr>
<td>Confirm/exclude occult cardiac disease</td>
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<tr>
<td>Hemodynamics</td>
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<tr>
<td>Hypotension</td>
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<tr>
<td>Assess volume status</td>
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<tr>
<td>Left ventricular function</td>
</tr>
<tr>
<td>Regional wall motion abnormality</td>
</tr>
<tr>
<td>Global dysfunction</td>
</tr>
<tr>
<td>Transient dysfunction (sepsis, stunning)</td>
</tr>
<tr>
<td>Right ventricular function</td>
</tr>
<tr>
<td>Outflow tract obstruction</td>
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<tr>
<td>Valvular stenosis/insufficiency</td>
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<tr>
<td>Hypoxia</td>
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<tr>
<td>Right ventricular function</td>
</tr>
<tr>
<td>Right ventricular pressure</td>
</tr>
<tr>
<td>Intracardiac shunt</td>
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<tr>
<td>Pulmonary embolus</td>
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</tbody>
</table>

<p>| Infections                                                   |
| Bacterial endocarditis                                       |</p>
<table>
<thead>
<tr>
<th>Indication</th>
<th>Appropriateness Score (1–9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Symptoms or conditions potentially related to suspected cardiac etiology including but not limited to chest pain, shortness of breath, palpitations, TIA, stroke, or peripheral embolic event</td>
<td>A (9)</td>
</tr>
<tr>
<td>19. Hypotension or hemodynamic instability of uncertain or suspected cardiac etiology</td>
<td>A (9)</td>
</tr>
<tr>
<td>20. Assessment of volume status in critically ill patient</td>
<td>U (5)</td>
</tr>
<tr>
<td>26. Respiratory failure or hypoxemia of uncertain etiology</td>
<td>A (8)</td>
</tr>
<tr>
<td>27. Respiratory failure or hypoxemia when a noncardiac etiology of respiratory failure has been established</td>
<td>U (5)</td>
</tr>
<tr>
<td>29. Known acute pulmonary embolism to guide therapy (e.g., thrombectomy and thrombolytics)</td>
<td>A (8)</td>
</tr>
<tr>
<td>32. Severe deceleration injury or chest trauma when valve injury, pericardial effusion, or cardiac injury is possible or suspected</td>
<td>A (9)</td>
</tr>
<tr>
<td>33. Routine evaluation in the setting of mild chest trauma with no echocardiographic changes or biomarker elevation</td>
<td>rA (2)</td>
</tr>
<tr>
<td>62. Guidance of percutaneous noncoronary cardiac procedures including but not limited to pericardiocentesis, septal ablation, or right ventricular biopsy</td>
<td>A (9)</td>
</tr>
<tr>
<td>99. Use of TEE when there is a high likelihood of a nondiagnostic TTE due to patient characteristics or inadequate visualization of relevant structures</td>
<td>A (8)</td>
</tr>
<tr>
<td>103. Guidance during percutaneous noncoronary cardiac interventions including but not limited to closure device placement, radiofrequency ablation, and percutaneous valve procedures</td>
<td>A (9)</td>
</tr>
<tr>
<td>112. Evaluation to facilitate clinical decision making with regard to anticoagulation, cardioversion, and/or radiofrequency ablation</td>
<td>A (9)</td>
</tr>
<tr>
<td>201. Routine use of contrast All LV segments visualized in noncontrast images</td>
<td>rA (1)</td>
</tr>
<tr>
<td>202. Selective use of contrast ≥2 contiguous LV segments are not seen on noncontrast images</td>
<td>A (8)</td>
</tr>
</tbody>
</table>

A, appropriate; rA, rarely appropriate; U, uncertain.

FIGURE 23.1. Parasternal long-axis view recorded in systole in a patient with a structurally normal heart and no previous evidence of organic heart disease. This image was recorded in the medical intensive care unit in a patient with severe sepsis and demonstrates mild dilation and global hypokinesis of the left ventricle. The inset at the upper left was recorded 4 weeks after full resolution. Notice in this end-systolic view the decrease in end-systolic dimension and in the real-time images recovery of normal left ventricular systolic function. Video 23-1a
FIGURE 23.2. Parasternal long-axis view recorded in an elderly patient with pneumonia who presented with hypotension and shock. Note the very small left ventricular cavity (double-headed arrow) with normal systolic function suggesting that hypovolemia is the etiology for hypotension.
FIGURE 23.3. Apical four-chamber view recorded in a patient with hypotension and shock after an acute febrile illness. Note the global hypokinesis of the left ventricle (consistent with an underlying cardiomyopathy). The Doppler pattern suggests pseudonormal filling (grade 2 diastolic dysfunction). In this instance, there was no recovery of function with treatment of the underlying illness. Incidental note is made of a pseudochord in the left ventricular apex (arrow).
Because of the critically ill nature of these patients, and the fact that many are often on ventilatory support, transthoracic imaging may be suboptimal. It is often feasible, however, to evaluate the status of left ventricular function and exclude systolic dysfunction as a cause of hypotension even in poor-quality images. The use of intravenous contrast for left ventricular opacification is often essential for assessment of left ventricular function in ventilated patients (Fig. 23.7). Although it may be possible to determine the status of left and right ventricular function from transthoracic echocardiography using contrast echocardiography, detailed evaluation of valvular anatomy and hemodynamics may require transesophageal echocardiography. Several studies have demonstrated the incremental value of transesophageal echocardiography for elucidating the underlying mechanism of hypotension or hypoxia in patients hospitalized in an intensive care unit with a broad range of disorders. Because one of the more common questions to be answered in the intensive care unit is that of left ventricular systolic function, a limited examination providing this isolated answer, even when an assessment of detailed valvular anatomy cannot be made, often enhances clinical decision making.
FIGURE 23.4. Transthoracic echocardiogram recorded in a patient with multilobar pneumonia. The middle right figure was recorded 1 year previously and demonstrates normal left and right ventricular size and function. At the time of presentation with hypoxia and multilobar pneumonia requiring mechanical ventilation, note the dilation of the right atrium and right ventricle with a right ventricular overload pattern on the ventricular septum (arrows). The tricuspid regurgitation velocity is mildly elevated at 3 m/s, in line with secondary pressure elevation in a nonpreconditioned ventricle.
FIGURE 23.5. Parasternal short-axis view recorded in a 37-year-old woman presenting with a febrile illness and hypotension. Note the massively dilated and hypertrophied right ventricle and the small slit-like left ventricle consistent with a severe right ventricular pressure overload. The tricuspid regurgitation gradient suggests systemic right ventricular systolic pressure due to previously unrecognized primary pulmonary hypertension.
One cause of hypotension in critically ill patients, especially in a surgical or trauma intensive care unit, is hemorrhage and hypovolemia. This entity is suggested on an echocardiogram when small left ventricular volume and hyperdynamic motion are noted (Fig. 23.2). This is reliable evidence of intravascular volume depletion and has obvious therapeutic implications. On occasion, one encounters a patient with progressive hypotension in whom the use of intravenous pressors results in no improvement or further deterioration. There is a subset of patients, many of whom have a history of hypertension, who with volume depletion develop acquired dynamic left ventricular outflow tract obstruction which mimics obstructive hypertrophic cardiomyopathy. Systolic anterior motion of the mitral valve with secondary mitral regurgitation also may be seen. The overall hemodynamic result of this syndrome is progressive hypotension with the development of a prominent systolic murmur (due to outflow tract obstruction and/or mitral regurgitation). The etiology of the hypotension in this situation is the relatively low left ventricular stroke volume due to hypovolemia, complicated by outflow tract obstruction. Left ventricular outflow tract gradients exceeding 100 mm Hg have been noted in this situation. In this instance, right heart catheterization reveals an elevated pulmonary capillary wedge pressure that is then assumed to reflect left ventricular filling volume. When the syndrome of significant mitral regurgitation with outflow tract obstruction is identified, one should recognize that the elevated pulmonary capillary wedge pressure is the result of a hyperdynamic but noncompliant left ventricle and mitral regurgitation.
Failure to appreciate this phenomenon results in the inappropriate course of increasing pressor support and diuretics, which obviously has the effect of worsening rather than improving the clinical situation. Figures 23.8 and 23.9 were recorded in a patient with this syndrome. Recognition of hypovolemia with dynamic outflow tract obstruction should lead to the appropriate management decision to resuscitate the patient with fluids and withdraw agents, which increase contractility and/or reduce vascular resistance.

**FIGURE 23.6.** Serial parasternal long-axis echocardiograms recorded in a young patient 4 hours after renal transplantation who developed hypotension and was unable to be weaned from the ventilator. For each pair of images diastole is on the top and systole is on the bottom. A, C: Images recorded at the time of clinical deterioration reveal septal akinesis with otherwise global hypokinesis. B, D: Images recorded 2 days later demonstrate complete recovery of function. In this instance, the left ventricular dysfunction was due to myocardial stunning of uncertain provocation not related to obstructive coronary disease.
Diastolic dysfunction may result in pulmonary congestion and heart failure in patients hospitalized with medical illnesses or in the postoperative period. These patients typically are elderly and have a history of hypertension. In the operative or postoperative period, overly aggressive fluid resuscitation may result in congestive heart failure. The echocardiogram will typically reveal normal systolic function and left ventricular hypertrophy (Fig. 23.10). Mitral inflow patterns may be highly variable and show either delayed relaxation or a restrictive filling pattern. If intravascular volume overload is present, a pseudonormal inflow pattern is not uncommon.

**Evaluation of the Hypoxic Patient**
Echocardiography can be used in an intensive care unit for evaluation of unexplained hypoxia or inability to wean from ventilatory support. Etiologies of hypoxia that can be documented by echocardiography are listed in Table 23.1. A comprehensive echocardiographic examination is useful in patients with hypotension and shock to exclude a primary cardiac abnormality. If no primary cardiac abnormality is identified, including right-to-left shunting, then the etiology of hypoxia can reliably be assumed to be noncardiac and appropriate diagnostic and therapeutic efforts directed to pulmonary or other causes. A cause of hypoxia in the intensive care unit that is uniquely evaluated by echocardiography is the opening of a patent foramen ovale (PFO) with subsequent right-to-left shunting (Fig. 23.11). This generally requires not only the presence of a PFO but also a concurrent process that elevates right heart pressure such as pulmonary hypertension, acute pulmonary embolus, or right ventricular dysfunction. Additionally, reactive pulmonary hypertension of any etiology, including that provoked by bronchospasm, can result in sufficient elevation of right heart pressure that a patent foramen becomes a source for significant right-to-left shunting.
FIGURE 23.7. Apical four-chamber view recorded in a patient hospitalized in a medical intensive care unit with sepsis and multiorgan system failure. **A:** An apical four-chamber view from which an accurate assessment of left ventricular function...
cannot be made. B: Recorded after an injection of intravenous contrast for left ventricular opacification, after which normal left ventricular systolic function is noted.
FIGURE 23.8. Transesophageal echocardiogram recorded in a patient in the medical intensive care unit with gastrointestinal bleeding. The patient was being supported with intravenous inotropes but became progressively hypotensive. This figure demonstrates development of “pseudohypertrophic cardiomyopathy” related to volume depletion and inotropic support. A: Note the systolic anterior motion of the anterior mitral valve leaflet (arrow). B: was recorded with color Doppler and reveals severe mitral regurgitation with a peak pressure gradient between the left ventricle and left atrium 192 mm Hg due to dynamic outflow tract obstruction. While this continuous-wave Doppler was not recorded through the left ventricular outflow tract, it demonstrates a markedly elevated left ventricular to left atrial pressure gradient of 192 mm Hg in a patient with systemic hypotension. The implication is that the difference between the left ventricular–left atrial pressure gradient and systolic systemic pressure is equivalent to the dynamic intracavitary gradient. Video 23-8a
FIGURE 23.9. Transthoracic echocardiogram recorded in the same patient presented in Figure 23.8 following discontinuation of inotropic therapy and full volume resuscitation. Note the larger left ventricular cavity and absence of obstructive systolic anterior motion (arrow). The inset at the upper left is continuous-wave Doppler through the outflow tract demonstrating a normal gradient.
An additional source of right-to-left shunting is a pulmonary arteriovenous malformation (AVM). AVMs are seen in chronic liver disease as well as in Osler–Weber–Rendu syndrome. Most AVMs result in clinically inconsequential degrees of shunting and rarely result in clinically relevant, or even detectable, hypoxia. On occasion, large or multiple AVMs may result in substantial right-to-left shunting with clinically relevant hypoxia. Separation of an AVM from atrial level communication is discussed in Chapter 3 and relies on the timing and other characteristics of contrast appearance in the left side of the heart. Typically, contrast appearance in the left side of the heart related to an AVM is delayed by several cardiac cycles, but then builds persistently rather than appearing phasically, as is typically seen in atrial level shunts (Fig. 23.12). The basis for this echocardiographic finding is that before it appears in the left side of the heart, contrast must pass through the entire pulmonary vascular circuit. This typically takes three to six cardiac cycles depending on cardiac output. The pulmonary circuit then acts as a reservoir of contrast that continues to flow into the left side of the heart even after the initial intravenous bolus has begun clearing from the right side of the heart.
FIGURE 23.10. Parasternal long-axis echocardiogram recorded in a patient with long-standing hypertension admitted to an intensive care unit with ketoacidosis. Note the left ventricular hypertrophy and normal systolic function in this end-systolic image. The accompanying Doppler profile suggests diastolic dysfunction, which may make this patient more susceptible to pulmonary congestion during aggressive volume resuscitation.
Echocardiography in the Neurosurgical and Neurology ICU

Patients presenting with acute neurologic events, or who have undergone intracranial procedures, may have a variety of cardiovascular complications. Obviously, echocardiography can be used to identify pre-existing cardiovascular diseases which may have an impact on patient management. One finding which is unique to the neurologic and neurosurgical patient is that of “neurogenic stunning,” a phenomenon similar to Takotsubo cardiomyopathy. Following a neurologic event, typically an intracranial hemorrhage, a number of patients develop acute wall motion abnormalities predominately in the septum and apex. This is associated with deep symmetric T-wave inversion on the electrocardiogram and only a minimal troponin leak. This is seen in the absence of obstructive coronary disease and typically follows a time course of resolution similar to that in Takotsubo syndrome. **Figure 23.13** was recorded in a patient presenting with an intracranial hemorrhage who developed marked EKG changes and was noted to have significant wall motion abnormalities mimicking a left anterior descending coronary artery infarct.
FIGURE 23.11. Apical four-chamber view recorded in a patient with obstructive lung disease and significant hypoxia. Note the significant opacification of the left ventricular cavity after intravenous injection of agitated saline. This is indicative of a significant right-to-left shunt due to opening of a patent foramen ovale.

Video 23-11
FIGURE 23.12. Transesophageal echocardiogram recorded in a patient with hypoxia while on mechanical ventilatory support 24 hours after liver transplantation. In the upper panel, note the mild right heart dilation but the otherwise structurally normal heart and the absence of atrial septal defect. The lower panel was recorded 7 seconds after appearance of intravenous saline in the right side of the heart and demonstrates a substantial right-to-left shunt related to a pulmonary AVM. In the real-time images, note the smooth homogeneous buildup of contrast in the left side of the heart, which is a characteristic of a pulmonary AVM, as opposed to phasic appearance typically seen with atrial level shunts.
FIGURE 23.13. Apical four-chamber view recorded in a patient in the neurosurgical intensive care unit with an intracranial hemorrhage. In the systolic view note the dyskinesis of the distal septum and apex (arrows) in a young patient subsequently demonstrated to be free of coronary artery disease. At the lower right note the symmetric T-wave inversion in the electrocardiogram. The head CT at the upper right demonstrates a right temporal lobe hematoma.
FIGURE 23.14. Subcostal images recorded in a patient undergoing V–A ECMO. In the central image note the large bore cannula in the inferior vena cava with robust color flow (arrows) related to active withdrawal of venous blood from the inferior vena cava. The inset at the upper left was recorded without color Doppler and better visualizes the large bore inferior vena cava cannula (arrows).
Echocardiographic Monitoring of Patients on Circulatory Support

Patients who are critically ill with severely compromised oxygenation and/or cardiac output may be placed on a variety of circulatory support devices including extracorporeal membrane oxygenation (ECMO), partial left or right ventricular assist devices, and other systems. ECMO allows for oxygenation of blood by way of a dedicated extracorporeal circuit. The intake and output circuit location can be variable and may include withdrawal from the central venous system and injection into the central arterial system (V–A ECMO) or other combinations of intake and output. ECMO is typically undertaken for patients who have a pulmonary limitation to oxygenation such as severe multi lobar pneumonia. It can also be used to support patients with severe right ventricular failure. Figures 23.14 to 23.17 were recorded in patients with a variety of conditions in whom an ECMO circuit is being utilized and demonstrate several of the anticipated echocardiographic findings. As a part of evaluation of patients on ECMO, an assessment for suitability of withdrawal of circulatory support is occasionally undertaken. Echocardiographic monitoring can be used to assess the impact of changing ECMO flow on right or left ventricular systolic function and chamber sizes. Figure 23.18 was the recorded time of a trial of ECMO withdrawal in a patient with severe pulmonary hypertension. Note the worsening tricuspid regurgitation and right ventricular enlargement during ECMO withdrawal. Also note, related to reduced left atrial flow from the ECMO circuit, the
reduction in left ventricular systolic function and size.

**FIGURE 23.15.** Apical four-chamber view recorded in a patient undergoing V–V ECMO for pulmonary-related hypoxia. In the central image (recorded in systole) note the highly turbulent flow in the right atrium consistent with outlet flow from the ECMO circuit (*single arrow*) as well as tricuspid regurgitation (*downward-pointing arrows*). The inset at the upper left is continuous-wave Doppler suggesting moderate pulmonary hypertension. The inset at the lower left was recorded in diastole showing cannula flow (*arrows*).
FIGURE 23.16. Parasternal long-axis view recorded in a patient with end-stage pulmonary hypertension undergoing ECMO. In this instance the inlet cannula is in the inferior vena cava and the outlet cannula has been placed into the left atrium (inward-facing arrows). Note the right ventricular pressure overload pattern of the ventricular septum (downward-pointing arrows) and the marked dilation and hypertrophy of the right ventricle (bold arrow). The inset at the lower left is recorded with color Doppler and reveals continuous high-volume flow into the left atrium from the ECMO outlet cannula. (9)
The Impella device is also a percutaneously placed temporary left ventricular systolic device placed in the catheterization laboratory. Transfemoral approach is typically used and the device is placed across the aortic valve into the left ventricle. Within the 9 French catheter device is a larger section containing the actual pump. Blood is withdrawn from distal ports on the catheter which is in the cavity of the left ventricle, and subsequently injected into the ascending aorta. Transthoracic or transesophageal echocardiography can be used to confirm appropriate placement of the device with the inlet portion in the body of the left ventricle and the output portion in the ascending aorta (Figs. 23.19 and 23.20).
FIGURE 23.17. Apical four-chamber view recorded in the same patient presented in Figure 23.16. Note the marked dilation of the right atrium and right ventricle and the small compressed left ventricle. This image was recorded during full ECMO support. The inset at the upper left is recorded with color Doppler and reveals mild tricuspid regurgitation. [Video 23-17 CFD]
FIGURE 23.18. Apical four-chamber view recorded in same patient presented in Figures 23.16 and 23.17. This image was recorded during a trial of ECMO withdrawal. With reduction in ECMO support there has been progressive dilation of the right atrium and right ventricle with further compromise of left ventricular size. The inset at the upper left was recorded with color Doppler and reveals an increase in tricuspid regurgitation severity.
An additional means for temporary left ventricular circulatory support is the TandemHeart device. The device is placed percutaneously in the catheterization laboratory or operating room. It consists of an input cannula placed across the atrial septum into the left atrium which withdraws blood from the left atrium by way of an externally placed centrifugal pump. Blood is then delivered at the equivalent of arterial pressure into the femoral artery by way of a percutaneously placed cannula. It both supports arterial pressure and perfusion and decompresses the left heart. From an echocardiographic perspective only the left atrial outflow catheter can be visualized (Fig. 23.21). Its placement in the left atrium can be confirmed either with transthoracic or transesophageal echocardiography.
Echocardiography in the Emergency Department

Guidelines have been developed outlining the appropriate level of training required to perform echocardiography in the emergency department by physicians other than fully certified echocardiographers. Multiple studies have demonstrated that with appropriate training noncardiologists can acquire the skills to obtain adequate quality images sufficient to diagnose many basic cardiovascular abnormalities relevant to emergency department decision making using of either full-service or hand-held echocardiographic devices. It should be stressed that the major limitation to accuracy and decision making using these devices is operator skill for obtaining and interpreting images. Without appropriate training utilization of abbreviated echocardiography may result in substantial error rates.

FIGURE 23.19. Parasternal long-axis view recorded in a patient with cardiogenic shock and temporary left ventricular support using an Impella device. The large bore catheter is placed across the aortic valve (arrows) into the left ventricle. Echocardiographic monitoring has been utilized to ensure appropriate positioning of the inlet portion of the device.
FIGURE 23.20. Transthoracic apical long-axis view recorded in a patient with an Impella device. In this systolic frame note the concurrent mitral regurgitation (MR). The longer downward-pointing arrow depicts a color Doppler signal which is a combination of electronic artifact and high-volume flow into the ascending aorta. The two inward-pointing arrows denote the left ventricular outflow tract. Notice there is no evidence of aortic insufficiency, nor is there evidence that the outflow of the Impella is inappropriately located in the left ventricle. The inset at the upper left was recorded with spectral Doppler through the left ventricular outflow tract. Note the marked electronic interference from the device which precludes accurate
For patients presenting to the emergency department with hypotension, shock, or major trauma (especially thoracic), many of the same considerations noted above regarding use of echocardiography in the intensive care unit apply. Obviously for patients with major trauma, hemorrhagic shock is a consideration, in which case echocardiography can quickly document a small, underfilled ventricle. Additionally, in patients with major blunt chest trauma, such as after a high-speed motor vehicle accident, echocardiography can be instrumental in documenting cardiac involvement including myocardial contusion, pericardial effusion, or aortic trauma. By confirming the absence of significant cardiac involvement, echocardiography allows the clinician to redirect efforts to alternate explanations for hypotension.
FIGURE 23.21. Transesophageal echocardiogram recorded in a four-chamber view in a patient immediately after placement of a TandemHeart device. This was a procedure monitoring transesophageal echocardiogram in the catheterization laboratory. Note the large bore catheter crossing the atrial septum (arrow) into the left atrium. The inset is a real-time three-dimensional transesophageal echocardiogram from the same study nicely demonstrating the full extent of the catheter in the cavity of the left atrium (single arrow). The plane of the atrial septum is noted by the pair of inward-pointing arrows.
FIGURE 23.22. Attempt to obtain a parasternal long-axis echocardiogram in a patient after a motor vehicle accident. Identical images were obtained from multiple transthoracic transducer positions and reveal only ultrasound “noise.” In the real-time image, notice the oscillating nature of the echoes in the near field. These images are consistent with subcutaneous air secondary to chest trauma.
Patients likely to have sustained cardiac trauma often have concurrent, thoracic, abdominal, or major limb trauma. As a consequence of this, the independent specific cardiac abnormalities may be masked by hypovolemia from hemorrhage. The majority of patients with significant cardiac and/or aortic trauma have multiple rib fractures, hemo- or pneumothorax, and other complications which render transthoracic imaging problematic. Because of chest trauma, atypical imaging windows may be necessary. Occasionally, after an intrathoracic procedure or major chest trauma, transthoracic echocardiography results in total failure to visualize any cardiac structures. This may be associated with strong and occasionally dynamic reverberation signals (Figs. 23.22 and 23.23). When this scenario is encountered, one should suspect either subcutaneous air, pneumothorax, or pneumomediastinum. In these instances, transesophageal echocardiography is essential and can identify the majority of cardiac injuries.
FIGURE 23.23. Attempted apical four-chamber view recorded in a patient in the trauma ICU following a high-speed vehicular accident. Note the failure to visualize any cardiovascular structures from the apical view and the four reverberation signals originating at the apex (arrows) which obscure the left ventricle. This is the result of air either in the mediastinum or pleural space. Video 23-23
FIGURE 23.24. Transesophageal echocardiogram recorded in a young patient with hypotension, shock, and a loud murmur after a stab wound to the chest. 

A: In the longitudinal view, note the global hypokinesis of both the left and right ventricles, the etiology of which is presumed to be coronary injury. 

B: Color flow imaging reveals an abnormal communication between the left ventricular cavity and the left atrium consistent with a direct penetrating injury of the mitral valve.
The majority of modern emergency departments, especially those associated with a trauma care center will have computed tomography available either within the Emergency Department or immediately adjacent. Typically this may be the first examination undertaken in patients with significant trauma and serves to provide evaluation of pericardial effusion and aortic trauma. Because of this availability the specific need for and utilization of echocardiography on an emergency basis in trauma patients has declined.

Forms of trauma other than blunt chest trauma include penetrating injuries from knife or bullet wounds. The echocardiographer should be cognizant of the unpredictable path of a high-velocity penetrating injury and the need for
atypical imaging planes. In general, patients with any significant penetrating cardiac trauma will have a pericardial effusion, and its absence is circumstantial evidence that a penetrating cardiac injury has not occurred. Cardiac contusion and injury, however, can occur from the “shock” effect of bullet wounds to the chest, in which case penetration of cardiac structures will not be noted. Figures 23.24 to 23.26 were recorded in patients with penetrating cardiac trauma.

**Echocardiography Following Cardiac Arrest**

Cardiac arrest is the result of a variety of mechanisms and is typically classified as being arrhythmic, pulseless electrical activity, or asystolic. The underlying mechanisms can be distinct for each and outcomes are dependent on the nature of the arrest. Specific therapy may be indicated on the basis of the precipitating cause. Several studies have suggested the utility of a limited, rapid, bedside echocardiogram, often performed with handheld devices, to facilitate rapid diagnosis and decision making in patients suffering witnessed cardiac arrest. Figures 23.27 and 23.28 were recorded in patients shortly after resuscitation from cardiac arrest. Note in Figure 23.27 the apical dyskinesia suggesting ischemic disease as the substrate. In Figure 23.28, note the normal left ventricle with marked right ventricular dilation and dysfunction raising the possibility of an acute pulmonary embolus as the etiology. Obviously, management would be altered on the basis of these findings.
FIGURE 23.25. Echocardiogram recorded in the emergency department in a patient with a minor shotgun wound to the chest. In four-chamber view, note the normal anatomy of the four cardiac chambers. There is a reverberation artifact originating at the apex of the right ventricle (arrows) also demonstrated in the subcostal image at the upper right. The image at the lower left is a chest radiograph demonstrating a single shotgun pellet over the cardiac silhouette. The image at the upper left is a chest CT also demonstrating a metallic object in the right ventricle. [Video]
coming soon

Video 23-25
FIGURE 23.26. Parasternal echocardiogram recorded in a patient with hypotension and shock after a gunshot wound to the chest. Note in both the parasternal long-axis and short-axis views that there is a “cloudy” pericardial effusion (arrows) consistent with acute hemorrhage into the pericardium. In the real-time image, note the apical akinesia consistent with myocardial or coronary
arterial injury. There was no evidence of penetration of the heart in this case.

Video 23-26a

coming soon

Video 23-26b

coming soon
FIGURE 23.27. Apical four-chamber view recorded in a patient in the emergency department after being resuscitated in the field. The 12-lead electrocardiogram showed only nonspecific ST- and T-wave changes. On the echocardiogram, there is clearly a distal septal and apical wall motion abnormality (arrows) consistent with occlusive coronary artery disease in the left anterior descending coronary artery which was subsequently demonstrated on catheterization.
FIGURE 23.28. Echocardiogram recorded in a 32-year-old patient immediately following resuscitation from cardiac arrest characterized as pulseless electrical activity (PEA). Note the marked dilation of the right atrium and ventricle and the small, underfilled left ventricle with normal left ventricular function. This pattern should direct attention toward an acute right ventricular insult such as massive pulmonary embolus, which was the diagnosis in this patient.
It is not uncommon following cardiac arrest and subsequent resuscitation, for global left ventricular dysfunction to be noted initially. This may be seen in the absence of underlying structural cardiovascular disease and is the result of an ischemic insult to the heart related to the arrest and not necessarily a primary cardiovascular event. Recovery of function typically occurs if resuscitation efforts have been timely and successful and may follow a somewhat unusual pattern of apical recovery followed by basal recovery.
The use of echocardiography in conjunction with cardiac and noncardiac surgical procedures can be divided into its use before surgery, in the operating room and in the postoperative period (Table 23.3). Although the most common echocardiographic modality to use in the operating room is transesophageal echocardiography, there are occasional situations in which transthoracic or other probes designed for epicardial scanning in the open chest, typically covered with a sterile sheath, are used for direct application to the heart or vascular structures.

The most common intraoperative application of echocardiography is for monitoring of valvular, congenital, or other complex cardiovascular surgical procedures. This includes mitral valve repair and implantation of newer bioprostheses. Intraoperative transesophageal echocardiography is standard of care for confirming the success of mitral valve repair and is also used to assess the success of valve replacement with respect to residual gradients and paravalvular regurgitation. Preoperative echocardiography is instrumental in assessing the indications for, and the likelihood of success of virtually all forms of valve surgery.

Performing echocardiography in the operating room poses a number of challenges. First, while echocardiography is typically performed in a dimly lit environment, the operating room is frequently brightly lit and visualizing images with appropriate gray-scale intensity on a video screen becomes problematic. A tendency to increase output and gain settings to compensate for this results in an abnormal appearance of myocardial texture, valvular and other structures when these same images are viewed in the more ideal environment of an analysis room. Second, echocardiography is often undertaken at the time of ongoing anesthetic or surgical procedures resulting in a rushed environment. Once the pericardium is opened and the heart exposed for an operative procedure, it is often no longer in a normal anatomical position. As such, imaging planes are often distorted and some standard views may become unobtainable (Fig. 23.29). Electronic interference, especially from electrocautery, results in substantial degradation of images (Fig. 23.30). The echocardiographer (whether a cardiologist or an anesthesiologist) should be prepared, within the limits of not impeding the surgical procedure, to intermittently request a pause in activity which interferes with optimal imaging. It is incumbent on the echocardiographer to acquire the skills necessary to rapidly acquire the critical information for
decision making without impeding the pace of an operative procedure. A final technical complexity in the postoperative setting is that patients may be undergoing temporary atrioventricular pacing. The atrial pacing spike may be misinterpreted by sensing algorithms in the ultrasound equipment and result in inappropriate capture of digital loops. The echocardiographer should check the integrity of digital capture early in the process of acquiring images in this setting.

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<thead>
<tr>
<th>Table 23.3</th>
<th>ECHOCARDIOGRAPHY IN THE OPERATING ROOM</th>
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<tbody>
<tr>
<td><strong>Preoperative</strong></td>
<td>Assess need for valvular surgery</td>
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<td>Left ventricular function</td>
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<td>Pulmonary artery pressure</td>
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<td>Aortic valve procedures</td>
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<td>Left ventricular outflow tract size</td>
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<td></td>
<td>Mechanism of regurgitation</td>
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<td>Feasibility of repair</td>
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<td><strong>Intraoperative</strong></td>
<td>Monitor LV and RV function for noncardiac procedures</td>
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<td>Placement of cannulas, occlusive devices</td>
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<td><strong>Postoperative</strong></td>
<td>Success of valve repair/replacement</td>
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<td>Detect complications (see Table 23.4)</td>
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FIGURE 23.29. Transesophageal echocardiogram recorded on an outpatient basis (A) and in the operating room (B) after opening the chest and pericardium but before institution of cardiopulmonary bypass. **A:** Note the more ideal orientation of the atria as well as the size of the left atrium. **B:** Recorded at the same plane rotation (0 degrees), note the distortion of atrial anatomy and the less optimal visualization of the plane of the mitral valve and foreshortening of the left ventricle, which is the result of the position of the heart within the chest after
opening of the pericardium.

It is imperative to recognize the characteristics of ventricular performance and aortic flow in a patient on cardiopulmonary bypass. While on complete bypass, the left ventricle is unloaded and its diastolic volume is reduced. In this situation, even in the beating heart, the ventricle may appear globally hypokinetic (Fig. 23.31). Once fully removed from bypass and after appropriate volume resuscitation, ventricular size and function should return to baseline. Depending on the nature of the surgery and its success, and the use of inotropic agents, ventricular function may be improved compared with baseline. Partial bypass, or incomplete volume restoration, results in
intermediate levels of ventricular performance. While on complete bypass, continuous-nonphasic flow will be seen in the aorta, related to the cardiopulmonary bypass cannula flow (Fig. 23.32).
FIGURE 23.30. Intraoperative transesophageal echocardiogram recorded before
Role of Echocardiography in Mitral Valve Surgery

Determination of the feasibility of mitral valve repair relies on the preoperative transesophageal echocardiogram. In general, posterior leaflet pathology is more easily repaired than anterior, and in general any disease process that scars or foreshortens the mitral valve apparatus results in anatomy less likely to be successfully repaired than diseases associated with excess or redundant tissue.

As surgical expertise for mitral valve repair has advanced and become more widespread, the actual incremental value of immediate preoperative transesophageal echocardiography has become somewhat less clear, as skilled surgeons are prepared to alter the intended procedure based on direct observation. Additionally, more complex procedures, including the use of neochords and other advanced techniques have become more common. Not infrequently a decision to utilize more advanced forms of surgical therapy is made at the time of direct inspection of the mitral apparatus.

When performing echocardiography for the purpose of assessing the mitral valve before repair, it is important that a thorough and detailed evaluation of the mitral valve be undertaken in a systematic fashion. The primary purpose of the examination is to determine the underlying anatomic
abnormality responsible for the regurgitation or stenosis. It is important to recognize that there are three different viewing perspectives on mitral valve anatomy (Fig. 23.33). The surgeon will be viewing the mitral valve from within the left atrium so that the anterolateral commissures will be to the left of the field of view and the medial commissures to the right. When viewed with either transesophageal or transthoracic echocardiography, this orientation will be reversed (assuming traditional recommended viewing formats on a video screen). Also, depending on whether the reference is a transthoracic or transesophageal echocardiogram, the anterior and posterior leaflets of the mitral valve will vary in position compared with the surgical perspective.
FIGURE 23.31. Transesophageal echocardiograms recorded in a patient on complete (A) and partial (B) cardiopulmonary bypass. A: Note that while on complete bypass, the left atrium and left ventricle are filled with homogeneous echoes consistent with marked stasis of blood flow. Note the fibrillating left
ventricle. Also note the relative absence of stasis in the aorta, which receives flow from a cardiopulmonary bypass cannula. B: Recorded after restoration of sinus rhythm and while on partial (1.5 L/min) cardiopulmonary bypass. Again, note the underfilled left ventricle with the poor ventricular function due to reduced filling and the substantial clearing of the spontaneous contrast within the chambers.

Video 23-31a

Video 23-31b

When determining the severity of mitral regurgitation, it is critical to recognize that intracardiac hemodynamics in an anesthetized and ventilated open chest patient are substantially different than hemodynamics in the awake or mildly sedated patient. For this reason, there can be marked differences in the apparent severity of mitral regurgitation when comparing an intraoperative transesophageal echocardiogram with one performed on an
ambulatory patient. In general, diseases resulting in functional mitral regurgitation will tend to have a reduction in the severity of mitral regurgitation when comparing the intraoperative with the preoperative studies. There is less reduction in the apparent severity of mitral regurgitation for patients with anatomic disruption of a valve leaflet than for those with functional mitral regurgitation. Figure 23.34 was recorded in two patients preoperatively and reveals severe mitral regurgitation. The lower panels were recorded in the same patients during an intraoperative study and reveal substantially less mitral regurgitation. Of note, blood pressure and heart rate were equivalent at the time of the examinations.
FIGURE 23.32. Intraoperative transesophageal echocardiogram of the aortic arch during cardiopulmonary bypass. Note the continuous high-velocity flow in the aortic arch on the color flow image, which is also appreciated in the color Doppler M-mode image. This is the result of continuous flow into the aorta from the cardiopulmonary bypass apparatus and does not represent pathology.
Evaluation of the mitral valve is one of the more successful applications of three-dimensional echocardiography. Its greatest impact has been with the utilization of new real-time three-dimensional scanners, capable of providing real-time, subpyramidal imaging of the mitral valve from a perspective within the left atrium (Fig. 23.35). Experience suggests that real-time three-dimensional imaging confers an advantage with respect to complete evaluation of mitral valve pathology, including isolated mitral flail chordae and precise localization of flail scallops compared with routine, two-dimensional imaging, although the true clinical impact of this has yet to be demonstrated. Figures 23.36 to 23.38 were recorded in patients with mitral pathology and demonstrate the unique capabilities of this type of imaging. Similar images can be obtained from reconstructed images but are limited by artifacts inherent in stitching subvolumes and the fact that they do not provide true real-time images. Sophisticated on- and off-line analysis systems have been developed for quantitation of the three-dimensional mitral valve data set allowing determined quantitation of the actual amount of mitral valve tissue involved with a flail leaflet as well as the overall area of the mitral valve (Fig. 23.38). Experience suggests that this imaging technique confers substantial clinical value with respect to accuracy and speed of anatomical diagnosis, both before and following surgery for mitral valve disease.
FIGURE 23.33. Schematic of the mitral valve from multiple perspectives. **Bottom:** The mitral valve from the surgical perspective, from inside the left atrium. **Top:** The mitral valve as viewed from a traditional transthoracic parasternal short-axis view. **Middle:** The mitral valve is seen from a transesophageal approach at the midgastric level. In each instance, the proximal aorta is as noted in the schematic, as is the left atrial appendage. The three distinct scallops of the anterior (A1, A2, A3) and posterior (P1, P2, P3) leaflets are also schematized.
FIGURE 23.34. Transesophageal echocardiograms recorded in two ambulatory outpatients (A, upper panels) and in the operating room while intubated and under general anesthesia (B, lower panels). On the left, note the decrease in apparent severity of mitral regurgitation after institution of general anesthesia and on the right, the marked decrease in size of the left ventricular cavity and absence of previously noted moderate mitral regurgitation.
Video 23-34b
FIGURE 23.35. Real-time three-dimensional transesophageal image recorded in
a patient with a myxomatous mitral valve and mitral valve prolapse (systole, upper panel; diastole, lower panel). Because of the thickened myxomatous leaflets, the individual scallops are easily appreciated. This image is recorded from a “surgical perspective” with the anterior aspect at the top of the image. The individual scallops of the mitral valve are as noted (A₁, P₁, etc.) and the interscallop commissures also easily visualized (small arrows).

Video 23-35

**FIGURE 23.36.** Intraoperative transesophageal echocardiogram recorded in a young patient with severe functional mitral regurgitation related to a congenital abnormality of the mitral valve. A was recorded prior to repair. In the central figure note the dilated left atrium and the failure of coaptation of the mitral valve leaflets (arrows). The inset at the upper left is recorded with color Doppler and confirms the presence of severe functional mitral regurgitation. B was recorded after repair with an annular ring (arrows). Note the narrowing of the mitral annulus and the reduction in size of the posterior leaflet. The inset at the upper left was recorded with color Doppler and confirms absence of any significant residual regurgitation.
Video 23-36a CFD

coming soon

Video 23-36a

coming soon

Video 23-36a

coming soon
Video 23-36b CFD

coming soon

Video 23-36b
FIGURE 23.37. Real-time three-dimensional transesophageal imaging in a patient with myxomatous mitral valve disease and pronounced buckling of the posterior leaflet. This image was recorded from the perspective of the surgeon’s view within the left atrium. Note the large, bulky myxomatous posterior leaflet protruding into the left atrium in systole (arrows). The smaller inset is a real-time image of the same patient revealing the myxomatous posterior leaflet buckling into the left atrium (arrow). The intraoperative anatomy is also illustrated for comparison.
Video 23-37a

Video 23-37b
FIGURE 23.38. Intraoperative transesophageal echocardiogram recorded in a patient with mitral regurgitation related to myxomatous changes and mitral valve
prolapse. The upper panel is a real-time three-dimensional image in which thickened redundant leaflets with prolapse can be seen. The lower panel is a detailed anatomical reconstruction of the mitral annulus and mitral leaflets. The components encoded in red are prolapsing beyond the plane of the mitral annulus. The panel at the right depicts numerous detailed measurements of the magnitude of prolapsing tissue which are automatically calculated from the reconstructed model.

Video 23-38

Figures 23.39 through 23.41 were recorded in a patient with dilated cardiomyopathy and severe functional mitral regurgitation. The mitral valve is anatomically normal; however, there is failure of coaptation, resulting in severe mitral regurgitation. In this instance, the mechanism of regurgitation can be demonstrated to be apical displacement of the papillary muscles in the dilated and spherical left ventricle. Note the central location of the mitral regurgitation jet that arises at the area of noncoaptation of the mitral leaflets. This type of mitral regurgitation can be addressed by placement of an annular ring, which corrects the abnormal coaptation of the mitral valve. For patients with functional ischemic mitral regurgitation, due to restricted motion, of one or both mitral valve leaflets, a similar surgical approach is taken.

The patient illustrated in Figures 23.42 and 23.43 has mitral regurgitation due to a flail scallop of the posterior mitral valve leaflet. In this instance, regurgitation is due to anatomic disruption of the mitral valve and the repair will necessitate resection of the flail scallop with reapposition of the intact margins. For a posterior flail, the most common repair is resection of the redundant portion of the flail leaflet with reapproximation of the intact edges.
A mitral annular ring is then placed (Fig. 23.44). Depending on the initial pathology and the amount of resected valve tissue, this may result in the valve being converted to a nearly unicuspid valve with the anterior leaflet providing the majority of functional valve tissue. More complex repairs may include transposition of a portion of a leaflet and its attached chordae to the opposite leaflet to provide intact chordae to the previously flail leaflet. Finally, prosthetic chords can be attached to a flail mitral leaflet and subsequently to a papillary muscle to replace chordal structures that are damaged beyond repair. The goal of mitral valve repair is to reduce the severity of mitral regurgitation to no more than mild without creating iatrogenic mitral stenosis. In the examples presented, note the smaller annular dimensions due to an annular ring as well as the areas of thickening on the mitral valve that represent areas of resection. Figure 23.45 depicts intraoperative pre- and postoperative imaging in a patient with a flail A2 scallop.
FIGURE 23.39. Pre- and postoperative transesophageal echocardiograms recorded in a patient with left ventricular dysfunction and mitral regurgitation due to failure of mitral valve coaptation. Longitudinal views recorded at end-systole are presented. A: Prerepair, note the apical displacement of the mitral valve tips and the failure to coapt (arrows) in this systolic frame. The schematic in the upper left of (A) depicts the effect of apical and lateral tethering of the papillary muscles with incomplete valve coaptation. Normal coaptation is depicted in the lower schematic. B: Recorded after successful repair by placement of an annular ring (arrows). Video 23-39a

An infrequent method for repair of functional mitral valve regurgitation is to place an annular ring in conjunction with a stitch through the center of the anterior and posterior mitral valve leaflets to further restrict mobility (Alfieri
stitch). This results in limitation of motion of the mitral valve in diastole and more of a “figure 8” opening pattern in the short-axis view (Fig. 23.46).
FIGURE 23.40. Color Doppler flow images corresponding to the images presented in Figure 23.39 are shown. A: Note the severe mitral regurgitation arising centrally with the vena contracta location identified by the area of noncoaptation in Figure 23.40. B: A systolic frame recorded after placement of a mitral ring. Note the absence of mitral regurgitation after ring placement.

After surgical repair, it is important to determine the severity of any residual mitral regurgitation (Figs. 23.47 and 23.48). Although many investigators stress the importance of making this determination at normal systolic blood pressure, it should be emphasized that systolic blood pressure is not the only factor that alters the apparent severity of mitral regurgitation. Although restitution of normal blood pressure does not guarantee accuracy of
the assessment of regurgitation that may be present subsequently, it should be emphasized that the assessment of regurgitation should not be undertaken in patients who are overtly hypotensive or incompletely volume resuscitated.

Assessment of iatrogenic mitral stenosis is undertaken using pulsed- and continuous-wave Doppler. Ideally, after mitral valve repair, a mean gradient of 2 to 4 mm Hg will be present because of the narrowing effects of annular ring and the essential reduction in the total length and volume of mitral valve tissue. Transmitral gradients exceeding 5 or 6 mm Hg should be viewed as a possible indicator of iatrogenic stenosis. It should be emphasized that intraoperative hemodynamics may be misleading, especially if the patient is on inotropic support or significantly tachycardiac, which may increase transmitral gradients over what can be expected under basal conditions.
FIGURE 23.41. **A:** Continuous-wave Doppler image recording through the mitral orifice after placement of a mitral ring demonstrates a mean pressure gradient of 1.9 mm Hg after repair (same patient presented in Fig. 23.39). **B:** Recorded in a patient with a less ideal repair and a residual gradient of 6 mm Hg.
FIGURE 23.42. Intraoperative transesophageal echocardiogram recorded in a patient with myxomatous mitral valve disease and a flail posterior (P2) mitral valve. Note the distinct buckling of the mitral valve into the left atrium (arrows) and the highly eccentric mitral regurgitation jet on color flow Doppler image.
Video 23-42a

coming soon

Video 23-42b

coming soon
FIGURE 23.43. Real-time three-dimensional transesophageal echocardiographic imaging performed in the same patient depicted in Figure 23.42. This image is recorded from a surgeon’s perspective within the left atrium with anterior structures at the top of the image. Note the myxomatous portion of the midposterior leaflet buckling into the left atrium (arrows) and the evidence of a thickened flail chord (small arrow). The lower panel was recorded after repair with a three-quarter circumferential annuloplasty ring (arrows). Note on the postoperative Doppler image, a mean transvalvular pressure gradient of 3 mm Hg.
FIGURE 23.44. Intraoperative two-dimensional transesophageal echocardiogram recorded after implantation of an annuloplasty ring in the same patient depicted in Figure 23.37 who presented with a flail posterior leaflet. In the 0-degree view, note the ring in the lateral mitral annulus and the apparent absence of functional posterior leaflet tissue. In the 61-degree view, two edges of the ring are clearly visualized as are portions of the anterior and posterior leaflets (arrows).
Video 23-44a

coming soon

Video 23-44b

coming soon
FIGURE 23.45. Post-operative real-time three-dimensional echocardiogram recorded in the same patient depicted in Figure 23.37. Note the partially circumferential annuloplasty ring (small arrows) and the myxomatous redundant anterior leaflet in the diastolic frame. The small inset is the post-operative surgical image photographed in the operating room in which the annuloplasty ring and sutures in the posterior leaflet of the mitral valve from the resection of the prolapsing leaflet tissue are seen.

Video 23-45

In addition to evaluating the feasibility of repair in native valve disease, transesophageal echocardiography can also assist in determining the feasibility of a repeat repair in patients who have previously undergone a
mitral valve procedure. If failure of the repair is due to problems with structural integrity of the mitral valve itself, it is unlikely that a repeat repair will provide a durable benefit. Conversely, if it is due to a technical problem with a mitral valve ring, a repeat surgical procedure may be beneficial. Figures 23.49 and 23.50 were recorded in a patient who had previously undergone a successful mitral valve repair for a flail posterior leaflet and had an excellent symptomatic response. Three months postoperatively, he developed recurrent symptoms and was noted to have mitral regurgitation on physical examination. Note from the baseline images that there is dehiscence of annular ring from the lateral mitral annulus. This allowed apical and lateral displacement of the papillary muscles to interfere with normal mitral coaptation and resulted in significant functional mitral regurgitation, as can be seen in Figure 23.50. In this instance, the mitral annular ring was resuspended, recreating normal coaptation of the mitral valve (Fig. 23.49) and completely eliminating mitral regurgitation.

FIGURE 23.46. Real-time three-dimensional transesophageal image recorded in a patient after mitral valve repair which included a mitral valve ring as well as an Alfieri stitch (arrow). In the central image the ring is noted by the arrows. This
image was recorded in diastole and reveals a “double barrel” mitral orifice with each orifice noted by the “x.” The panel at the upper left is recorded with color Doppler and demonstrates the dual left ventricular inflow from the left atrium (arrows). Video 23-46
FIGURE 23.47. Transesophageal echocardiogram recorded immediately after implantation of a mitral annular ring (arrows) in a patient with a flail posterior leaflet. These images are from the same patient depicted in Figure 23.37. In the upper diastolic panel, note the absence of any significant flow convergence, implying nonrestricted transmitial flow and in the lower systolic panel, the presence of only trivial mitral regurgitation immediately behind the closed mitral leaflets (arrow). The Doppler inset confirms a mean transvalvular gradient of 2 mm Hg consistent with excellent technical result.
FIGURE 23.48. Intraoperative transesophageal echocardiogram recorded immediately following an attempt at repair of a myxomatous mitral valve with an annuloplasty ring (upper panel). Note the significant residual mitral regurgitation necessitating returning the patient onto cardiopulmonary bypass for a mitral valve replacement (lower panel). Note, in this patient with annular calcification, the small residual paravalvular leak around the edge of the prosthetic mitral valve (large arrow).
Video 23-48a

coming soon

Video 23-48b
FIGURE 23.49. Transesophageal echocardiogram recorded in the horizontal
plane in a patient who had previously undergone mitral repair with an annular ring. **A:** Note the separation between the lateral mitral annulus and the ring consistent with dehiscence. This has resulted in severe functional mitral regurgitation as can be seen in Figure 23.50. **B:** Recorded after re-repair and demonstrates that the ring is now reattached to the annulus (arrows) with improved leaflet coaptation.

Real-time three-dimensional echocardiography combined with two-dimensional color flow imaging either immediately postoperatively in the operating room or as part of a postoperative evaluation for suspected complications is incrementally valuable for identification of dehiscence of a mitral ring or prosthetic mitral valve. Depending on the size of the area of dehiscence and its location routine, two-dimensional imaging is often
diagnostic. Either reconstructed or (preferably) real-time three-dimensional imaging from a transesophageal approach provides a high-resolution view of the full circumference of an annular ring or sewing ring of a prosthetic valve and is an accurate method for localization and quantitation of areas of dehiscence (Figs. 23.51 to 23.53).

There are several other complications of mitral valve repair. In patients with redundant mitral valve tissue and a normally contracting or hyperdynamic left ventricle, placement of the mitral ring along with reduction in left ventricular volume may allow systolic anterior motion of the residual mitral valve tissue into the left ventricular outflow tract (Fig. 23.54). Mild degrees of systolic anterior motion are not uncommon, especially in patients receiving inotropic therapy. If there is evidence of significant mitral regurgitation or outflow tract obstruction, further evaluation is usually necessary. This syndrome can result in significant dynamic outflow obstruction, mimicking hypertrophic cardiomyopathy. Mitral regurgitation may be induced as part of this syndrome as well. If outflow obstruction is significant and does not respond to volume resuscitation and reduction in inotropic therapy (Fig. 23.55), modification of the surgical repair or placement of a prosthetic valve may be necessary.
FIGURE 23.50. Intraoperative transesophageal echocardiogram recorded in the
same patient depicted in Figure 23.49. **A:** Recorded before repeat repair and demonstrates severe mitral regurgitation due to apical displacement of the mitral valve. Note the location of the convergence zone well into the left ventricular cavity (arrow). **B:** Recorded after repair and confirms the absence of residual mitral regurgitation.

Although intraoperative transesophageal echocardiography is employed in most patients undergoing mitral valve repair, it is also used in patients undergoing replacement with a prosthetic valve. For patients who are undergoing repeat mitral valve replacement, for those in whom the initial indication for valve replacement is paravalvular regurgitation or endocarditis, or in patients with a heavily calcified mitral annulus, intraoperative
transesophageal echocardiography is often used to confirm the successful “seating” of the valve ring within the annulus and to confirm that there is no paravalvular mitral regurgitation (Fig. 23.56).

In many institutions, mitral valve replacement with a prosthetic valve is accomplished by leaving the posterior mitral valve leaflet and the associated papillary muscle and chordae intact. This protects against adverse remodeling of the left ventricle, which can be seen if the entire apparatus is severed. A potential complication of leaving residual mitral valve tissue is that the retained chordae can interrupt function of a disc prosthesis. This is a complication that can be screened for intraoperatively but unfortunately may be a delayed development rather than an immediate postoperative complication.
FIGURE 23.51. Real-time three-dimensional transesophageal echocardiogram recorded in a patient following implantation of a stented bioprosthesis in the mitral position. **A:** Recorded from a perspective within the left ventricle (looking toward the left atrium). Notice the three struts of the stented bioprosthesis (*white arrows*) and the small, crescent-like area of dehiscence of the sewing ring at its inferomedial aspect (*small dark arrows*). Also note in this view, the left ventricular outflow tract, which should not be confused for an additional larger area of dehiscence. **B:** In the two-dimensional color flow Doppler image, notice the small, highly eccentric medially located paravalvular mitral regurgitation jet (*small arrows*). The *larger arrows* depict the outer boundary of the sewing ring.
coming soon

Video 23-51a

coming soon

Video 23-51b
FIGURE 23.52. Real-time three-dimensional transesophageal imaging recorded in a patient following implantation of a bileaflet mechanical mitral prosthesis. This image is recorded from a perspective within the left atrium and rotated to match the two-dimensional image. Note the lack of seating of the mitral ring in the lateral aspect of the sewing ring (black arrows). In the lower panel, recorded with color Doppler, note the eccentric mitral regurgitation jet arising from the area of dehiscence (white arrow).
FIGURE 23.53. Reconstructed three-dimensional echocardiographic image with color flow Doppler recorded in the same patient depicted in Figure 23.52 (upper panel). Notice the mitral regurgitation arising from the lateral aspect of the sewing ring. The lower panel is an en face view of the actual regurgitant orifice.
TRICUSPID VALVE REPAIR

Tricuspid valve repair for functional or organic tricuspid regurgitation is less commonly performed and techniques are in evolution. Current opinion is that concurrent tricuspid valve repair should be considered at the time of other valvular or coronary surgery if moderate or greater tricuspid regurgitation is present. The role of tricuspid regurgitation repair as an isolated procedure is less well established, but there is a trend toward a more aggressive surgical approach to isolated moderate and severe tricuspid regurgitation. Many of the techniques used are similar to those for the mitral valve, and
echocardiographic imaging plays a similar role. Investigational procedures and devices for percutaneous approaches to functional tricuspid regurgitation are also being evaluated.

**FIGURE 23.54.** Intraoperative transesophageal echocardiogram recorded in a patient following placement of a mitral annular ring for repair of severe mitral regurgitation. This split screen image was recorded at midsystole. In the image on the left note the systolic anterior motion of the mitral valve (*leftward-pointing arrow*). This has caused malcoaptation of the leaflets with separation of the anterior and posterior leaflets (*downward-pointing arrow*) resulting in mitral regurgitation. The image on the right was recorded with color Doppler and reveals moderate to severe mitral regurgitation. The small inset is a continuous-wave Doppler through the left ventricular outflow tract demonstrating an outflow tract gradient of approximately 45 mm Hg.
coming soon

Video 23-54
FIGURE 23.55. Color Doppler recorded in the operating room immediately following mitral valve repair with an annuloplasty ring. The upper panel (A) was recorded immediately postoperatively and demonstrates turbulence in the outflow tract as well as moderate mitral regurgitation. The lower panel (B) was recorded in the same patient after full volume resuscitation and demonstrates the resolution of the systolic anterior motion and mitral regurgitation.
For patients undergoing tricuspid valve annuloplasty or replacement with a prosthetic valve most of the same considerations discussed for mitral valve procedures pertain. There are several techniques for tricuspid annuloplasty, including placement of a prosthetic ring and less frequently the De Vega annuloplasty, in which the tricuspid annulus is essentially “cinched” with a series of sutures around its circumference. For each technique the procedure typically avoids placement of sutures along the ventricular septal aspect of the tricuspid annulus to avoid injury to the conducting system. As such, the visualized annuloplasty may appear incomplete (Fig. 23.57). The majority of tricuspid annuloplasty procedures are performed for functional tricuspid regurgitation which, depending on the severity of underlying right ventricular
dysfunction, may be corrected with variable success. Greater degrees of residual tricuspid regurgitation are often seen following a tricuspid annuloplasty than following mitral valve repair.

**Role of Echocardiography in Aortic Valve Procedures**

Transesophageal echocardiography plays less of a role in conventional aortic valve procedures than it does in mitral valve procedures. It provides incremental clinical information, including determination of the feasibility of aortic valve repair and in sizing the aortic annulus and proximal aorta for placement of some of the newer bioprostheses. Obviously, intraoperative echocardiography can detect residual regurgitation and the majority of procedure-related complications. Transcatheter aortic valve replacement (TAVR) has become standard of care for an increasing number of patients with aortic valve disease. The role of echocardiography in assessing patients for suitability for TAVR as well as intraprocedure monitoring and post procedure follow-up is discussed in Chapter 13 which deals with prosthetic valves.
FIGURE 23.56. Pre- and postoperative echocardiograms recorded in a patient with left ventricular hypertrophy and heavy annular calcification resulting in severe mitral stenosis. A: Preoperative apical four-chamber view reveals left ventricular hypertrophy and marked calcification of the mitral annulus (arrows). Note the significant transmitral gradient with a mean pressure gradient of 20 mm Hg (inset). B: A transesophageal echocardiogram revealing moderate to severe
paravalvular mitral regurgitation (*arrow*) due to lack of complete seating of the prosthetic valve ring in the calcified mitral annulus.

Video 23-56a

Video 23-56b

When considering a patient for aortic valve replacement, the size of the left ventricular outflow tract and the degree, if any, of subvalvular septal hypertrophy have a direct bearing on the surgical technique. The need for an outflow tract widening procedure or concurrent myectomy increases the complexity and risk of aortic valve replacement. As part of the assessment of a patient for aortic valve replacement, it is essential to evaluate the proximal ascending aorta for significant dilation and the need for a concurrent aortic root procedure.
The aortic valve is less amenable to repair than is the mitral valve. Aortic valve repair has been attempted for at least five decades and has met with variable success. Typically, a repairable aortic valve will have regurgitation due to a limited perforation or prolapse of one cusp edge. If a perforation is present, a small pericardial patch can be placed successfully in many instances. If there is malcoaptation due to prolapse of an edge, this can often be surgically approached by resecting a small wedge of tissue and then placing buttressing sutures in the proximal commissures to effectively shorten the coaptation line. Patients with regurgitation due to marked valve destruction secondary to endocarditis, aortic insufficiency coexisting with aortic stenosis, or advanced fibrosis of a bicuspid leaflet are not candidates for repair. Figure 23.58 was recorded in a patient with aortic insufficiency for whom aortic valve repair was performed. Notice that there is an isolated perforation that is amenable to repair. The patient presented in Figure 23.59 has an unremarkable three-leaflet valve with a central aortic insufficiency jet due to malcoaptation related to aneurysmal dilation of the ascending aorta. In this setting, replacement of the aortic root and restitution of normal sinotubular geometry allowed normal aortic cusp coaptation and resolution of aortic regurgitation.
FIGURE 23.57. Intraoperative transesophageal echocardiogram recorded in a patient following placement of a prosthetic ring in the tricuspid annulus for severe functional tricuspid regurgitation. In the 1-degree view, note the portion of the prosthetic ring in the lateral annulus (arrow) and the absence of any prosthetic material at the ventricular septum (small arrow). In the 126-degree view, note the appearance of a prosthetic ring (arrows) narrowing the tricuspid annulus and in the color Doppler image (inset), the significant residual tricuspid regurgitation related to ongoing functional disturbances of the tricuspid apparatus and right ventricle.
Video 23-57a

coming soon

Video 23-57b

coming soon

Video 23-57b

coming soon
FIGURE 23.58. Transesophageal echocardiogram recorded in a longitudinal view in a patient with a small aortic valve perforation due to healed endocarditis. Note the area of focal thickening on the noncoronary cusp (arrow) and the aortic regurgitation jet arising from a perforation in the area of the healed vegetation. Limited cusp disruption such as depicted here constitutes favorable anatomy for aortic valve repair.
**FIGURE 23.59.** Intraoperative transesophageal echocardiograms recorded in a patient with marked dilation of the sinuses of Valsalva and severe functional aortic insufficiency. A was recorded preoperatively. In the two-dimensional image note the malcoaptation of the aortic cusps (arrow) and the resultant severe aortic
insufficiency. The inset at the bottom is a short-axis view of the aorta demonstrating the functional aortic insufficiency. B was recorded following repair of the aortic root. Notice the relatively normal relationship of the sinuses to the rest of the aorta. Also note the absence of residual aortic insufficiency.
An additional area in which feasibility of repair can be determined with transesophageal echocardiography is aortic insufficiency associated with type A aortic dissection or disease of the aortic root. In the presence of aortic dissection, there are multiple etiologies for aortic insufficiency (Fig. 23.60). Aortic insufficiency due to dilation of the sinotubular junction often can be corrected with a valve-sparing procedure as can aortic insufficiency due to disruption of the base of the aortic cusp secondary to dissection propagating toward the annulus. Figures 23.61 through 23.63 were recorded in patients in whom a valve-sparing procedure could be performed at the time of aortic dissection repair.

Transesophageal echocardiography is often used for intraoperative decision making in the evaluation of patients for placement of newer bioprostheses, including cryopreserved homografts and stentless porcine prostheses. The human homograft includes the donor annulus, aortic cusps, and ascending aorta and must be sized to the recipient heart. Because homograft valves are in short supply and once thawed cannot be refrozen, it is important to ensure a good match between the available homograft valve and the patient under consideration. This is typically done with the preoperative transesophageal echocardiogram.

After implantation, the echocardiographer should appreciate the full range of appearance of these newer valves. Depending on the implantation technique, the valve may be indistinguishable from a normal native valve or there may be substantial areas of excess fluid and tissue accumulation when
the valve is implanted with an inclusion technique. In this technique, the bioprosthesis is implanted within the native recipient aorta, resulting in a double-density wall to the aorta, either circumferentially or more commonly localized to the noncoronary cusp area. Figures 23.64 and 23.65 were recorded in a patient with aortic insufficiency related to endocarditis who underwent implantation of a stentless porcine prosthesis. Notice the double density of the aortic wall in the noncoronary cusp area and the soft tissue and fluid collection between the donor and recipient aortic walls, mimicking an aortic abscess in the postoperative images (Fig. 23.65). Commonly, a small paravalvular leak may be noted at the anastomosis to the aortic annulus. These minor leaks generally seal off after administration of protamine in the operating room (Fig. 23.66). There is a broad range of appearance immediately after surgery. Typically, 3 to 6 weeks after this type of implantation, the free space between the bioprosthesis and the recipient aorta has been obliterated and the appearance of the wall is markedly changed (Fig. 23.67). In the long term, these valves may be nearly indistinguishable from a native aortic valve.

**FIGURE 23.60.** Multiple mechanisms can be responsible for aortic insufficiency in aortic dissection including effacement or dilation of the sinotubular junction resulting in malcoaptation of the aortic valve (A), aortic dissection in the presence of intrinsic aortic valve disease (B), disruption of the insertion of an aortic cusp (C), and prolapse of the intimal dissection flap through the aortic valve, which serves as a conduit for aortic regurgitation (D).
FIGURE 23.61. Intraoperative transesophageal echocardiogram recorded in a patient with an extensive acute type A aortic dissection. In the central image note the dilation of the aorta and the complex intimal flap (longer arrows). The aortic valve is noted by the shorter arrow. The inset at the upper left demonstrates the severe aortic insufficiency related to acute aortic dissection. [Video 23-61a]

Video 23-61a
FIGURE 23.62. Intraoperative transesophageal echocardiogram recorded in the same patient presented in Figure 23.61. Note the reduction in size in the more distal portions of the aorta (black arrows) and the unremarkable aortic cusps. The inset at the upper left was recorded in diastole with color Doppler and reveals only mild residual aortic insufficiency.
FIGURE 23.63. Intraoperative transesophageal echocardiogram recorded in a patient presenting with an acute type A intramural hematoma. The extent of hematoma is relatively limited and largely confined to the area of the right coronary sinus (arrows). There is a combination of acute and pre-existing dilation of the aorta at the sinotubular junction resulting in functional aortic insufficiency. The inset at the upper left was recorded in the operating room after repair of the intramural hematoma and demonstrates absence of aortic insufficiency. Video 23-63 post
FIGURE 23.64. Preoperative transesophageal echocardiogram recorded in a young patient with aortic valve endocarditis and severe aortic insufficiency. A: Notice the normal contour of the ascending aorta and absence of complications such as annular abscess. This is thickening and cusp disruption (arrow). B: Recorded with color flow Doppler revealing severe aortic regurgitation is a disorganized jet.
Video 23-64a

coming soon

Video 23-64b

coming soon
FIGURE 23.65. Intraoperative transesophageal echocardiogram recorded immediately after replacement of the aortic valve in the patient depicted in Figure 23.64. In this instance, a stentless aortic valve prosthesis has been implanted. Note the 1-cm thick echo density along the posterior wall of the aorta adjacent to the left atrium. This echo density is a result of the surgical technique and does not represent pathologic hematoma or abscess. Note on the spectral Doppler image, the peak gradient through the aortic valve of 6 mm Hg consistent with an excellent technical result.

Video 23-65
FIGURE 23.66. Intraoperative transesophageal echocardiogram recorded in a patient immediately following aortic valve replacement with a stentless bioprosthesis. A: Recorded immediately after cessation of cardiopulmonary
bypass and a small paravalvular leak (arrow) is noted at the posterior suture line between the native aorta and the prosthetic valve. B: Recorded 10 minutes later after protamine therapy and reveals resolution of the paravalvular leak.

One complication of aortic valve replacement, especially if a concurrent myectomy or annular enlarging procedure has been performed, is ventricular septal defect (VSD). Because the defect may be small and located underneath the sewing ring, it may be difficult for a surgeon to directly visualize. Postoperative transesophageal echocardiography typically can accurately identify these defects and allow the surgeon to make a decision regarding the necessity of returning the patient to cardiopulmonary bypass for further repair (Fig. 23.68). In addition to screening for iatrogenic VSD, intraoperative
transesophageal echocardiography can be used to confirm the success of VSD repair of other etiologies. Figure 23.69 was recorded in a patient with an apically located postinfarct VSD and demonstrates both pre- and postrepair transesophageal echocardiograms recorded in the operating room.

A complication unique to septal myectomy is the creation of a coronary fistula in the area of the myectomy (Fig. 23.70). As a consequence of surgical excision of septal muscle, an intramural coronary artery may be sheared off and result in a fistula in the left ventricular outflow tract. These are not noted at the time of the actual procedure because the patient is on cardiopulmonary bypass and the heart is arrested. Such fistula is rarely of clinical concern but has been mistaken for paravalvular aortic regurgitation in patients in whom concurrent aortic valve replacement has been performed.

A rare complication of an aortic valve replacement especially if complex and associated with an annular widening procedure or myectomy is inadvertent trauma to the proximal anterior mitral valve. This can result in a perforation and mitral regurgitation. Figure 23.71 was recorded in a patient with a mitral valve perforation related to a complex aortic valve procedure. The postoperative transesophageal echocardiogram clearly demonstrates a proximal mitral regurgitation jet, not noted in the preoperative examination. This is also demonstrated in the real-time three-dimensional images which allow precise characterization of the size of the perforation.
FIGURE 23.67. Transesophageal echocardiograms recorded in a patient after aortic valve replacement using a stentless aortic valve prosthesis and an “inclusion” technique. A: Note the free space between the prosthesis wall and the native aorta in this intraoperative echocardiogram. B: Over time, this free space disappears and there will be smooth continuity between the included prosthesis and the native aortic wall as noted on a transesophageal echocardiogram recorded 6 weeks later.
coming soon

Video 23-67a

coming soon

Video 23-67b
FIGURE 23.68. Intraoperative transesophageal echocardiogram recorded in a patient following aortic valve replacement and septal myectomy. A: Recorded immediately following the procedure and reveals a ventricular septal defect just below the level of the prosthetic valve (arrows). B: The patient was returned to pump for correction of ventricular septal defect after which was recorded confirming absence of residual shunt.
FIGURE 23.69. Intraoperative transesophageal echocardiogram recorded in the operating room before (A) and after (B) closure of a post infarct ventricular septal defect. Both sets of images are recorded with a split screen format with standard imaging on the left and color Doppler on the right. In the preoperative image note
the suggestion of breakdown of tissue at the apical septum (arrow) and the color Doppler flow diagnostic of a ventricular septal defect. In the postoperative image note the residual dilation of the right ventricle. The patch can be seen in the left ventricular apex (arrows). Also note the minimal residual flow around the age of the patch (arrow) but absence of communication into the right ventricle.

An uncommon sequela following aortic valve replacement for severe aortic stenosis is the development of dynamic outflow tract obstruction (Fig. 23.72). This complication is easily detected with intraoperative transesophageal echocardiography. There is a subset of patients, typically female and elderly, who have severe aortic stenosis, marked left ventricular hypertrophy, and a small left ventricular cavity. After relief of the afterload
obstruction due to critical aortic stenosis, the ventricle becomes hyperdynamic and a pattern mimicking hypertrophic cardiomyopathy of the elderly is unmasked. This can result in hemodynamic instability and hypotension in the perioperative period. Transesophageal echocardiography can identify this syndrome when there is evidence of a small hyperdynamic left ventricle with dynamic outflow tract obstruction, with or without secondary mitral regurgitation, after aortic valve replacement. Some investigators have suggested that the combination of a small preoperative cavity with marked left ventricular hypertrophy accurately predicts development of this syndrome and that the surgical procedure should include not only aortic valve replacement but also proximal septal myectomy.

**FIGURE 23.70.** Intraoperative transesophageal echocardiogram recorded in a patient with hypertrophic cardiomyopathy immediately after surgical myectomy. Incidental note is made of mild residual aortic insufficiency (*upward-pointing arrow*). There is a color Doppler signal originating from the proximal septum here (*horizontal arrow*) which represents a small coronary fistula related to severing of a small intramyocardial vessel at the time of myectomy.
Video 23-70
FIGURE 23.71. Transesophageal echocardiogram recorded in a patient after a complex procedure for severe calcific aortic stenosis with a small aortic annulus and marked proximal septal hypertrophy. The procedure necessitated a myectomy and an annular enlarging procedure. The postoperative echocardiogram revealed moderate mitral regurgitation which in the routine, two-dimensional image (A) is arising from the base of the anterior leaflet (arrow). The larger, three-dimensional image (B) confirms the localization of this jet to be a perforation of the proximal anterior leaflet (arrows). The smaller inset is an en face view of the color flow Doppler image in which the actual perforation dimension can be appreciated (black arrows).
FIGURE 23.72. Transesophageal echocardiogram recorded 24 hours after otherwise uncomplicated aortic valve replacement with a stentless prosthesis. The patient developed hypotension and a variable systolic murmur. A was recorded in a split screen format with standard imaging on the left and color Doppler on the right. In this systolic frame note the systolic anterior motion of the mitral valve (arrow) and a relatively small left ventricular cavity with septal hypertrophy. The color Doppler image demonstrates systolic turbulence. B is a Doppler recording demonstrating a variable left ventricular outflow tract dynamic gradient (arrows) which following a premature beat exceeds 50 mm Hg.

MISCELLANEOUS APPLICATIONS

There are several miscellaneous applications of echocardiography in the operating room, including assessment of aortic atherosclerosis and assisting with placement of cannulae or other devices. When operating on patients with atherosclerotic heart or vascular disease, it is not uncommon to
encounter significant amounts of aortic atherosclerosis. These areas are
preferably avoided when placing aortic cannulae or cross clamps. Most
surgeons rely on palpation of the aorta to assess for underlying atheroma at
the site of intended cannulation or device placement. Transesophageal or, less
often, direct epicardial imaging can be used to evaluate the location and
extent of underlying atheroma before instrumenting the aorta at that location
(Fig. 23.73).

Other Intraoperative Observations Following Cardiac Surgery

The echocardiographer involved in intraoperative evaluation should be aware
of the full range of cardiac complications that can occur at the time of
cardiovascular surgery (Table 23.4). One consideration after cardiac surgery
is the presence and location of any residual intracardiac air. Individual small
contrast targets indicative of small bubbles (similar in size and character to
those seen during saline contrast injection) are not uncommon after any
cardiac procedure. Intracardiac air can take on one of three appearances. It
should be recalled that air is an intense echo reflector, and a significant
pocket of air will result in both acoustic shadowing and reverberation. As
such, the appearance of air may be that of a single bright linear line with
shadowing behind it. It should also be emphasized that air will float to the
surface of the blood pool, and, as such, a free air collection is expected in the
more anterior locations (assuming supine patient position). The most
common sites to find significant pockets of residual air are the left atrial
appendage, the pulmonary veins (Figs. 23.74 and 23.75), and along the atrial
wall (Fig. 23.76). If intracavitary, this type of echo represents a variable but
typically clinically relevant amount of intracardiac air that should be
identified and addressed before the patient is removed from cardiopulmonary
bypass. Often, the visualized air pocket may be trapped between two cardiac
structures (intercardiac), such as the aorta and left atrial wall, as opposed to
being contained within a cardiac chamber (intracardiac). It may not be
possible to distinguish extracardiac air from true intracavity air. If it is not in
an anterior location and does not migrate with physical motion, it is more
likely to be noncavitary and, as such, of little clinical relevance.

A second appearance of intracardiac air is that of an air embolus to the
myocardium. This typically will present as a bright white area in the ventricular myocardium, much as would be expected during myocardial perfusion echocardiography (Fig. 23.77). This may result in reduced myocardial blood flow and a regional wall motion abnormality. A related phenomenon is an air embolus to a major coronary artery that results in interruption of flow, resulting in a wall motion abnormality but not in diffuse air accumulation within the myocardium. This is typically identified as a regional wall motion abnormality, corresponding to a coronary distribution but without evidence of air in the myocardium. Obviously, this appearance could be due to any other factor resulting in interruption of coronary blood flow.
FIGURE 23.73. A: Transesophageal echocardiogram recorded in a patient being considered for coronary artery bypass surgery and mitral valve repair. Note the atheroma throughout the arch of the aorta (arrow), which would likely complicate cannula placement at this location. The mobility and complexity of the atheroma are best appreciated in the video images and in the real-time three-dimensional image (B).
Table 23.4  CARDIAC SURGERY COMPLICATIONS DETECTED WITH INTRAOPERATIVE TRANSESOPHAGEAL ECHOCARDIOGRAPHY

<table>
<thead>
<tr>
<th>“Intracardiac” air</th>
<th>Intracavitary</th>
<th>Intercavitary</th>
<th>Myocardial</th>
<th>Individual targets</th>
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<tbody>
<tr>
<td>Right ventricular dysfunction</td>
<td>Left ventricular dysfunction</td>
<td>Regional</td>
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<td>Global</td>
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<td>Following myectomy</td>
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<td>Ventricular septal defect</td>
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<td>Residual obstruction</td>
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<tr>
<td>Coronary fistula</td>
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<tr>
<td>Following aortic valve replacement</td>
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<tr>
<td>Paravalvular regurgitation</td>
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<td>Patient-prosthesis mismatch</td>
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<td>Outflow tract obstruction</td>
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<td>Following mitral valve repair</td>
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<tr>
<td>Residual mitral regurgitation</td>
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<tr>
<td>Iatrogenic mitral stenosis</td>
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<tr>
<td>Systolic anterior motion–dynamic outflow obstruction</td>
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<tr>
<td>Following mitral valve replacement</td>
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<tr>
<td>Paravalvular regurgitation</td>
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<td>Chordal interference with disc function</td>
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<td>Following repair of congenital heart lesions</td>
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<td>Residual shunt</td>
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<td>Baffle integrity</td>
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<tr>
<td>Right ventricular function</td>
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FIGURE 23.74. Intraoperative transesophageal echocardiogram recorded immediately following mitral valve repair. Note the bright, oscillating echo (arrow) within the left upper pulmonary vein (LUPV) due to retained air.
FIGURE 23.75. Two-dimensional echocardiogram recorded in a patient following mitral valve surgery. Note the air in the area of the left atrial appendage, some of which is being expressed into the body of the left atrium. In the real-time image, note the erratic motion in the area of the left atrial appendage which is the result of manipulation by the surgeon in an effort to expel all remaining air. LAA, left atrial appendage.
FIGURE 23.76. Intraoperative transesophageal echocardiogram recorded after mitral valve surgery. In this longitudinal view, note the discrete air “pocket” (arrow) in the left atrium resulting in distal shadowing. Also note the side lobe artifact arising from the main target. In the real-time image, note the oscillatory nature of this signal, confirming that it as an air pocket. In this instance, the air pocket is located anteriorly, which would be the anticipated area of free intracavitary air.

Video 23-76 MOV
Intracardiac air may also present as small pockets of air trapped within trabeculae at the left ventricular or right ventricular apex. This will be seen almost exclusively after valve procedures in which the cavity of the left ventricle has been emptied and exposed to air. Typically, if air is in this location, physical motion of the heart in that area by the surgeon may dislodge at least a portion of it and confirm its nature as being trapped in trabeculae rather than being truly intramyocardial.

Additional cardiac complications that can be screened for with intraoperative transesophageal echocardiography include the development of new or worsening left or right ventricular systolic function. Because the right ventricle is more exposed and less well preserved by cardioplegia, some degree of right ventricular dysfunction is common after cardiac surgery. The severity and likelihood of right ventricular dysfunction are directly related to
the complexity and duration of the procedure. On occasion, degrees of right ventricular dysfunction that may interfere with overall cardiac output are encountered. Right ventricular dysfunction of the transplanted heart is a worrisome complication because these patients frequently have a degree of pulmonary hypertension that exacerbates the right ventricular dysfunction occurring as a result of the transplantation procedure. Intraoperative transesophageal echocardiography is extremely useful for detecting and following the progress of this complication. Figure 23.78 was recorded in a patient in whom preoperative right ventricular function was normal, but, after a lengthy operative course, the right ventricle became dilated and hypokinetic. Right ventricular dysfunction in this setting may be transient and variable degrees of recovery are common. A secondary complication that can be seen in patients who develop right ventricular dysfunction is opening of a PFO resulting in right-to-left shunting and systemic hypoxia. This syndrome can lead to difficulty in weaning from ventilatory support and on occasion has required surgical or percutaneous closure. This complication can be screened for both in the operating room and in the perioperative period in the intensive care unit with saline contrast.

Left ventricular function should be reassessed immediately after cardiac procedures. Systolic dysfunction can occur either regionally, where it is assumed to be the result of interruption of flow to a coronary artery territory, or globally, which can have several etiologies. Figure 23.79 was recorded in a patient after a cardiac surgical procedure in whom global right and left ventricular dysfunction was noted immediately postoperatively. When a regional abnormality is noted, corresponding to a well-defined coronary circulation, direct visual inspection for the integrity of bypass grafts to ensure that there is no kink or other anatomic disruption is clearly in order (Fig. 23.80).
FIGURE 23.78. Intraoperative transesophageal echocardiogram recorded in a patient after aortic valve replacement and bypass surgery in whom there was difficulty weaning from cardiopulmonary bypass. Note the small, underfilled left ventricle and the markedly dilated right ventricle and right atrium due to severe right ventricular dysfunction. Also note the persistent bulging of the atrial septum into the left atrium (arrows), indicative of markedly elevated right atrial pressures.

Video 23-78
FIGURE 23.79. Intraoperative transesophageal echocardiogram recorded immediately after removing a patient from cardiopulmonary bypass after an otherwise uncomplicated procedure. Note the marked dilation of the left ventricle and the severe global hypokinesis representing severe diffuse myocardial stunning.

Video 23-79

A final intraoperative complication that should be considered is that of iatrogenic aortic dissection. This typically occurs at the site of aortic cannulation. As part of the postoperative evaluation, the ascending aorta, arch, and descending thoracic aorta should be screened for integrity of the
wall and to ensure that there is no evidence of iatrogenic dissection. Iatrogenic dissection may be limited and clinically silent but on occasion may result in major organ malperfusion with inadequate urine output or bowel or limb ischemia. Figure 23.81 was recorded in a patient after otherwise uncomplicated mitral valve repair and coronary artery bypass surgery. During the course of the surgical procedure, the patient became progressively oliguric, and postoperative transesophageal echocardiography demonstrated a new aortic dissection extending from the arch to beyond the gastroesophageal junction.

**Intraoperative Monitoring for Noncardiac Procedures**

Transesophageal echocardiography is occasionally used to monitor cardiac function during noncardiac procedures. Typically, this has been confined to patients undergoing high-risk procedures such as liver transplantation or major vascular procedures. Echocardiographic monitoring is most often used for determination of left ventricular systolic function and volume status. It should be emphasized that in patients with significant hypertension and coexistent organic heart disease, right heart catheterization may not provide an accurate assessment of true ventricular preload. Because of left ventricular hypertrophy and diastolic dysfunction, left ventricular filling pressures as measured by right heart catheterization are high; however, the ventricle itself is small and underfilled, resulting in reduced stroke volume, hypotension, and poor perfusion. Recognition of this pattern of a small, underfilled ventricle in the presence of hypotension with elevated filling pressure by right heart catheterization allows the appropriate therapy of volume resuscitation to be undertaken rather than the inappropriate maneuver of combined diuretics and pressors.
FIGURE 23.80. Intraoperative transesophageal echocardiogram recorded in a patient after multivessel coronary artery bypass surgery. Preoperatively, the patient had normal left ventricular systolic function. In this image, recorded immediately after discontinuation of cardiopulmonary bypass, note the significant area of inferior wall akinesis (arrows) suggesting graft failure. 

Video 23-80
FIGURE 23.81. Intraoperative transesophageal echocardiogram recorded in a patient immediately after cardiac surgery. Note the dissection in the arch (arrows) (A) and the position of the cannula at the origin of the dissection flap (arrows) (B).
Postoperative Cardiac Surgery Complications

Echocardiography is instrumental in evaluating the cause of deterioration in a patient’s clinical status after cardiac surgery. The differential of this is extensive and includes pericardial fluid and hemorrhage and failure of a valve repair or replacement. Additionally, attention should be paid to the possibility of early bypass graft closure, in which case one anticipates seeing a regional wall motion abnormality conforming to the distribution of either a native vessel or one of the implanted grafts. A not uncommon complication of cardiac surgery of any type is right ventricular systolic dysfunction, which may either be mild and transient, or more profound and a limiting factor to overall cardiovascular hemodynamics. On occasion, it may be refractory to conservative measures and be severe enough as to warrant placement of a right ventricular assist device. Postoperative right ventricular systolic dysfunction has multiple etiologies, including postoperative pulmonary hypertension as well as primary right ventricular systolic dysfunction related to cardiopulmonary bypass. It is most common after more prolonged procedures. Figures 23.82 and 23.83 were recorded in patients with hemodynamic compromise related to right ventricular dysfunction following otherwise uncomplicated cardiovascular surgery.
**FIGURE 23.82.** Parasternal short-axis view of the left ventricle recorded in a patient with refractory hypotension in the cardiac intensive care unit following mitral valve replacement. Note the normal size of the left ventricle but the marked dilation (arrows) and severe systolic dysfunction of the right ventricle which is the source of hemodynamic compromise.

**Video 23-82**

Figure 23.84 was recorded in a patient 24 hours after otherwise uncomplicated bypass surgery who subsequently developed hypotension and new electrocardiographic changes. The transthoracic echocardiogram
revealed evidence of myocardial ischemia in the left anterior descending distribution, prompting reevaluation for the integrity of the previously placed bypass graft. Occasionally after mitral valve repair, a delayed syndrome of dynamic outflow tract obstruction may result in the development of a new systolic murmur and hypotension. Measurement of hemodynamics with right heart catheterization may be misleading in this situation. The etiology, clinical decision making, and management are the same as for the scenario previously discussed for intraoperative evaluation.

Pericardial effusion is probably the most common complication following cardiovascular surgery. It should be recognized as part of the surgical procedure that the pericardium has been opened. As such classically distributed pericardial effusion may not be encountered and loculated effusions are common. Additionally, as the effusion is likely hemorrhagic in etiology, varying components of pericardial fluid and hematoma will be encountered. Obviously transthoracic imaging may be problematic in a patient shortly following cardiovascular surgery and secondary circumstantial findings need to be incorporated to determine the full clinical implications of the pericardial fluid and/or hematoma. These include evidence of atrial or ventricular compression by hematoma or fluid, as well as the appearance of an underfilled chamber which may suggest compression of either the vena cava or pulmonary veins by hematoma or fluid. Figures 23.85 through 23.89 were recorded in patients shortly following cardiovascular surgery with hemodynamic deterioration. Note the substantial range and variation of appearance of fluid as well as evidence of chamber compression or venous inflow obstruction. From an echocardiographic standpoint it may not be possible to accurately separate hematoma from other solid tissues in the mediastinum. For this reason, and because effusion may be atypically located and occasionally outside the field of view of echocardiography, computed tomography plays an incremental and valuable role in evaluating many of these patients prior to surgical reexploration.
FIGURE 23.83. Apical four-chamber view recorded in a patient 1 day following otherwise uncomplicated coronary bypass surgery. The patient was known to have a structurally normal heart with normal left and right ventricular size and function preoperatively. In the central illustration note the dilation of the right ventricle with systolic dysfunction apparent in the real-time image. The panel at the upper left was recorded with color Doppler and demonstrates moderate functional tricuspid regurgitation. These findings spontaneously resolved without specific therapy over the following 5 days. [Video 23-83 CFD]
FIGURE 23.84. Transthoracic echocardiogram recorded 24 hours after an otherwise uncomplicated coronary bypass surgery in a patient who subsequently developed new electrocardiographic changes. Note the dyskinesis of the apical septum (arrows) in a distribution typical for left anterior descending coronary artery ischemia or infarction suggesting early graft failure.
coming soon

Video 23-84
FIGURE 23.85. Parasternal transthoracic echocardiogram recorded in a patient 3 days following repeat aortic valve replacement who developed progressive hypotension and malperfusion. In the parasternal long-axis view, note the large, compressive pericardial effusion. In the short-axis view, note the 3/4 circumferential extent of the effusion and the compression of the midright ventricle to a small slit (black arrow).
FIGURE 23.86. Apical view recorded in the same patient depicted in Figure 23.85. Notice the pericardial effusion predominantly localized over the right side of the heart and the marked inversion of the right atrial wall in early systole (arrow).
FIGURE 23.87. Parasternal short-axis view recorded in a patient in the cardiac intensive care unit with progressive hypotension. In the short-axis view, note the normal geometry of the left ventricle but the compression of the right ventricle by a complex anterior mediastinal mass which represents a combination of free pericardial fluid and organized hematoma. The two images at the left are CT scans recorded in the same patient. In the upper image recorded at the ventricular level note the modest anterior mediastinal hematoma. The image at the lower left was recorded several centimeters superior showing a substantially
larger hematoma (arrows).  

FIGURE 23.88. Transthoracic echocardiograms recorded in a patient following a cardiovascular procedure who developed progressive hypotension on postoperative day 3. A: Apical four-chamber view revealing marked compression of the right atrium and lesser compression of the left atrium. There is a spherical echo-free space compressing the right atrium which measures approximately 7 cm in diameter (double headed arrows). This represents a loculated pericardial effusion. B was recorded in a right ventricular inflow tract view with color Doppler and demonstrates marked compromise of right ventricular inflow. The panel at the upper left was recorded with continuous-wave Doppler demonstrating high-velocity gradients from the right atrium to the right ventricle.
On occasion, presentation with pericardial fluid or hematoma may be delayed and occur several weeks or months after otherwise uneventful cardiovascular surgery. Figures 23.90 and 23.91 were recorded in patients several weeks remote from cardiac surgery who presented with dyspnea and echocardiographic evidence of pericardial fluid and/or mass effect. Figure 23.91 was recorded in a patient who presented to the emergency department after a motor vehicle accident with shortness of breath in whom the echocardiogram suggested anterior mediastinal hematoma. Chest CT however revealed that this was herniation of abdominal fat into the anterior mediastinum.

A final complication of intrathoracic procedures that can be identified with
Transesophageal echocardiography is pulmonary vein stenosis after lung transplantation. This results in obstruction of flow from the transplanted lung and may result in unilateral pulmonary edema of the recently transplanted lung. After lung transplantation, complete transesophageal echocardiographic evaluation should include visualization of all four pulmonary veins for size, flow turbulence, and determination of gradients. This is often a difficult examination to perform and, on occasion, not all pulmonary veins will be visualized. As the actual site of stenosis, which is most often at the anastomotic site, may not be within the field of view, careful evaluation of pulmonary vein flow is essential. If the actual level of stenosis can be identified one may detect an elevated pulmonary vein gradient. Conversely if the level of stenosis is not within the field of view, one may simply note reduced pulmonary vein flow. Figure 23.92 was recorded in a patient following double lung transplant with poor oxygenation and unilateral pulmonary edema. Notice that the level of stenosis cannot be identified, however there is a marked difference in flow between the two pulmonary veins as documented by Doppler.

**FIGURE 23.89.** Transesophageal echocardiogram recorded in a patient in a
cardiac surgery intensive care unit who developed hypotension 3 days following an otherwise uncomplicated mitral valve replacement. The central view is a longitudinal view of the left ventricle and left ventricular outflow tract. The leftward-pointing arrows denote the mitral valve prosthesis. Note the complex mass compressing the left atrium (arrows) which represents a posterior mediastinal hematoma. The inset at the lower left was recorded in a lower esophageal view and is a short-axis view of the left ventricle also demonstrating organizing hematoma (arrows) posterior to the left ventricle. [Video 23-89A]

Video 23-89A

Video 23-89b
FIGURE 23.90. Apical four-chamber view recorded 4 weeks following uncomplicated coronary artery bypass surgery. The patient presented with dyspnea. In this apical four-chamber view note the large oval echo-free space at the apex of the heart compressing both the right and left ventricles (downward-pointing arrows). The inset at the upper left is a chest CT recorded in the same patient also demonstrating a loculated apical effusion. Based on the signal intensity this represents organized hemorrhagic fluid.  

Video 23-90
FIGURE 23.91. Transthoracic echocardiography recorded in a patient 6 weeks following uncomplicated cardiovascular surgery who was involved in a motor vehicle accident and developed significant dyspnea. A is a parasternal long-axis view. Note the complex-appearing soft tissue density mass anterior to the right ventricle which compresses the right ventricular free wall (downward-pointing arrows). This mass was suspected to be a traumatic pericardial effusion. The image at the lower left is a chest CT demonstrating that the mass is actually herniated abdominal fat with a component of large bowel (small arrow) and not pericardial effusion. Panel B was recorded in an apical four-chamber view in the same patient revealing an extensive soft tissue density mass surrounding the apex and right ventricular free wall (arrows).

Video 23-91a
Role in Electrophysiology Procedures

Echocardiography plays a supporting role in many electrophysiology procedures. This includes preprocedural screening for the presence of left atrial thrombus which represents a contraindication to procedures such as catheter ablation of atrial fibrillation. Typically a thorough transesophageal echocardiographic examination is performed prior to this procedure to evaluate atrial septal anatomy and exclude the presence of left atrial or left atrial appendage thrombus. There are several variations of atrial septal anatomy which may make transseptal puncture for access to the left atrium more challenging (Figure 23.93). An atrial septum that is in neutral position with a well-defined foramen ovale represents less of a technical challenge for transatrial puncture than does an atrial septum with markedly distorted geometry related to pressure overload or marked areas of fibrosis, or lipomatous infiltration. Either transesophageal or intracardiac echocardiography is commonly used at the time of the procedure to assist guiding transatrial puncture and catheter placement (Fig. 23.94). In most high-volume centers online monitoring is done by the performing electrophysiologist rather than by a dedicated echocardiographer.

Following catheter ablation or other electrophysiology procedures echocardiography can be used to screen for complications such as perforation with pericardial effusion or, on rare occasions trauma to a valvular structure. A delayed complication unique to catheter ablation of atrial fibrillation is iatrogenic pulmonary vein stenosis (Fig. 23.95). Pulmonary vein stenosis can
be detected with transesophageal echocardiography when narrowing of a pulmonary vein in association with pathologically high-velocity pulmonary vein flow is detected. Computed tomography of the pulmonary veins is also highly accurate for detecting this complication.

The approach to left atrial catheter ablation procedures involves a transatrial septal approach. This may either be by way of a PFO or direct puncture of the atrial septum. Following the latter one may occasionally see evidence of a residual iatrogenic atrial level shunt (Fig. 23.96). The majority of these iatrogenic ASDs are small and hemodynamically insignificant and many resolve over time.

**FIGURE 23.92.** Transesophageal echocardiogram recorded in a patient 48 hours following double lung transplant who developed worsening hypoxia and unilateral pulmonary edema. Because of the postoperative state the study is technically challenging and views are suboptimal. A is a recording of the left upper pulmonary vein (LUPV). The left atrial appendage (LAA) is noted as well. In this midsystolic frame note the absence of any significant flow from the pulmonary vein into the atrium. The inset at the upper left was recorded with pulsed Doppler in the pulmonary vein. Note the essential absence of outflow. B is a recording of a right pulmonary vein demonstrating robust flow. The inset at the lower right was recorded with Doppler in the pulmonary vein. Note the high-volume continuous flow from the right pulmonary vein.
Video 23-92a

coming soon

Video 23-92b

coming soon
FIGURE 23.93. Transesophageal echocardiograms recorded in patients being considered for interventional procedures that require crossing the atrial septum. Atrial septal puncture is technically easiest in individuals in whom the atrial septum is thin and located in a “neutral” position (A). B: An atrial septum in “neutral” position but with significant lipomatous atrial hypertrophy (double headed arrow). Obviously, puncture will be difficult if not impossible at the areas of lipomatous infiltration compared to the thin foramen (single arrow). C: A diffusely and uniformly thickened atrial septum that may present technical difficulties for successful puncture in crossing the atrial septum. D: An atrial septum that is distorted because of marked left-to-right bowing as a consequence of mitral stenosis, which may also complicate transseptal puncture. In the schematic, the dotted line represents the path of a transseptal catheter passed from the inferior vena cava across the foramen ovale.
An infrequent but potentially fatal complication of electrophysiologic ablative procedures is formation of an atrial esophageal fistula. These may present either acutely or several weeks following the procedure typically with chest pain and dysphagia. Figure 23.97 was recorded in a patient 3 weeks following catheter ablation for atrial fibrillation who initially presented with a cerebrovascular accident. On echocardiography, there is an oscillating curtain originating in the anterior septum and apex which represents free air within the cavity of the left ventricle which is a consequence of the esophageal atrial fistula.
FIGURE 23.94. Intracardiac echocardiogram recorded in a patient undergoing an electrophysiologic procedure. **A:** Note the sheath (*horizontal arrow*) in the right atrium. The needle can be seen “tenting” the atrial septum (*vertical arrow*) into the left atrium. **B:** The needle has punctured the atrial septum and a small bolus of saline contrast has been injected (*arrows*) confirming the location of the needle within the body of the left atrium.
FIGURE 23.95. Transesophageal echocardiogram recorded in a patient following pulmonary vein isolation for atrial fibrillation. In the color flow image (inset), note the constricted flow in the pulmonary vein consistent with pulmonary stenosis and the peak velocity of 1.6 m/s in the continuous-wave Doppler profile.

Video 23-95

An additional procedure related to atrial fibrillation is placement of a left
atrial occluder device. This device is placed using venous access and a transatrial approach to place an expandable occluder in the left atrial appendage. When properly deployed this device excludes the left atrial appendage from the left atrium and has been demonstrated to reduce thromboembolic events in patients with atrial fibrillation who are not candidates for anticoagulation. Appropriate placement of the device requires precise preprocedure planning, including measurement of the depth and width of the left atrial appendage in multiple imaging planes. This allows selection of an appropriately sized device to fully exclude the left atrial appendage.

The precise measurements, exclusion and inclusion criteria for placement of these and other devices is in evolution and the reader is advised to consult the specific recommendations of the manufacturer regarding detailed measurements which are required for procedural planning. Figure 23.98 was recorded in a patient being considered for a Watchman device and depicts typical measurements required for procedural planning. These include the dimensions of the mouth of the left atrial appendage as well as the depth of the left atrial appendage. Additional features such as presence of thrombus, presence of multiple lobes etc. also need to be considered. Three-dimensional echocardiography can play a valuable role in obtaining precise measurement.
defect with color Doppler demonstrating flow from the left atrium to the right atrium (arrows). The inset at the lower right was the preprocedure transesophageal echocardiogram demonstrating an intact atrial septum. In this case the small atrial septal defect is the result of atrial puncture for catheter ablation. 

Video 23-96a

Video 23-96 96b
FIGURE 23.97. A: Parasternal long-axis view recorded in a patient 3 weeks following catheter ablation for atrial fibrillation presenting to an emergency department with an acute CVA. In this parasternal view note the curtain of echoes (left pointing arrows) originating at the anterior septum (single arrow) obscuring the left ventricular cavity. In the real-time image note the oscillating nature of this curtain of echoes which represents free air along the left septum. B is an apical four-chamber view and also reveals oscillating echoes originating at the apex obscuring the medial half of the left ventricle. These echoes originate from air trapped at the apex of the left ventricle. The inset at the upper left is a chest CT demonstrating a pocket of air at the apical septum (arrows). Video 23-97a
Video 23-97b

Actual placement of the device is performed with a combination of transesophageal echocardiographic and fluoroscopic guidance. Figure 23.99 was recorded at the time of implantation of an occluder device and demonstrates the appropriate positioning of the device within the left atrial appendage. At the time of implantation close approximation circumferentially of the device and the appendage wall is confirmed by both anatomical imaging and color Doppler flow imaging.
FIGURE 23.98. Transesophageal echocardiographic images recorded as a part of planning for placement of a left atrial occluder device in a patient with atrial fibrillation. At the upper left is a real-time three-dimensional image visualizing the orifice of the left atrial appendage. The image at the upper right demonstrates measurement of the circumference of the appendage orifice. The image at the lower left demonstrates measurement of the depth of the atrial appendage and the image at the lower right demonstrates orthogonal measurements of the orifice.

Following implantation of an atrial appendage occluder device, anticoagulation is typically continued for 3 months after which repeat echocardiography is performed to ensure appropriate seating of the device, exclusion of the body of the left atrial appendage, and absence of residual thrombus. Figure 23.100 was recorded in a patient during routine surveillance echocardiography after implantation of an atrial occluder device and demonstrates successful placement in a patient with complex atrial anatomy.
FIGURE 23.99. Transesophageal echocardiogram recorded time of placement of a left atrial appendage occluder device. In the upper panel note the large bore catheter which has traversed the atrial septum into the left atrium for delivery of the device (white arrow). The device itself (black arrows) is noted to be well seated in the left atrial appendage. The lower panel was recorded after full delivery of the device and demonstrates complete occlusion of the left atrial appendage, the boundaries of which are noted by the black arrows.
FIGURE 23.100. Transesophageal echocardiogram recorded 3 months after implantation of an atrial occluder device in a patient with persistent atrial fibrillation. In this instance there was complex left atrial appendage anatomy with
a side lobe. A: Recorded with standard two-dimensional imaging. Note the device which appears to fully occlude the left atrial appendage (left hand set of arrows) and the thrombosed side lobe of the left atrial appendage (rightward and downward pointing arrows). The inset at the lower left is a pre-placement transesophageal echocardiogram in the same view demonstrating the body of the left atrial appendage and the prominent side lobe (SL). B: Image in the same orientation with color flow Doppler. The atrial occluder device (W) is as noted. Note the minimal residual color flow Doppler around the margins of the device. There is no evidence of residual thrombus burden.
FIGURE 23.101. Transesophageal echocardiogram recorded in a patient 30 days following a left atrial occluder device implantation. In the central figure the Watchman device (W) is well visualized. In this image to the left of the device note the clear space (arrows) in which pectinate muscles are visualized demonstrating that the device is not fully occlusive. A similar finding is noted in the real-time three-dimensional image at the upper right. The image at the upper left was recorded with color Doppler and demonstrates continued communication into the nonoccluded area of the atrial appendage (arrow).
FIGURE 23.102. Transesophageal echocardiogram recorded in a patient following placement of an atrial clip with a minimally invasive surgical procedure. In the central image note the rigid nonbiologic line separating the left atrium from the left atrial appendage (downward-pointing arrows) and the residual left atrial appendage cavity (upward-pointing arrows). The inset at the upper right is a preprocedure image. 

Video 23-102

Residual issues that should be addressed following exclusion of the atrial appendage include incomplete exclusion with an anatomical space between the occluder device and the appendage wall (Fig. 23.101) which may pose an
ongoing risk for thrombus formation and subsequent embolic events.

A closely related procedure is thorascopically guided clipping of the atrial appendage, often performed in conjunction with a minimally invasive maze procedure. Similar considerations regarding follow-up for confirming full exclusion of left atrial appendage and absence of the thrombus are relevant. Figure 23.102 was recorded in a patient following a minimally invasive atrial clipping procedure and demonstrates the typical anatomy of an excluded left atrial appendage.

Suggested Readings


Intensive Care Unit/Shock


Intraoperative Application


** Procedure Monitoring**


** Structural Intervention**


To view video clips associated with the chapter, please access the eBook bundled with this text whenever this icon is present.
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