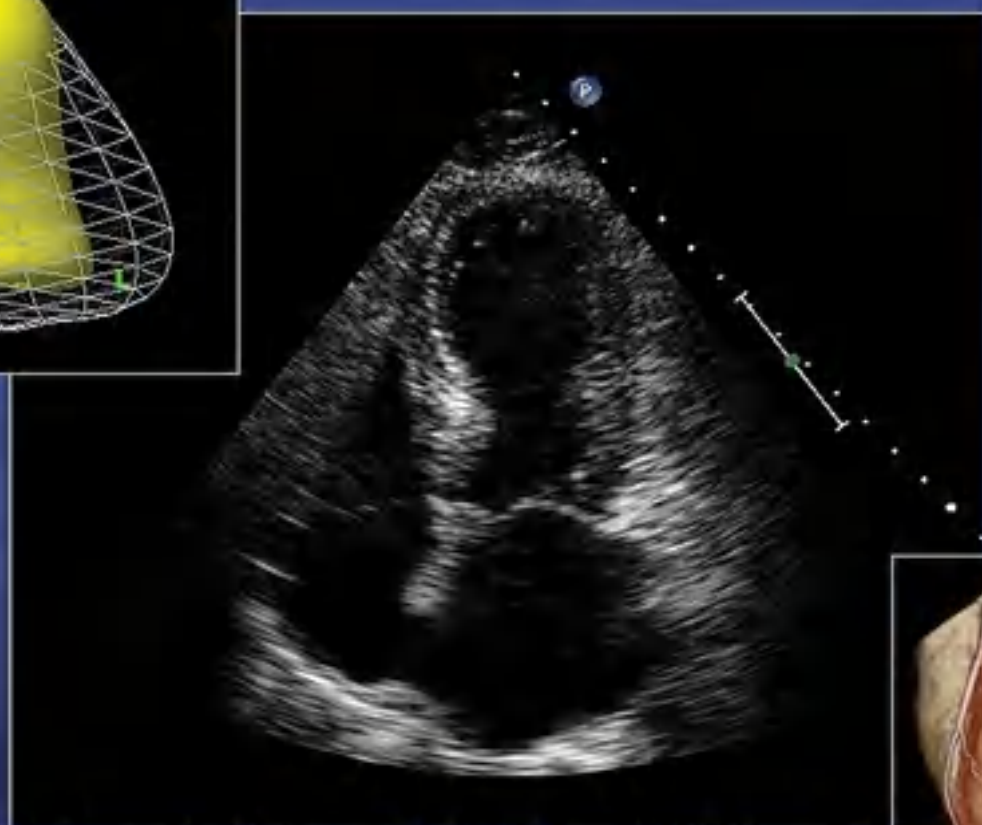


 **Feigenbaum's**  
**Echocardiography**

EIGHTH EDITION



 **Wolters Kluwer**

**William F. Armstrong**  
**Thomas Ryan**

# **Feigenbaum's** **Echocardiography**

## **EIGHTH EDITION**

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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

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“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  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.<sup>1–6</sup> 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.<sup>7</sup> A Franciscan friar, Marin Mersenne (1588–1648), is frequently called the “father of acoustics” because he first measured the velocity of sound.<sup>7</sup> Another early physicist, Robert Boyle (1627–1691), recognized that a medium was necessary for the propagation of sound.<sup>7</sup> Abbe Lazzaro Spallanzani (1727–1799) is frequently referred to as the “father of ultrasound.”<sup>8</sup> 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.<sup>9</sup> 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.<sup>10,11</sup> 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.<sup>3</sup> Sokolov<sup>12</sup> described a method for using reflected sound to detect metal flaws in 1929. In 1942, Floyd Firestone,<sup>13</sup> 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,<sup>14</sup> 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,<sup>15</sup> 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.<sup>16</sup>

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<sup>17</sup> 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<sup>18</sup> 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.<sup>19,20</sup> 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<sup>20</sup> was the detection of left atrial masses. Schmidt and Braun<sup>21</sup> 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.<sup>22</sup> developed a scientific film that was shown at the Third European Congress of Cardiology in Rome in 1960. Edler et al.<sup>23</sup> also wrote a large review of cardiac ultrasound as a supplement to *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.

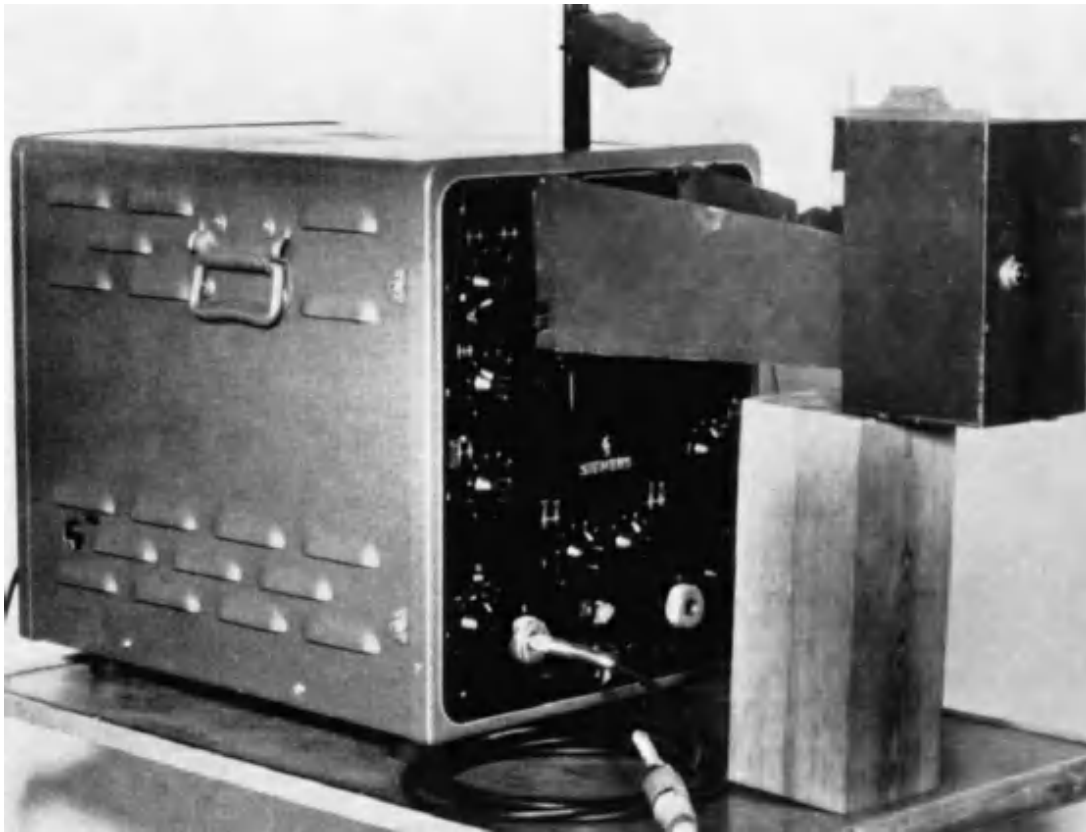
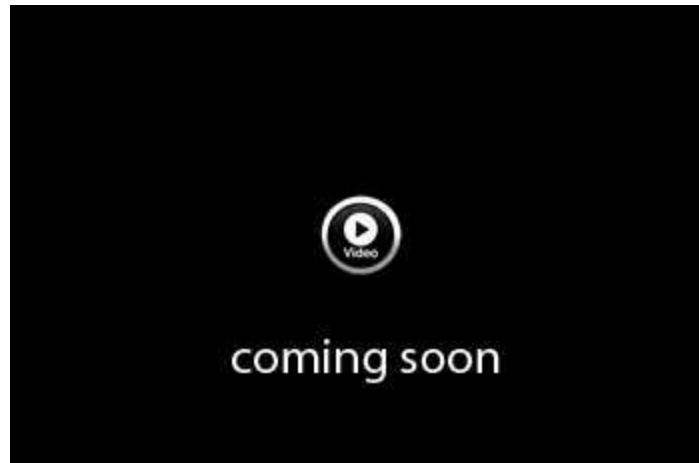


**FIGURE 1.1.** Ultrasonoscope initially used by Edler and Hertz for recording their early echocardiograms. (From Edler I. Ultrasound cardiography. *Acta Med Scand Suppl* 370 1961;170:39.)





### **Video 1-1**

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.<sup>24,25</sup> The investigators duplicated the findings of Edler and Effert with regard to mitral stenosis.<sup>26</sup> Unique contributions of the Chinese investigators included fetal echocardiography<sup>27</sup> and contrast echocardiography, using hydrogen peroxide and then carbon dioxide.<sup>28</sup>

In the United States, echocardiography was introduced by John J. Wild, H. D. Crawford, and John Reid,<sup>29</sup> 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.<sup>30</sup>

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.<sup>31</sup>

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.<sup>32,33</sup> 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.<sup>32,33</sup> American workers, such as Robert Rushmer in Seattle, were early investigators using Doppler techniques.<sup>34</sup> 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.<sup>35</sup> Eugene Strandness was a vascular surgeon in Seattle using Doppler for peripheral arterial disease.<sup>36</sup> European investigators were also very active in using Doppler technology. Several early French workers, namely, Peronneau<sup>37</sup> and later Kalmanson,<sup>38</sup> wrote extensively on the use of Doppler ultrasound to examine the cardiovascular system. A major development in Doppler ultrasound came when Holen<sup>39</sup> and then Hatle<sup>40</sup> 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.<sup>41</sup> 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.<sup>42</sup> 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.<sup>43</sup> 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.<sup>44</sup>

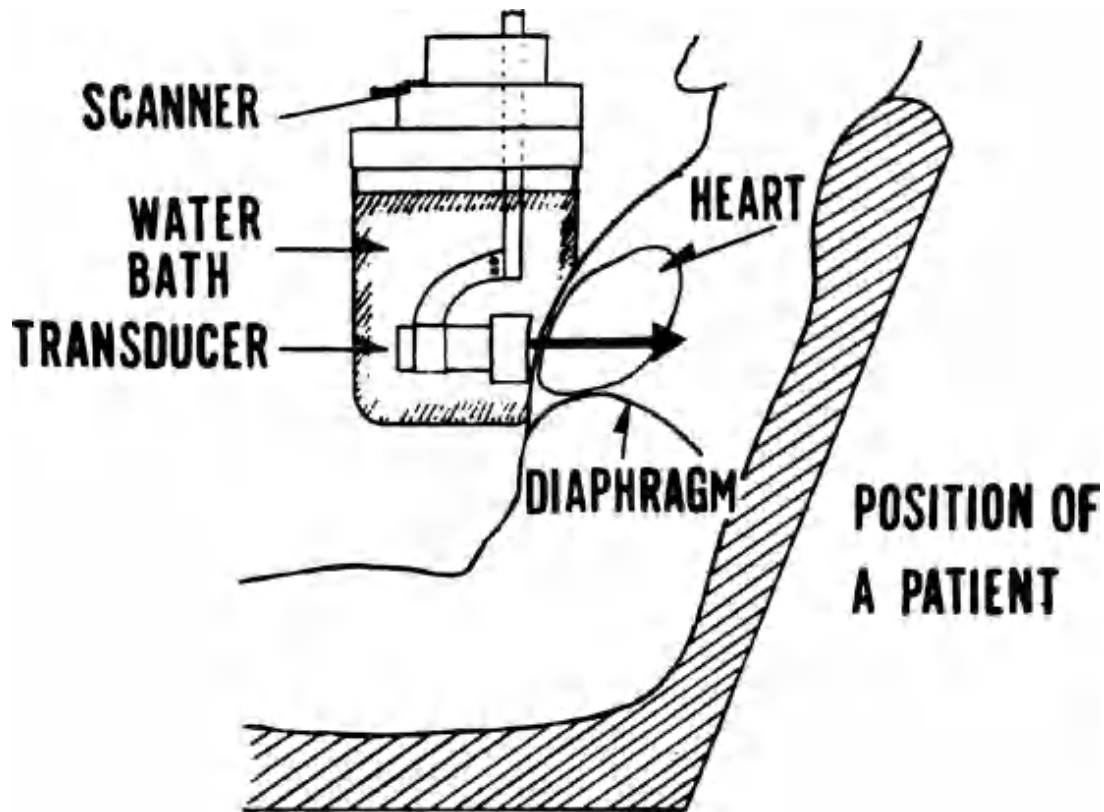


**FIGURE 1.2.** Relatively early system using a mechanical sector scanner and a water bath to obtain cross-sectional echograms of the heart. (Reprinted with permission from Ebina T, Oka S, Tanaka N, et al. The ultrasono-tomography of the heart and the great vessels in living human subjects by means of the ultrasonic reflection technique. *Jpn Heart J* 1967;8(4):331–353. Copyright © 1967 International Heart Journal Association.)

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.<sup>45</sup> The Japanese introduced a variety of ultrasonic devices to create two-dimensional recordings of the heart.<sup>46</sup> They used elaborate water baths and scanning techniques (Fig. 1.2). Gramiak et al.<sup>47</sup> at the University of Rochester used reconstructive two-dimensional M-mode techniques to create ultrasonic “cinematography” (Fig. 1.3). Donald King<sup>48</sup> in New York developed a stop-action type of technique for creating a reconstructed two-dimensional image of the heart (Fig. 1.4).

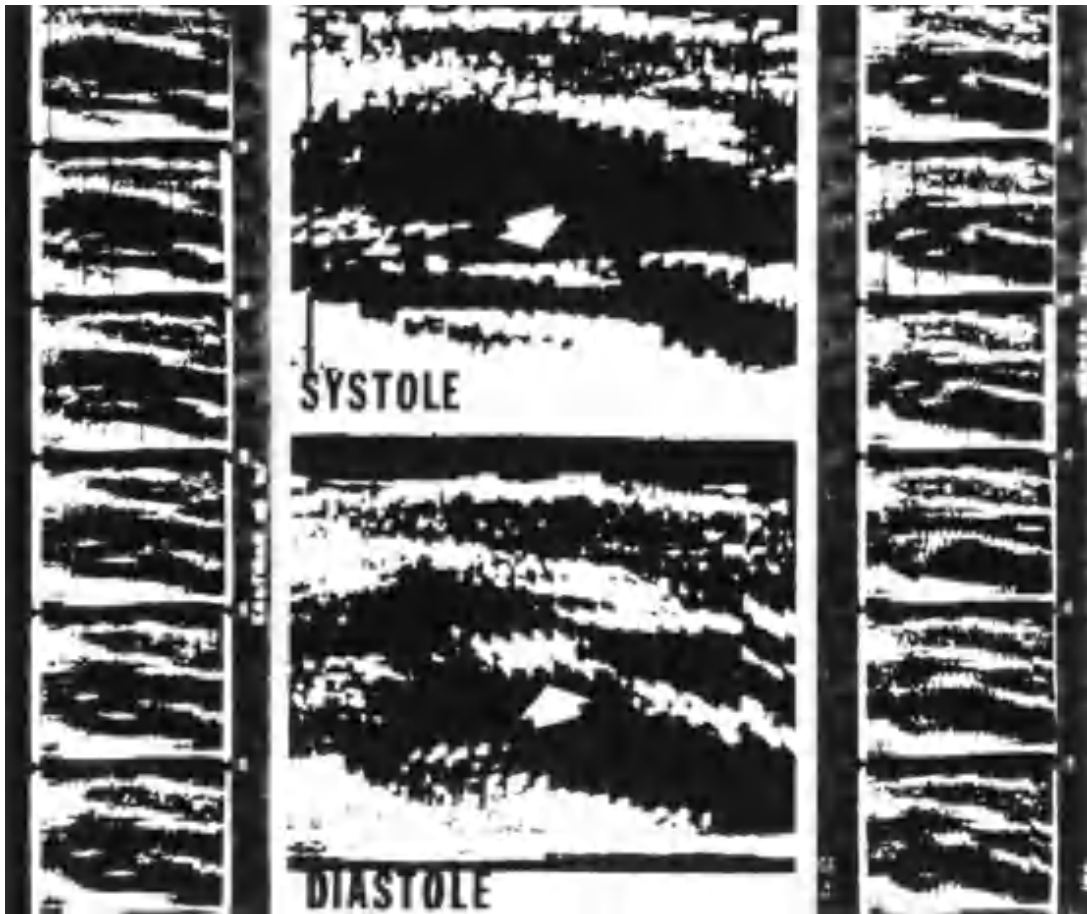


**FIGURE 1.3.** Frames from a movie film using a spatially oriented reconstruction of the M-mode echogram to produce a pseudo real-time, cross-sectional examination of the mitral valve motion. The two enlarged frames show the position of the mitral valve (*arrow*) in systole and diastole. (Reprinted 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.)



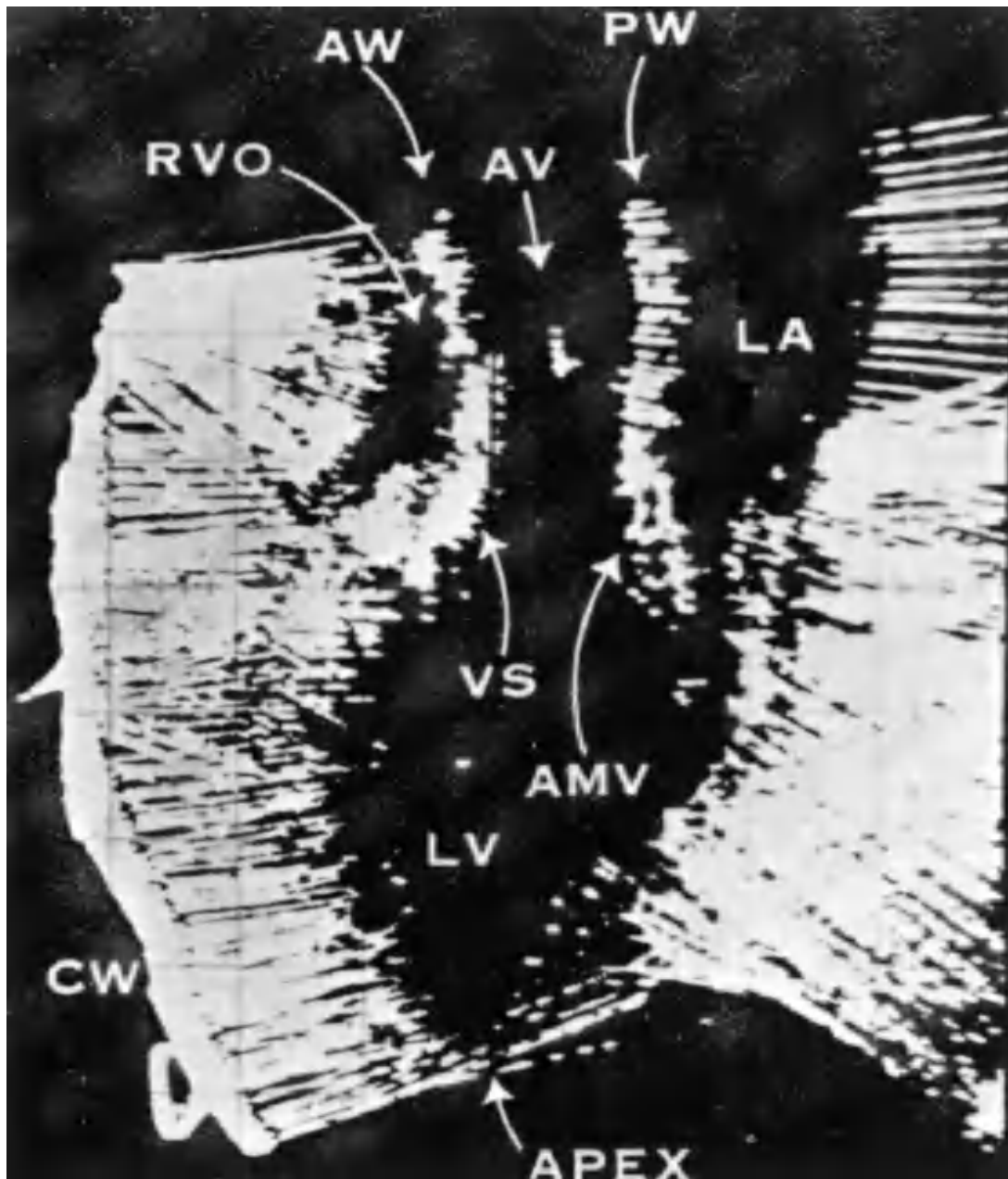


**FIGURE 1.4.** Compound, electrocardiograph-gated cross-sectional examination of the heart. AMV, anterior mitral valve leaflet; AW, anterior wall of the aorta; CW, chest wall; PW, posterior wall of the aorta; RVO, right ventricular outflow; VS, interventricular septum. (Reprinted with permission from King DL, Steeg CN, Ellis K. Visualization of ventricular septal defect by cardiac ultrasonography. *Circulation* 1973;48(6):1215–1220. Copyright © 1973 American Heart Association, Inc.)

A major breakthrough occurred when an engineer, Nicholas Bom, in Rotterdam, developed a linear scanner ([Fig. 1.5](#)).<sup>49</sup> 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<sup>50</sup> 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.<sup>51</sup> Eventually, mechanical sector scanners were replaced by phased-array technology, which was initially developed by Fritz Thurstone and Olaf vonRamm<sup>52</sup> at Duke University.



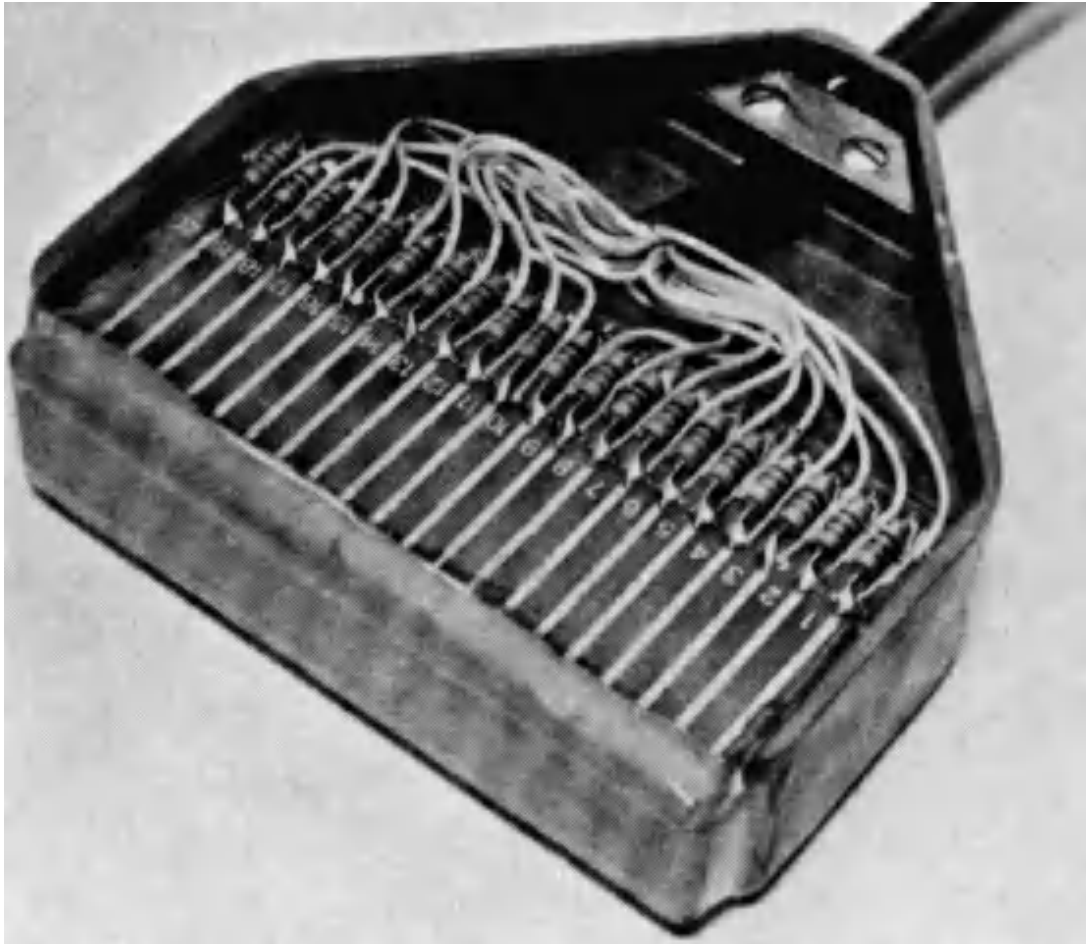


**FIGURE 1.5.** Photograph of a multi-element transducer that provides an electronic linear scan of the heart. This probe consists of 20 individual piezoelectric elements. (Reprinted with permission from Bom N, Lancee CT, Van Zwieten G, et al. Ultrascan echocardiography. I. Technical description. *Circulation* 1973;48(5):1066–1074. Copyright © 1973 American Heart Association, Inc.)



**FIGURE 1.6.** Photograph of a handheld mechanical sector scanner. (From Eggleton RC, Feigenbaum H, Johnston KW, et al. Visualization of cardiac dynamics with real-time B-mode ultrasonic scanner. In: White D, ed. *Ultrasound in Medicine*. New York: Plenum Publishing, 1975:1385.)

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).<sup>53</sup> 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.<sup>54</sup> 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<sup>55</sup> helped popularize the clinical value of two-dimensional color Doppler imaging.

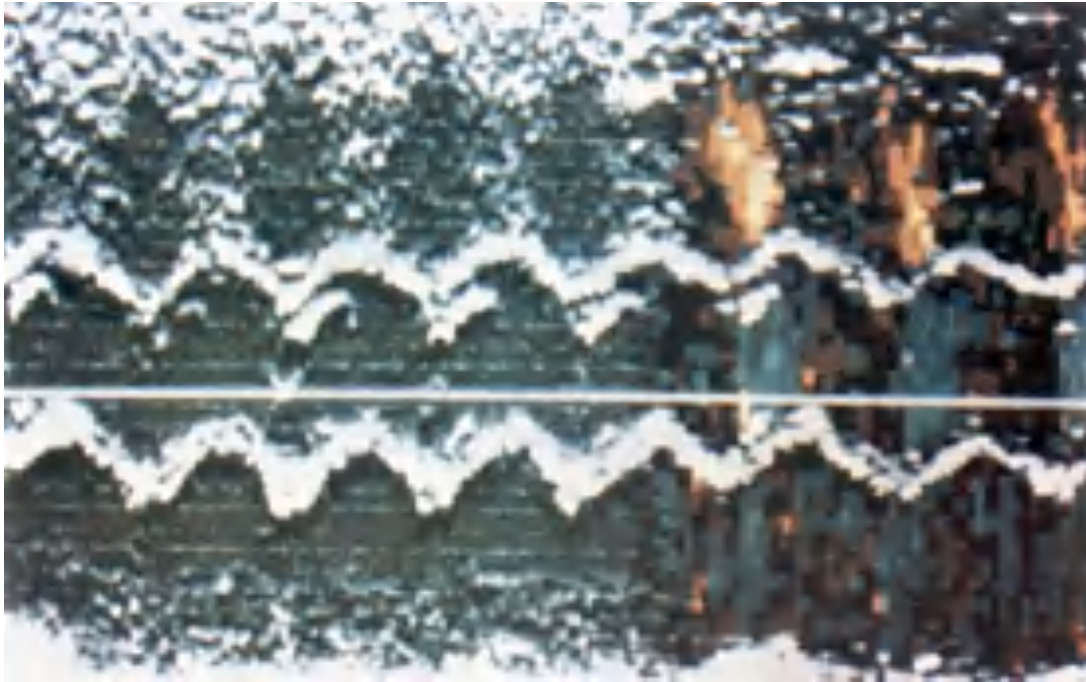


**FIGURE 1.7.** Combined M-mode and Doppler recording whereby the Doppler signal is superimposed on the M-mode tracing. The direction and velocity of the Doppler signal are displayed in varying colors. This particular recording shows the right ventricular outflow tract and aorta. (From Brandestini MA, 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.<sup>56</sup> This technique never became clinically popular. However, both Japanese and European investigators began working with this technology.<sup>57,58</sup> They all attempted to obtain two-dimensional images with a transesophageal probe. Initially, the devices were mechanical and later became electronic. Hisanaga et al.<sup>57</sup> were among the Japanese engineers, and Jacques Souquet was a European engineer who made a major contribution to transesophageal electronic probes in 1982.<sup>59</sup> 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.<sup>60</sup> 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.<sup>61</sup>

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.<sup>62,63</sup> 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.<sup>64</sup>



**FIGURE 1.8.** Early M-mode echocardiograph using a Polaroid camera to record an echocardiogram.

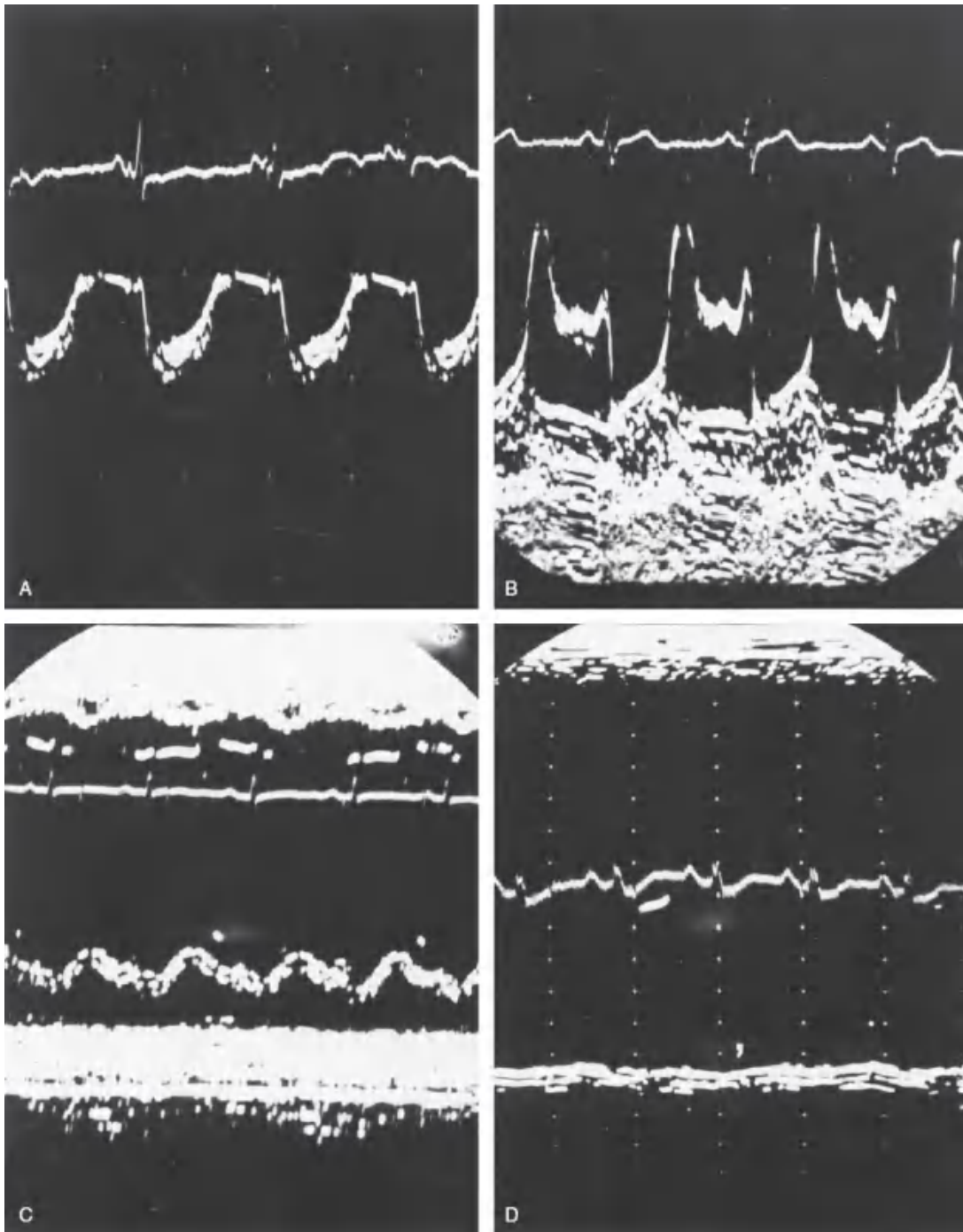


**FIGURE 1.9.** Early Polaroid recordings of M-mode echocardiogram. **A:** Mitral stenosis. **B:** Normal mitral valve. **C:** Pericardial effusion. **D:** Dilated nonmoving left ventricle.

Handheld echocardiographs date back to 1978.<sup>65</sup> This early device did not have sufficient image quality to be useful. However, now several such instruments are available and increasing in popularity.



## RECORDING ECHOCARDIOGRAMS

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.



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**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.

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.

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## Chapter 2

# Physics and Instrumentation

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:

$$v = f \times \lambda \quad [\text{Eq. 2.1}]$$

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/ $\mu$ s). Thus, to find the wavelength of a 3-MHz transducer, the solution would be given by:

$$v = f \times \lambda \quad [\text{Eq. 2.1}]$$

$$\lambda = v \div f$$

$$\lambda = 1,540 \text{ m/s} \div 3,000,000 \text{ cycles/s}$$

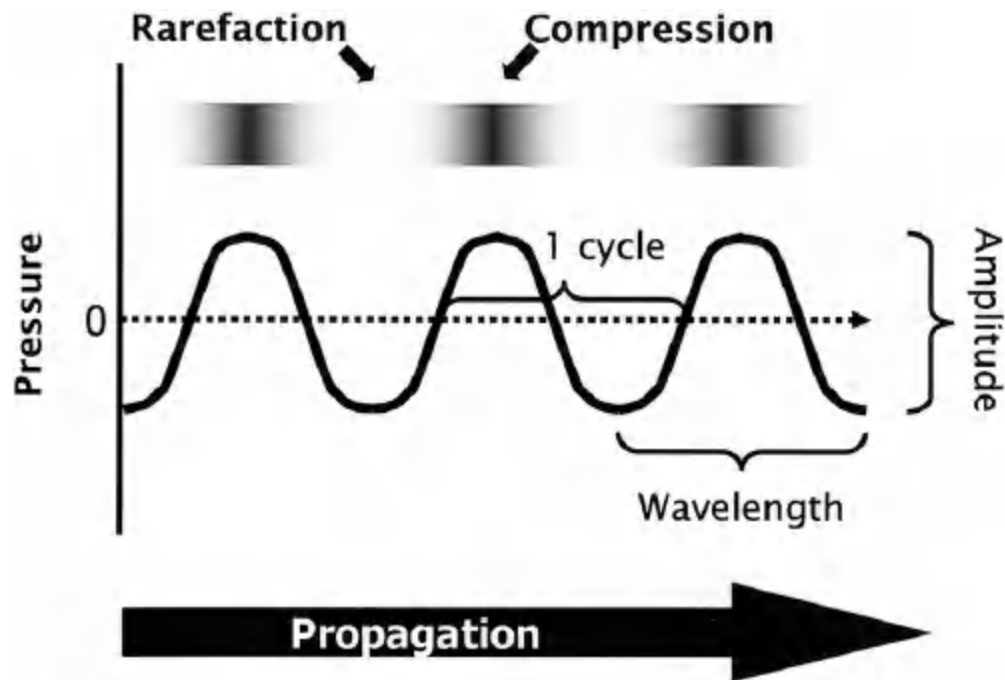


**FIGURE 2.1.** This schematic illustrates how sound can be depicted as a sine wave whose peaks and troughs correspond to areas of compression and rarefaction, respectively. As sound energy propagates through tissue, the wave has a fixed wavelength that is determined by the frequency and amplitude that is a measure of the magnitude of pressure changes. See text for further details.

**Table 2.1** DEFINITIONS OF BASIC TERMS

| Term               | Definition                                                                                                                                                                                         |
|--------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Absorption         | The transfer of ultrasound energy to the tissue during propagation                                                                                                                                 |
| Acoustic impedance | The product of the density of the medium and the velocity of sound; differences in acoustic impedance between two media determine the ratio of transmitted versus reflected sound at the interface |
| Amplitude          | The magnitude of the pressure changes along the wave; also, the strength of the wave (in decibels)                                                                                                 |
| Attenuation        | The net loss of ultrasound energy as a wave propagates through a medium                                                                                                                            |
| Cycle              | The combination or sum of one compression and one rarefaction of a propagating wave                                                                                                                |
| Dead time          | The time in between pulses that the echograph is not emitting ultrasound                                                                                                                           |
| Decibel            | A logarithmic measure of the intensity of sound, expressed as a ratio to a reference value (dB)                                                                                                    |
| Duty factor        | The fraction of time that the transducer is emitting ultrasound, a unitless number between 0 and 1                                                                                                 |

|                            |                                                                                                                                                                                                                                                                                     |
|----------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Far field                  | The diverging conical portion of the beam beyond the near field                                                                                                                                                                                                                     |
| Frequency                  | The number of cycles per second, measured in Hertz (Hz)                                                                                                                                                                                                                             |
| Gain                       | The degree, or percentage, of amplification of the returning ultrasound signal                                                                                                                                                                                                      |
| Half-layer value           | The distance an ultrasound beam penetrates into a medium before its intensity has attenuated to one-half the original value                                                                                                                                                         |
| Intensity                  | The concentration or distribution of power within an area, often the cross-sectional area of the ultrasound beam, analogous to loudness                                                                                                                                             |
| Longitudinal wave          | A cyclic disturbance in which the energy propagation is parallel to the direction of particle motion                                                                                                                                                                                |
| Near field                 | The proximal cylindrical-shaped portion of the ultrasound beam before divergence begins to occur                                                                                                                                                                                    |
| Period                     | The time required to complete one cycle, usually expressed in microseconds ( $\mu\text{sec}$ )                                                                                                                                                                                      |
| Piezoelectricity           | 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 |
| Power                      | The rate of transfer over time of the acoustic energy from the propagating wave to the medium, measured in Watts                                                                                                                                                                    |
| Pulse                      | A burst or packet of emitted ultrasound of finite duration, containing a fixed number of cycles traveling together                                                                                                                                                                  |
| Pulse length               | The physical length or distance that a pulse occupies in space, usually expressed in millimeters (mm)                                                                                                                                                                               |
| Pulse repetition frequency | 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                                                                                                                                                 |
| Resolution                 | The smallest distance between two points that allows the points to be distinguished as separate                                                                                                                                                                                     |
| Sensitivity                | The ability of the system to image small targets at a given depth                                                                                                                                                                                                                   |
| Ultrasound                 | A mechanical vibration in a physical medium, characterized by a frequency $>20,000$ Hz                                                                                                                                                                                              |
| Velocity                   | The speed at which sound moves through a given medium                                                                                                                                                                                                                               |
| Wavelength                 | The length of a single cycle of the ultrasound wave; a measure of distance, not time                                                                                                                                                                                                |

**Table 2.2**

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

| Medium | Velocity (m/s) |
|--------|----------------|
|        |                |

|             |       |
|-------------|-------|
| Air         | 330   |
| Fat         | 1,450 |
| Water       | 1,480 |
| Soft tissue | 1,540 |
| Kidney      | 1,560 |
| Blood       | 1,570 |
| Muscle      | 1,580 |
| Bone        | 4,080 |

A simpler version of this equation is given by  $\lambda$  (in millimeters) =  $1.54/f$ , where  $f$  is the transducer frequency (in megahertz). This converts 1,540 m/s to 1.54 mm/ $\mu$ s, expresses frequency in megahertz, and yields wavelength in millimeters. Thus,

$$\lambda = v \div f$$

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

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<sup>2</sup>). 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            |                          |                  |
|-----------------------------|--------------------------|------------------|
| Material                    | Half-power Distance (cm) |                  |
| Water                       | 380                      | Less attenuation |
| Blood                       | 15                       |                  |
| Soft tissue (except muscle) | 1–5                      |                  |
| Muscle                      | 0.6–1                    |                  |
| Bone                        | 0.2–0.7                  |                  |
| Air                         | 0.08                     |                  |
| Lung                        | 0.05                     |                  |
|                             |                          | More attenuation |

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.

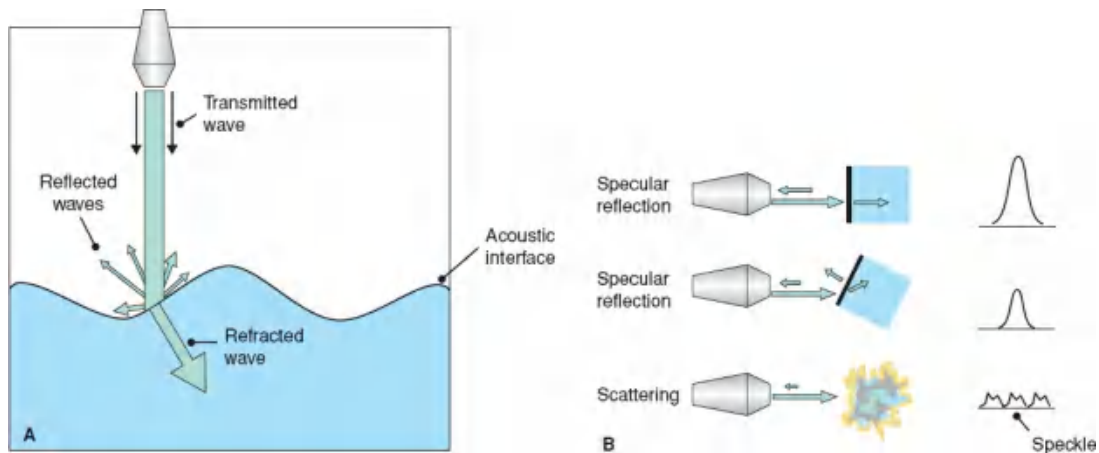


**FIGURE 2.2.** **A:** A transmitted wave interacts with an acoustic interface in a predictable way. Some of the ultrasound energy is reflected at the interface and some is transmitted through the interface. The transmitted portion of the energy is refracted, or bent, depending on the angle of incidence 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.

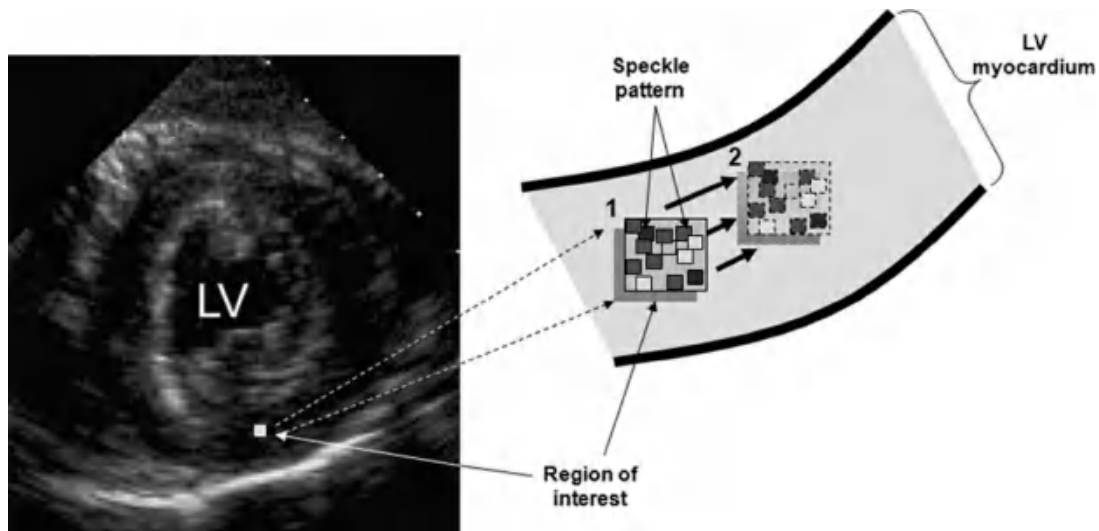


**FIGURE 2.3.** This schematic demonstrates how speckle tracking is performed. In



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.

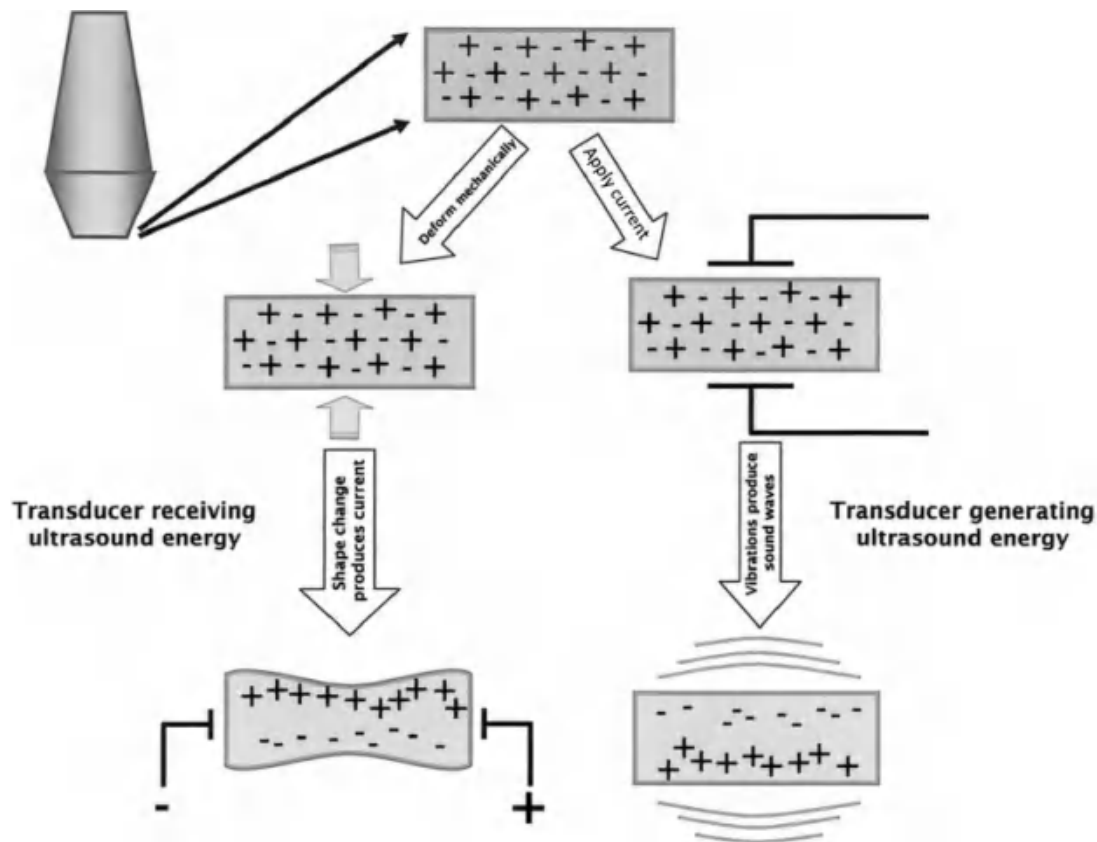


**FIGURE 2.4.** The principles of piezoelectricity are illustrated in this schematic. A piezoelectric crystal will vibrate when an electric current is applied, resulting in the generation and transmission of ultrasound energy. Conversely, when reflected energy encounters a piezoelectric crystal, the 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.

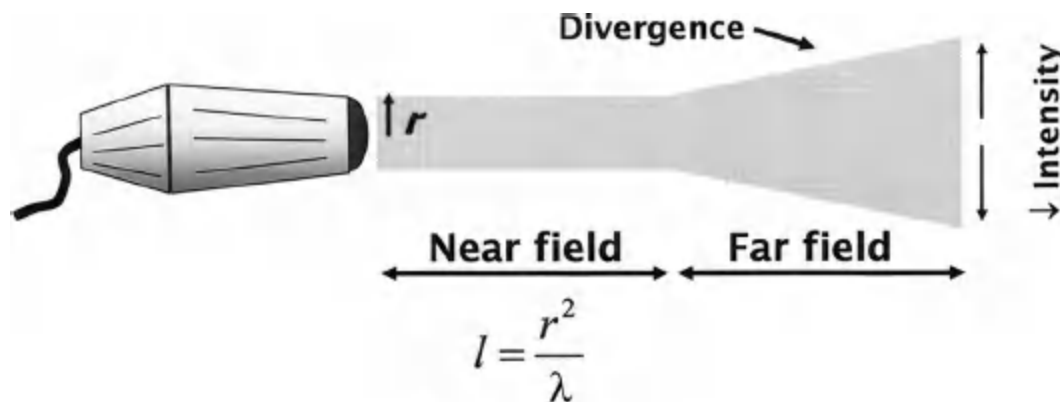


**FIGURE 2.5.** When ultrasound is emitted from a transducer, the shape of the beam behaves in a predictable manner. If the transducer face is round, the transmitted beam will remain cylindrical for a distance, defined as the near field. After propagating for a certain distance, the beam will begin to 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:

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

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.

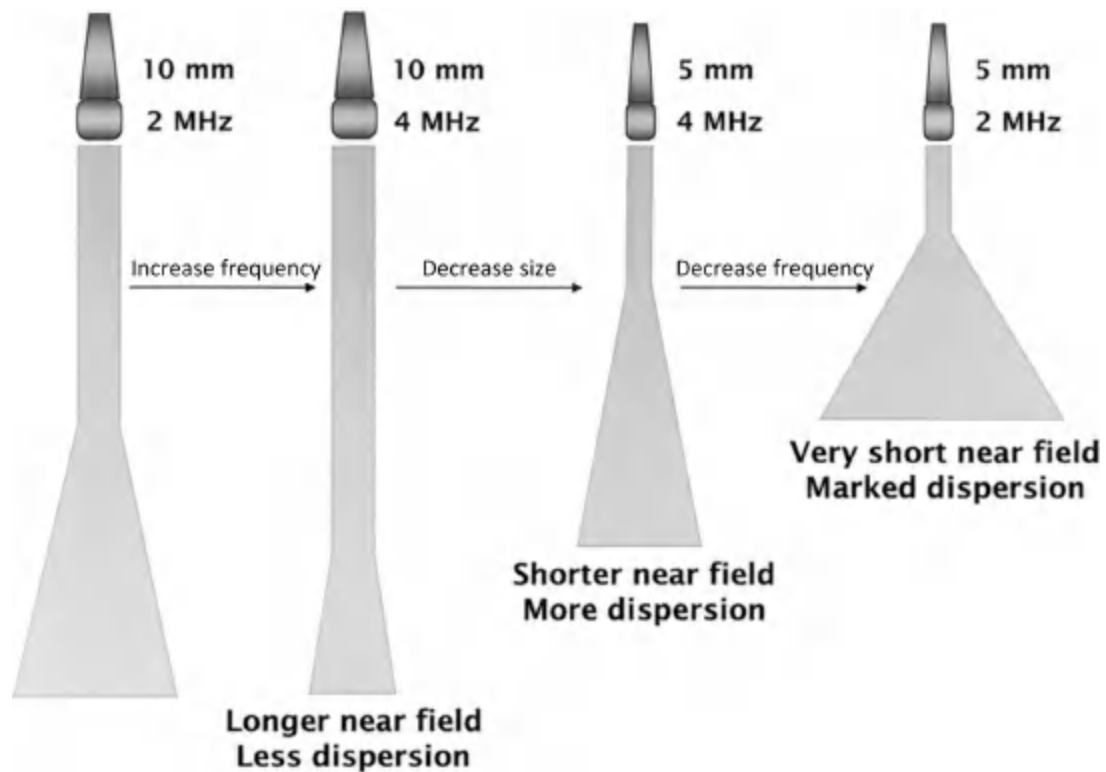


**FIGURE 2.6.** The length of the near field depends on transducer frequency and transducer size, as illustrated in these four examples. On the left, a transducer with a 10-mm diameter emits ultrasound at 2 MHz. This determines both the length of the near field and the rate of divergence in the far field. 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.

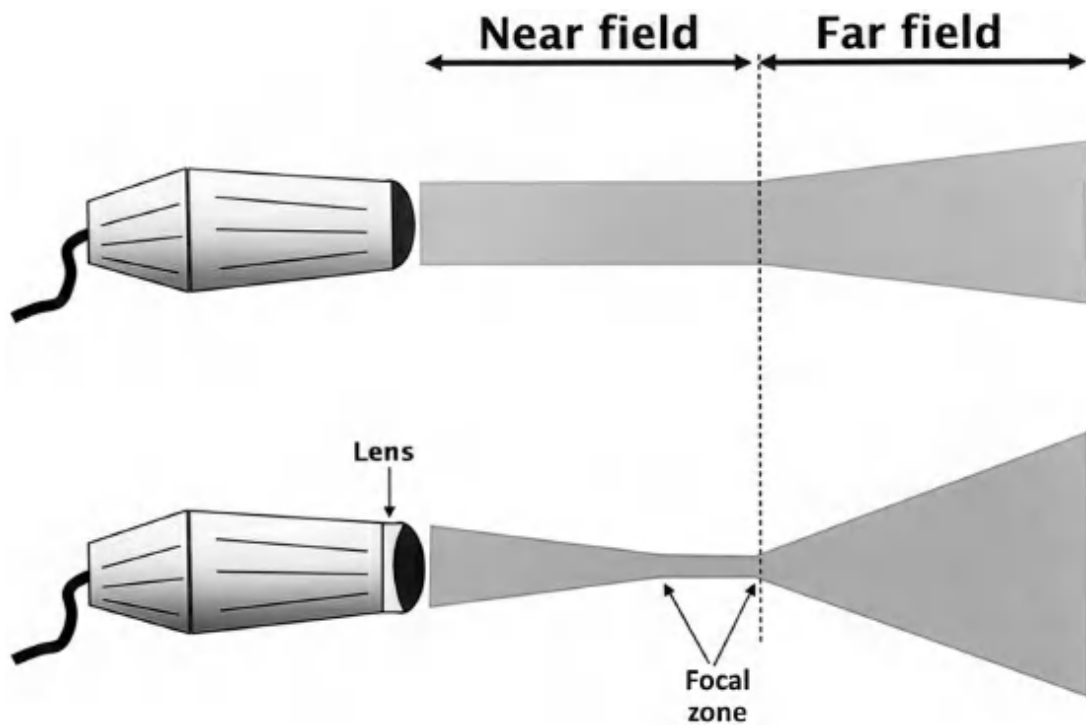


**FIGURE 2.7.** The ultrasound beam emitted by a transducer can be either unfocused (**top**) or can be focused by use of an acoustic lens (**bottom**). Focusing results in a narrower beam but does not change the length of the near field. An undesirable effect of focusing is that the rate of dispersion in the far field is greater.

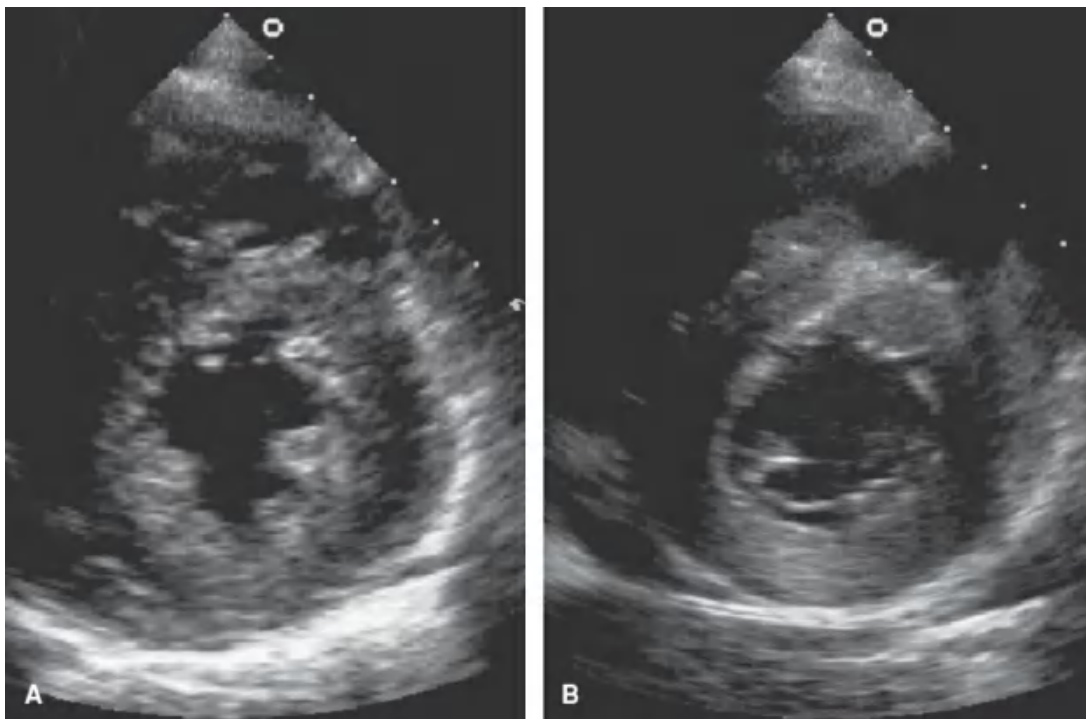


**FIGURE 2.8.** The effects of different transducer frequencies on image quality and

appearance are demonstrated. **A:** A 3-MHz transducer is used to record a short-axis view. **B:** The same image is recorded using a 5-MHz transducer.

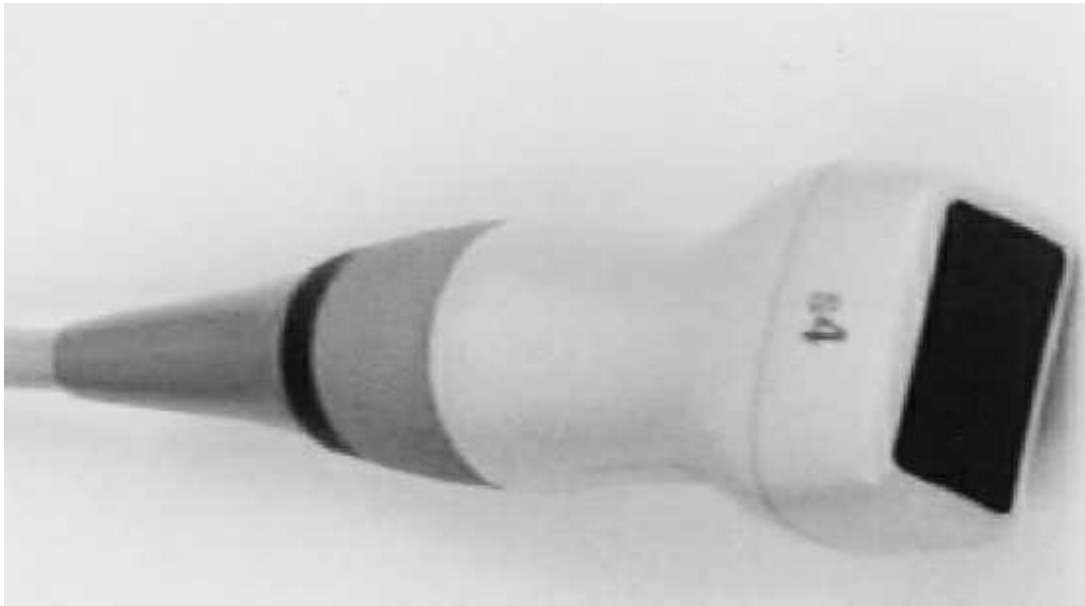


**FIGURE 2.9.** A phased-array ultrasound transducer is shown.



## 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*.

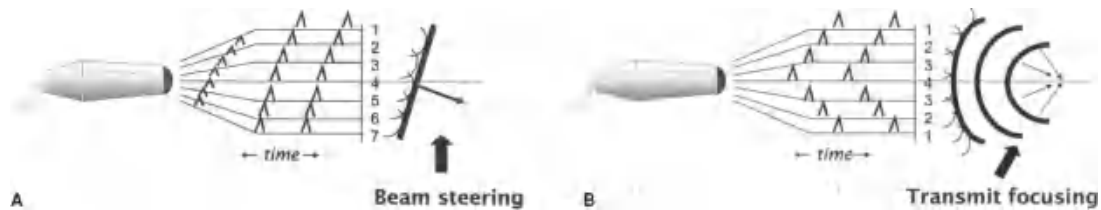


**FIGURE 2.10.** **A:** Phased-array technology permits steering of the ultrasound beam. By adjusting the timing of excitation of the individual piezoelectric crystals, the wave front of ultrasound energy can be directed, as shown. Beam steering is a fundamental feature of how two-dimensional images 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.

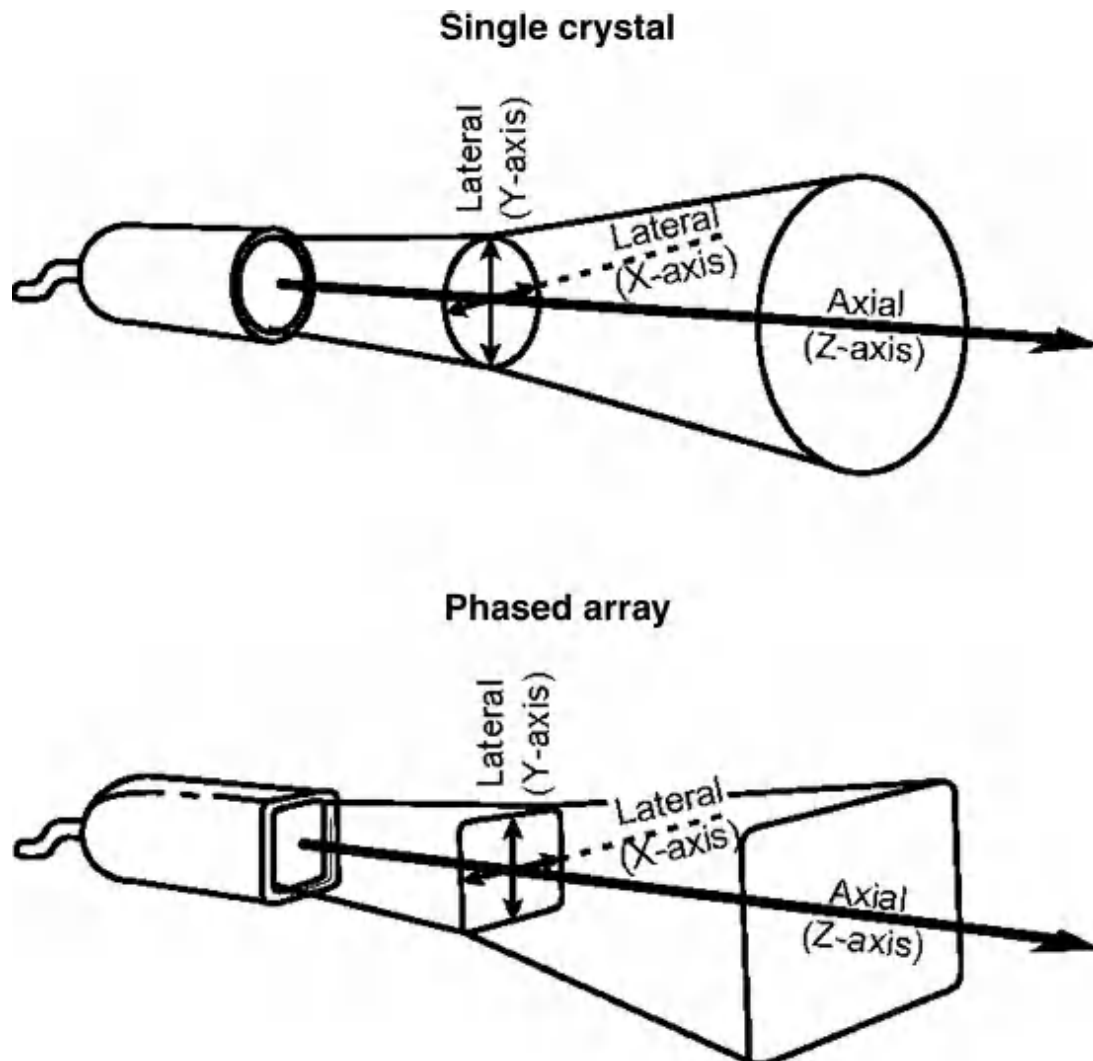


**FIGURE 2.11.** The ultrasound beam can be represented as a three-dimensional structure. A single-crystal transducer (**top**) will emit a cylindrically shaped beam. If the transducer face is rectangular shaped (**bottom**), the beam will also have a rectangular shape. The various beam axes are labeled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

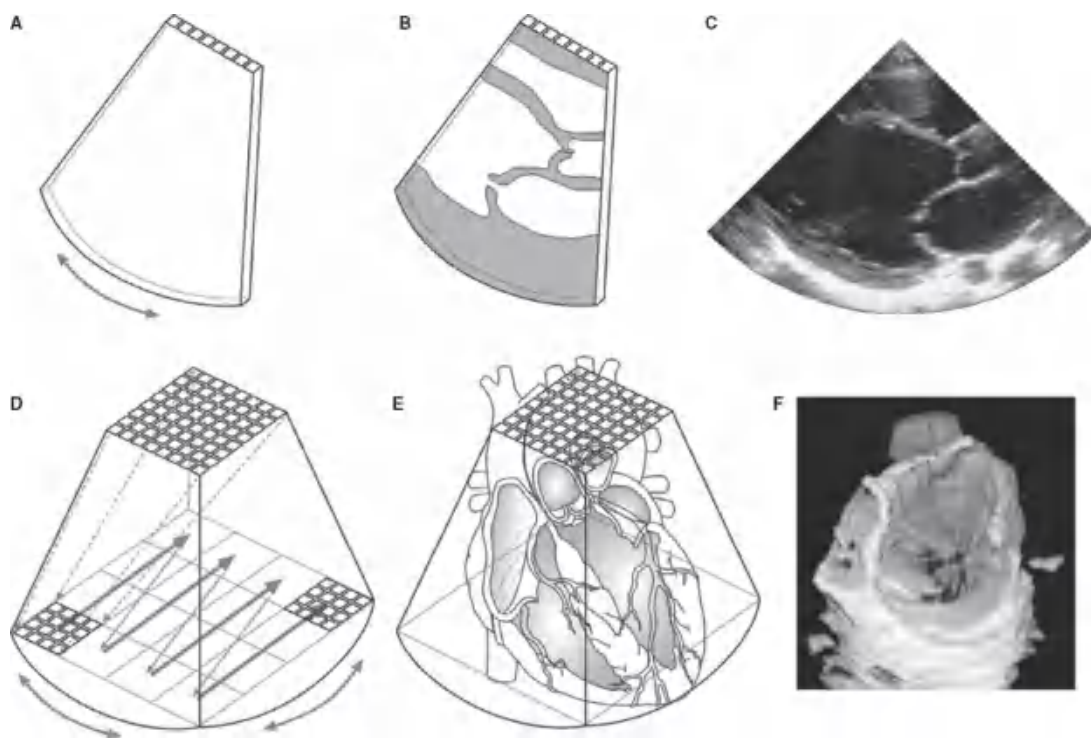


**FIGURE 2.12.** The relationship between two-dimensional and three-dimensional imaging is demonstrated in this schematic. In (A), the piezoelectric elements are arranged linearly, allowing the ultrasound beam to sweep through a sector arc to record a two-dimensional tomographic image of the left ventricle (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.

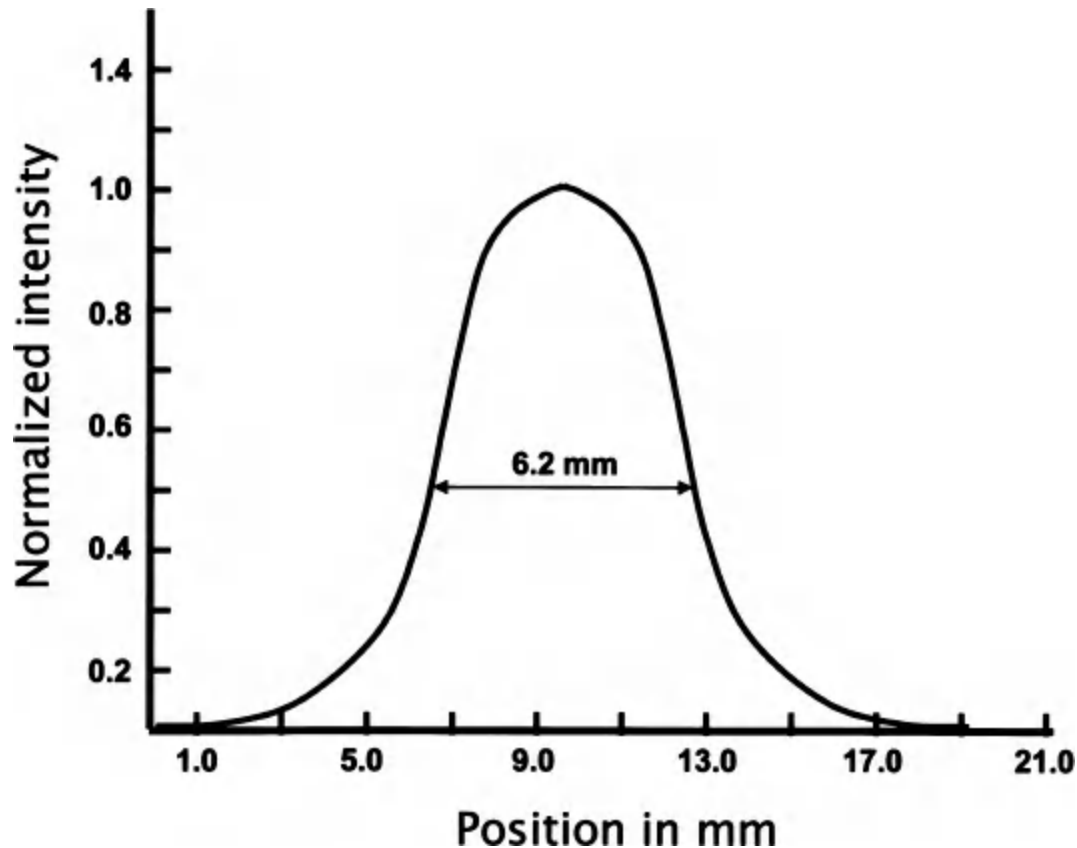


**FIGURE 2.13.** A transaxial beam plot is demonstrated. The beam width or lateral resolution is a function of the intensity of the ultrasonic beam. The beam width is commonly measured at the half-intensity level, and, in this case, the beam width would be reported as 6.2 mm.

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.

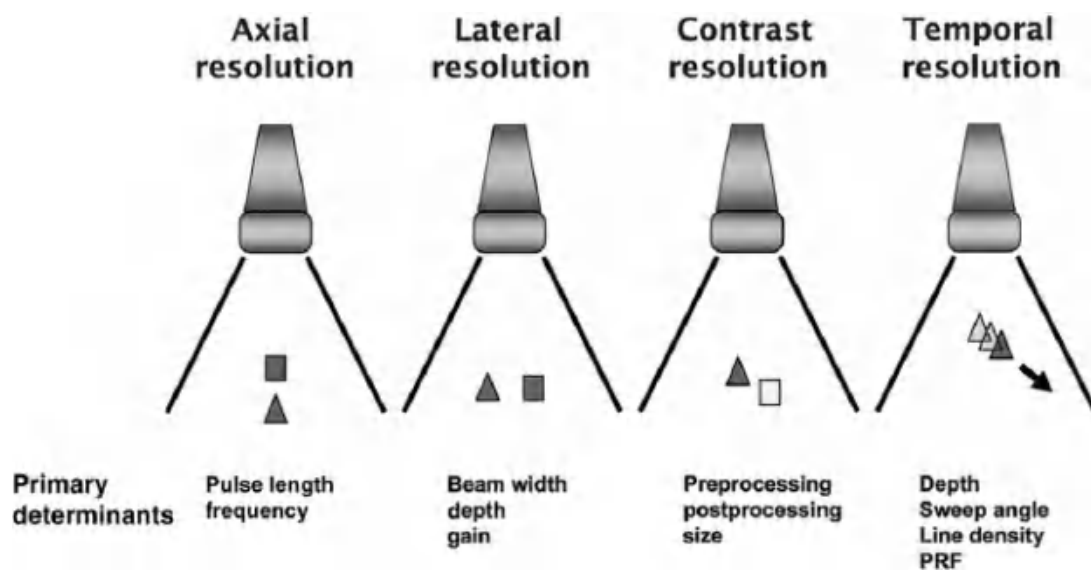


**FIGURE 2.14.** The different types of resolution are demonstrated in this schematic. See text for details.

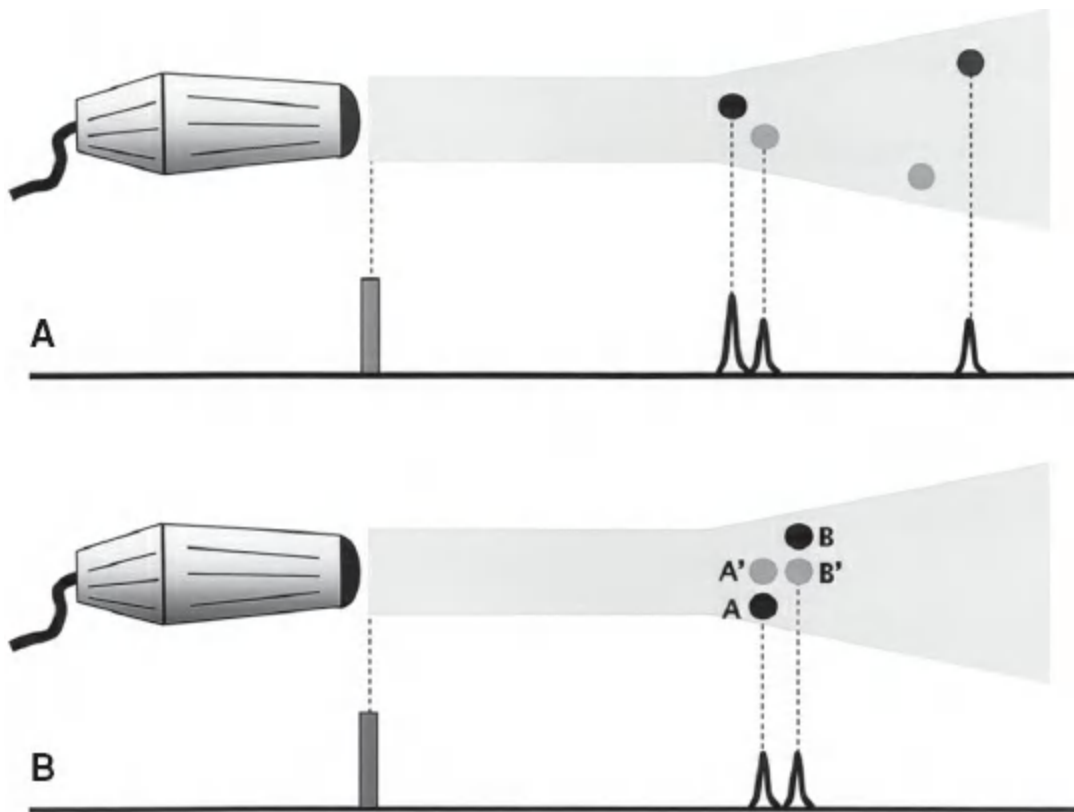


**FIGURE 2.15.** This schematic illustrates the interrelationship between beam intensity and acoustic impedance. The center of the beam has higher intensity compared with the edges. **A:** Whether an echo is produced, and with what amplitude it is recorded, depends on the relationship between intensity and acoustic impedance. Objects with higher impedance (*black dots*) produce stronger echoes and can therefore be detected even at the edges of the beam. Weaker echo-producing targets (*gray dots*) only produce echoes when they are located in the center of the beam. **B:** The effect of beam width on target location is shown. Objects A and B are nearly side by side with B slightly farther from the transducer. Because of the width of the beam, both objects are recorded simultaneously. The resulting echoes suggest that the two objects are directly behind each other (A' and B') rather than side by side.

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.

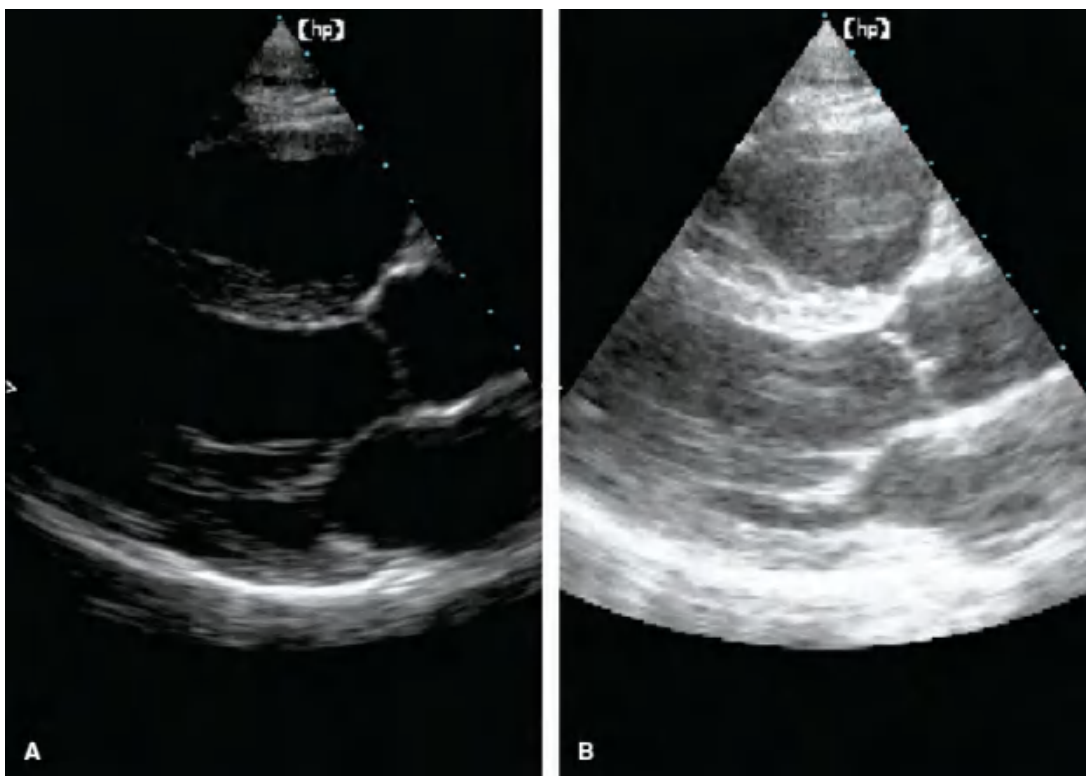


**FIGURE 2.16.** Parasternal long-axis images demonstrate the effect of gain on the appearance on the echocardiographic image. **A:** Gain is adjusted appropriately to allow recording of all relevant information. **B:** Too much gain is used, distorting the image, reducing resolution, and increasing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



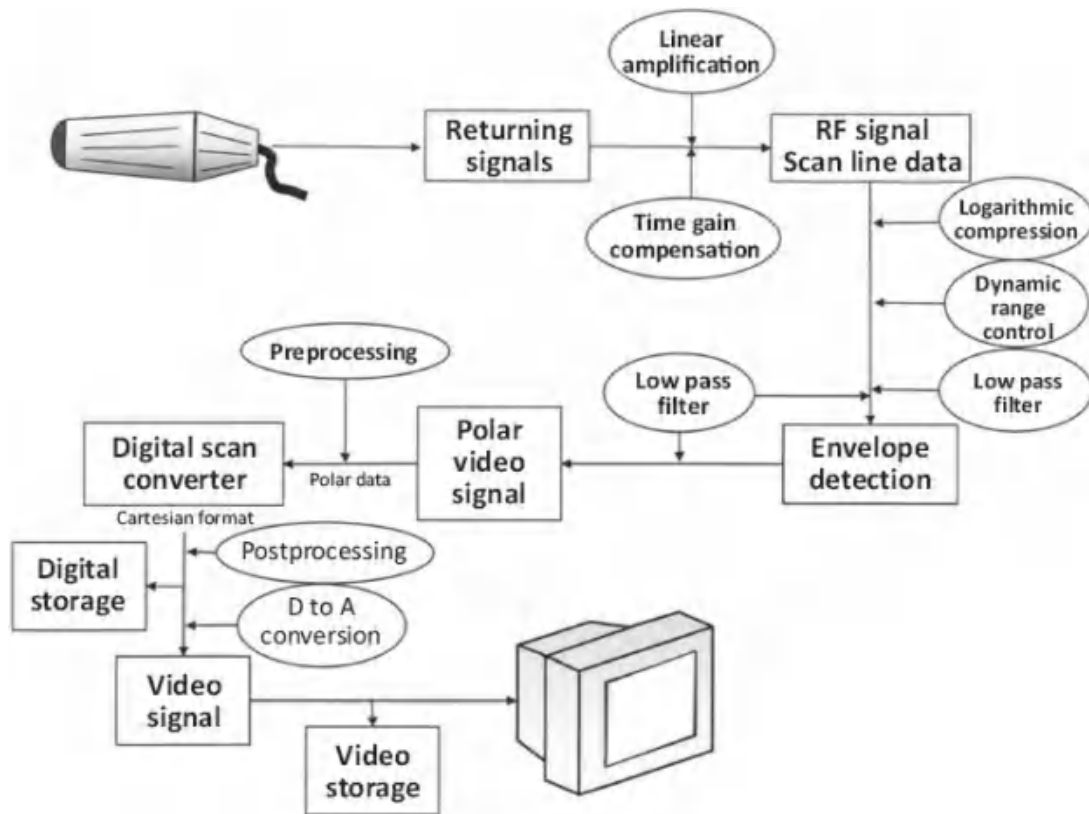


**FIGURE 2.17.** A block diagram shows the components of an echograph. The various steps needed to create an image, beginning at the transducer and continuing to the display, are included. See text for details.

## 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.

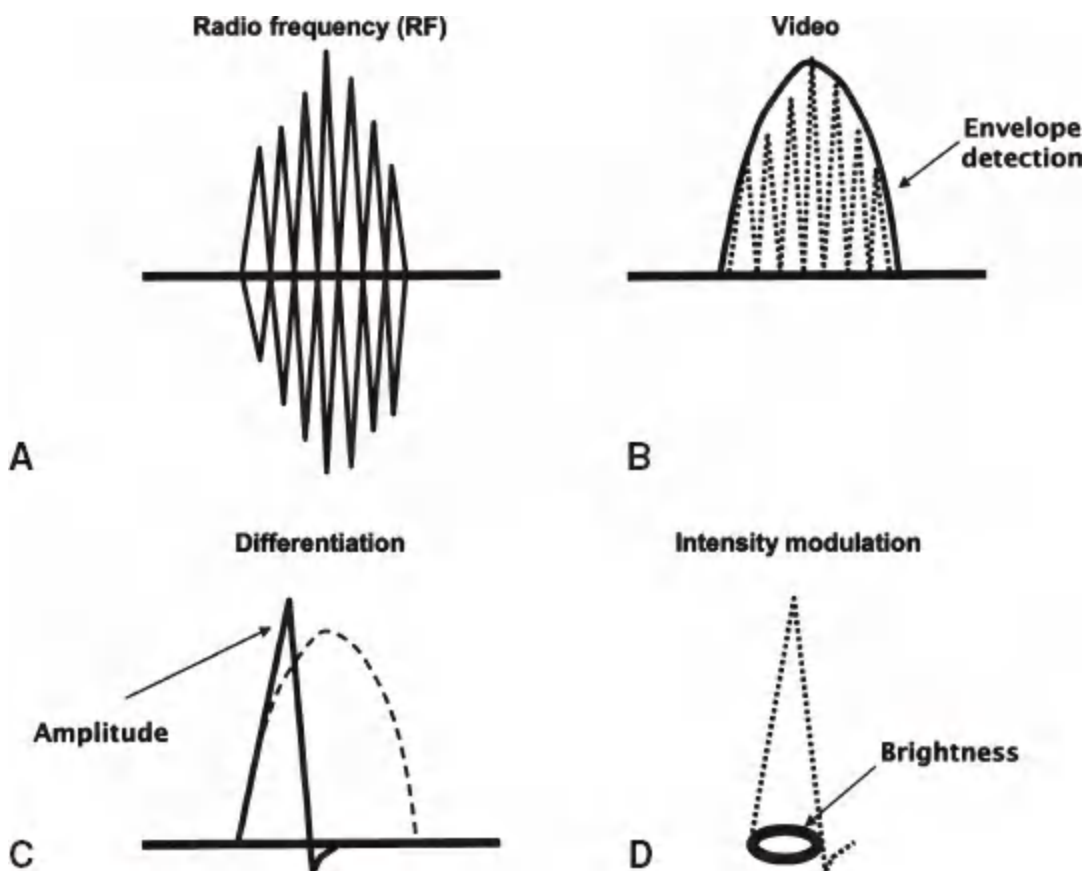


**FIGURE 2.18.** Some of the key steps in image creation are illustrated graphically. See text for details.

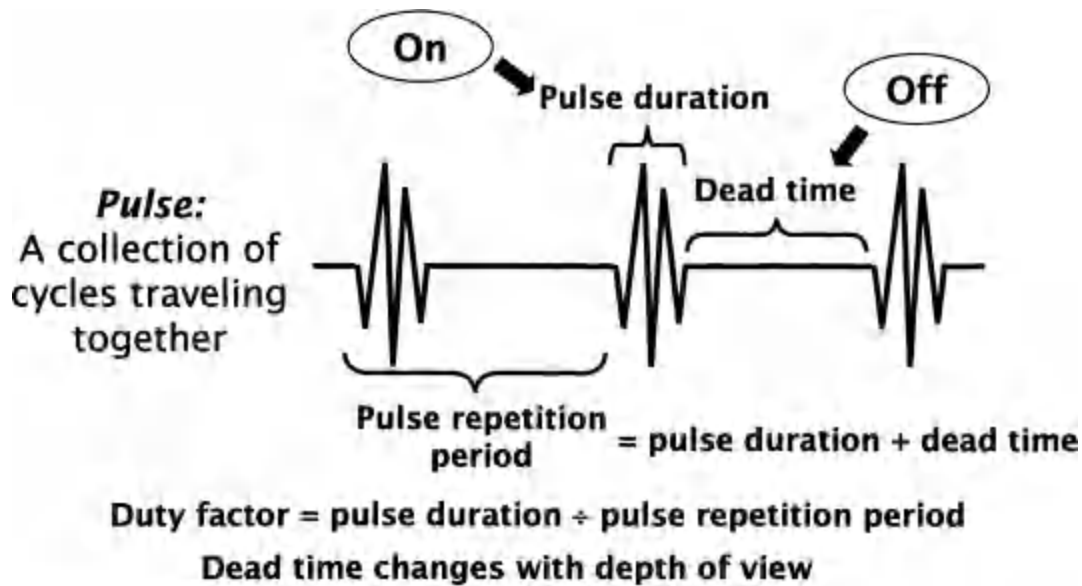
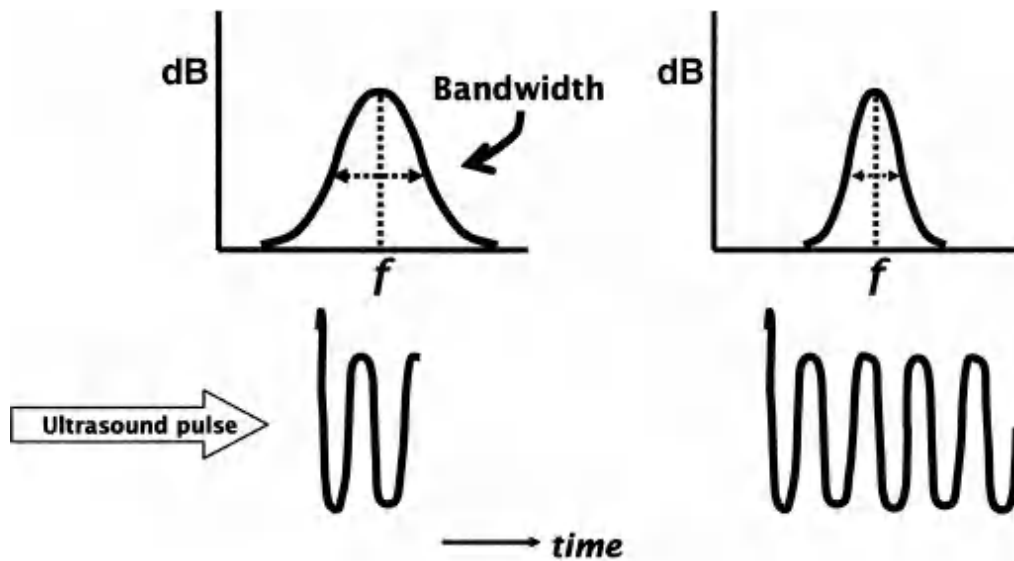


**FIGURE 2.19.** Ultrasound energy is usually emitted from the transducer in a series of pulses, each one representing a collection of cycles. Each pulse has a duration and is separated from the next pulse by the dead time. The diagram is not drawn to scale. In reality, dead time is much greater than 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**

**∴ Longer pulse length  $\Rightarrow$  narrower bandwidth  $\Rightarrow$  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.

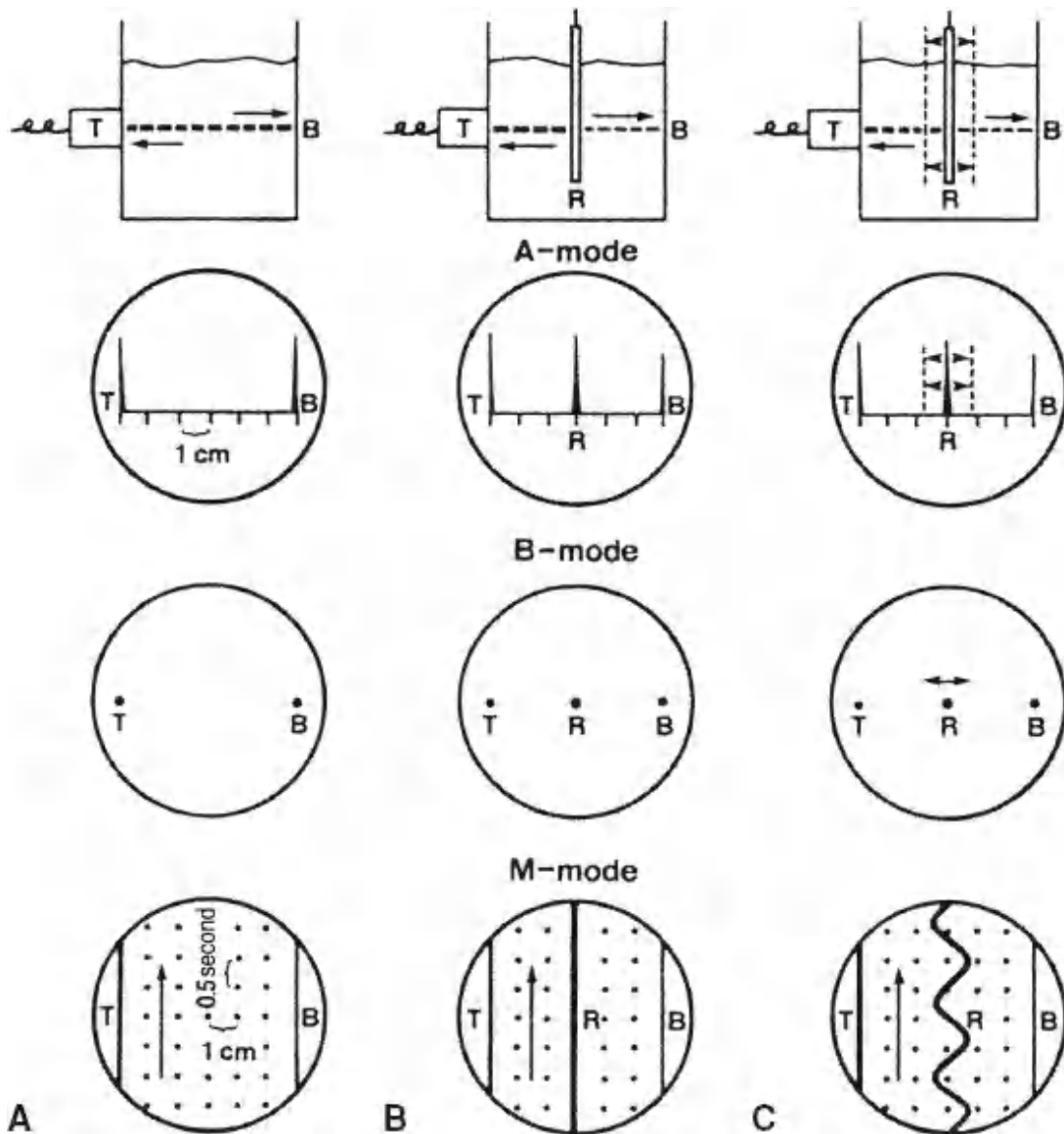


**FIGURE 2.21.** The basic principles of pulsed ultrasound are demonstrated in this schematic. See text for details. T, transducers; B, beaker; R, rod. (From Feigenbaum H, Zaky A. Use of diagnostic ultrasound in clinical cardiology. *J Indiana State Med Assoc* 1966;59:140, with permission.)

## DISPLAY OPTIONS

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.

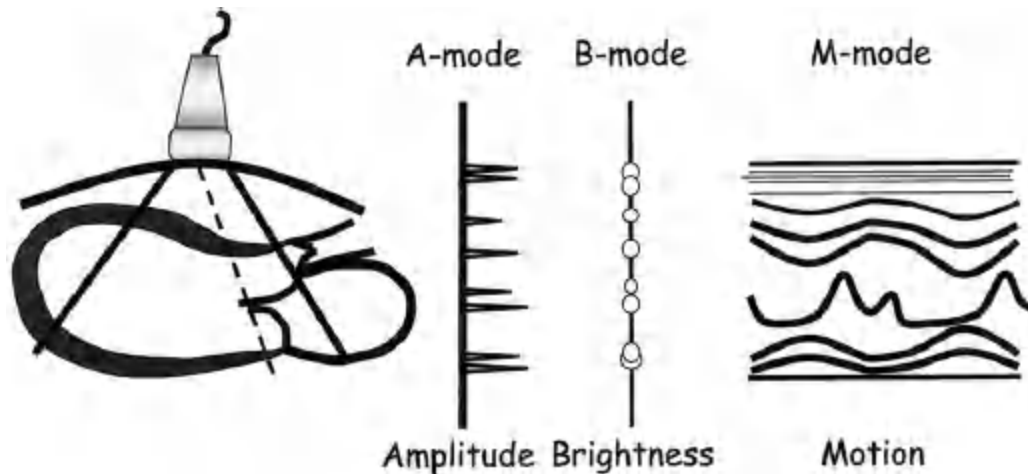


**FIGURE 2.22.** Echocardiography provides several display options. **Left:** A transducer is applied to the chest wall, and an ultrasound beam is directed through the heart at the level of the mitral valve. The returning ultrasound information can be displayed in amplitude mode (A-mode) in which the 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.

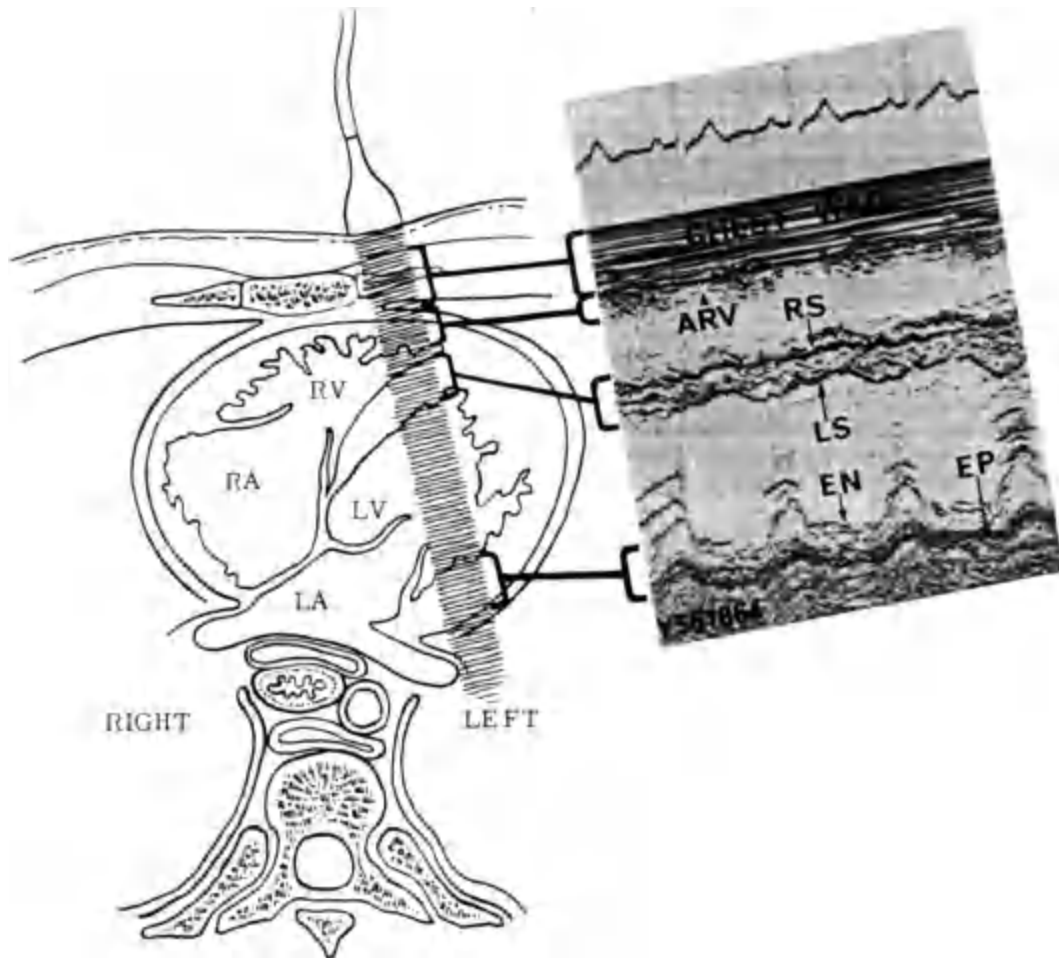


**FIGURE 2.23.** M-mode echocardiography is often described as an “ice-pick” view of the heart. The diagram shows the relationship of the transducer to the structures of the chest wall and heart. The corresponding M-mode echocardiogram provides relative anatomic information along a single line of information. 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.

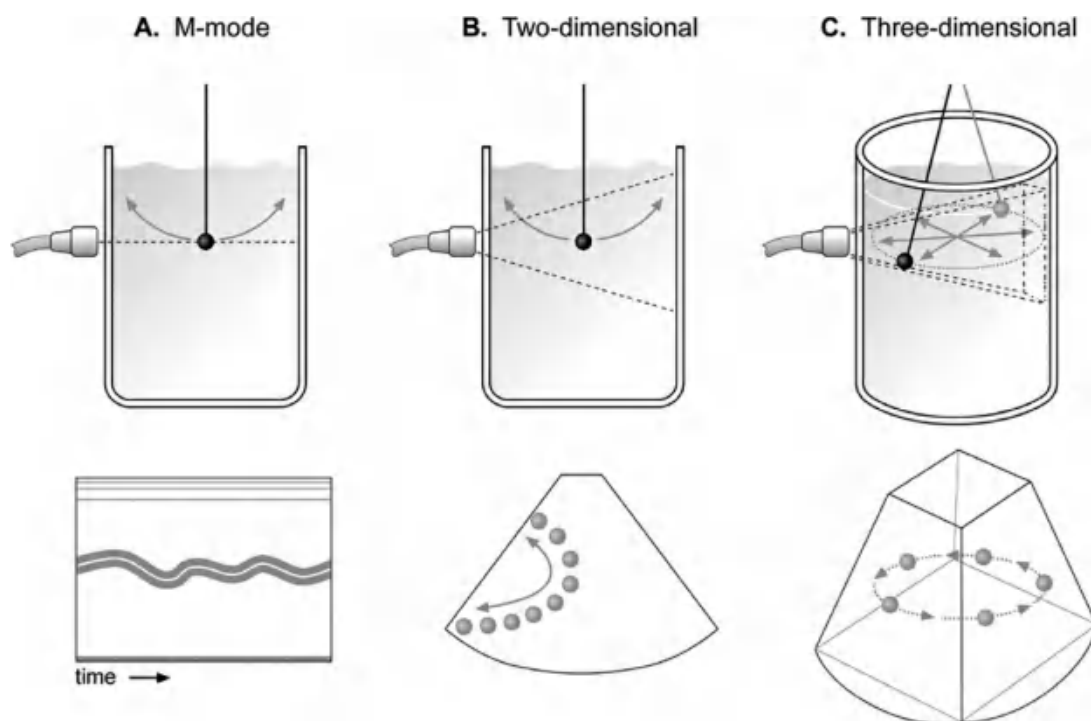


**FIGURE 2.24.** The relationship between M-mode and two-dimensional echocardiography is demonstrated. In (A), a circular object swings through a beaker of water on a string. The motion of the ball is recorded using M-mode echocardiography, as shown below. Only motion in a single dimension, relative 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

(Fig. 2.26). 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.

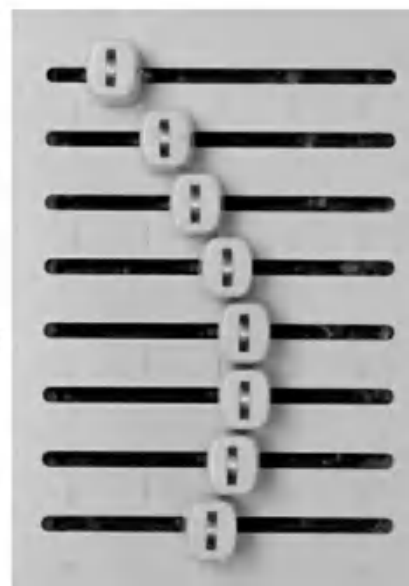
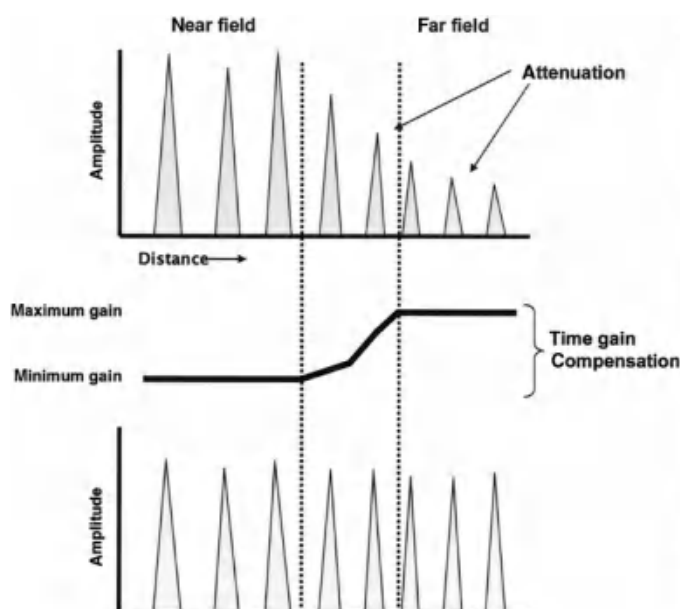


**FIGURE 2.25.** The amplitude of returning signals is plotted against distance, or depth, from the transducer. Time gain compensation can be used to enhance the amplitude of the weaker signals returning from targets at greater depth and permits similar targets at different depths to be displayed accurately. On the right, the time gain compensation controls from an ultrasound machine are shown.

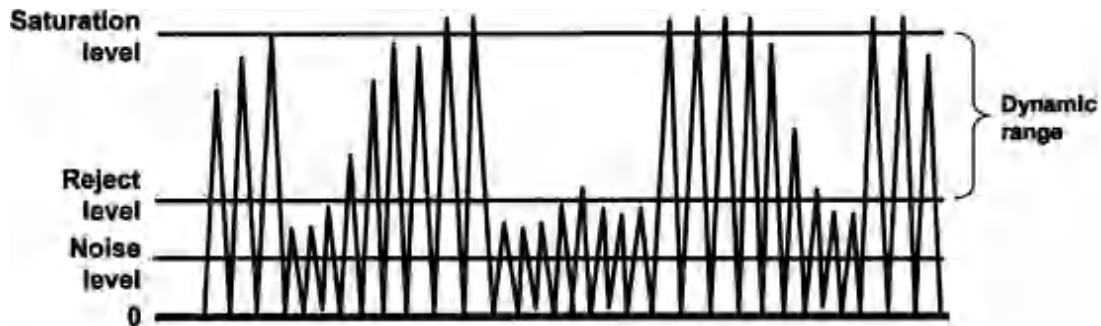


**FIGURE 2.26.** The schematic illustrates the concept of dynamic range. See text for details.

## 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.

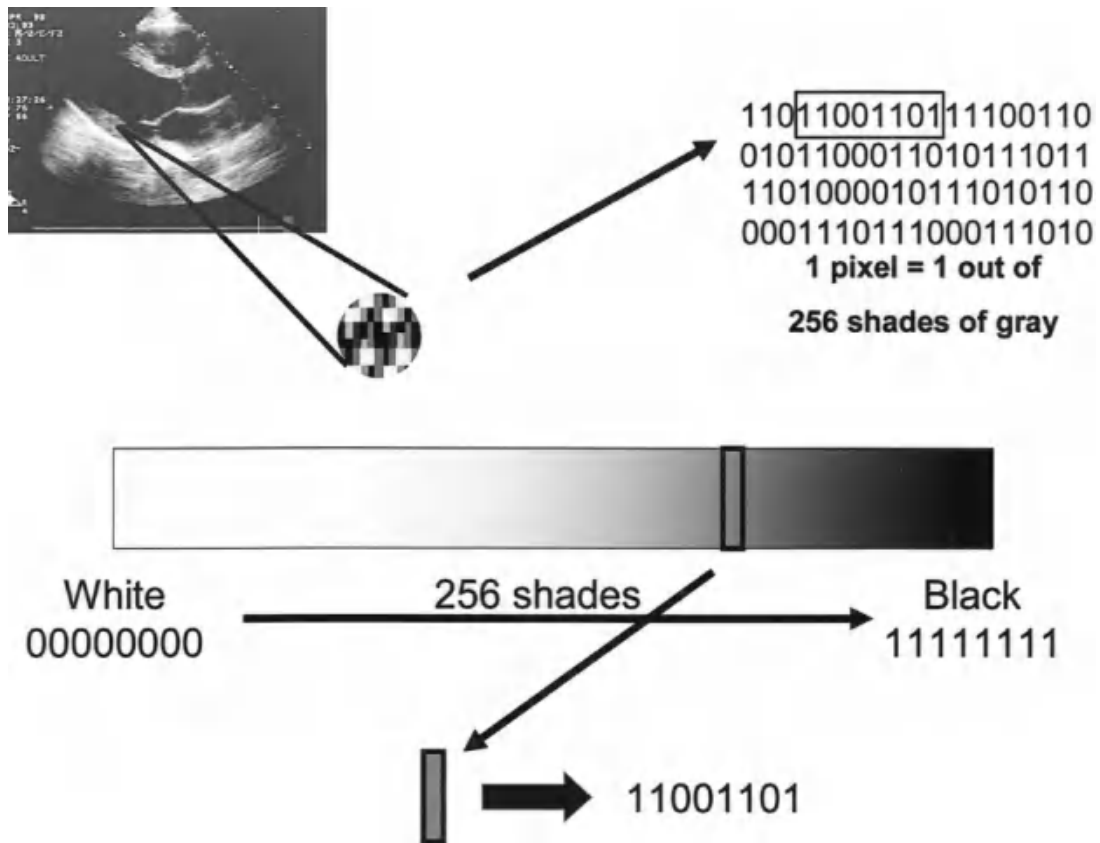


**FIGURE 2.27.** Gray scale is a key concept in the creation of a two-dimensional image. The gray scale refers to the number of shades that can possibly be displayed between the two extremes of white and black. In the example, 256 shades are depicted. Each pixel is assigned one of these shades. In a 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.

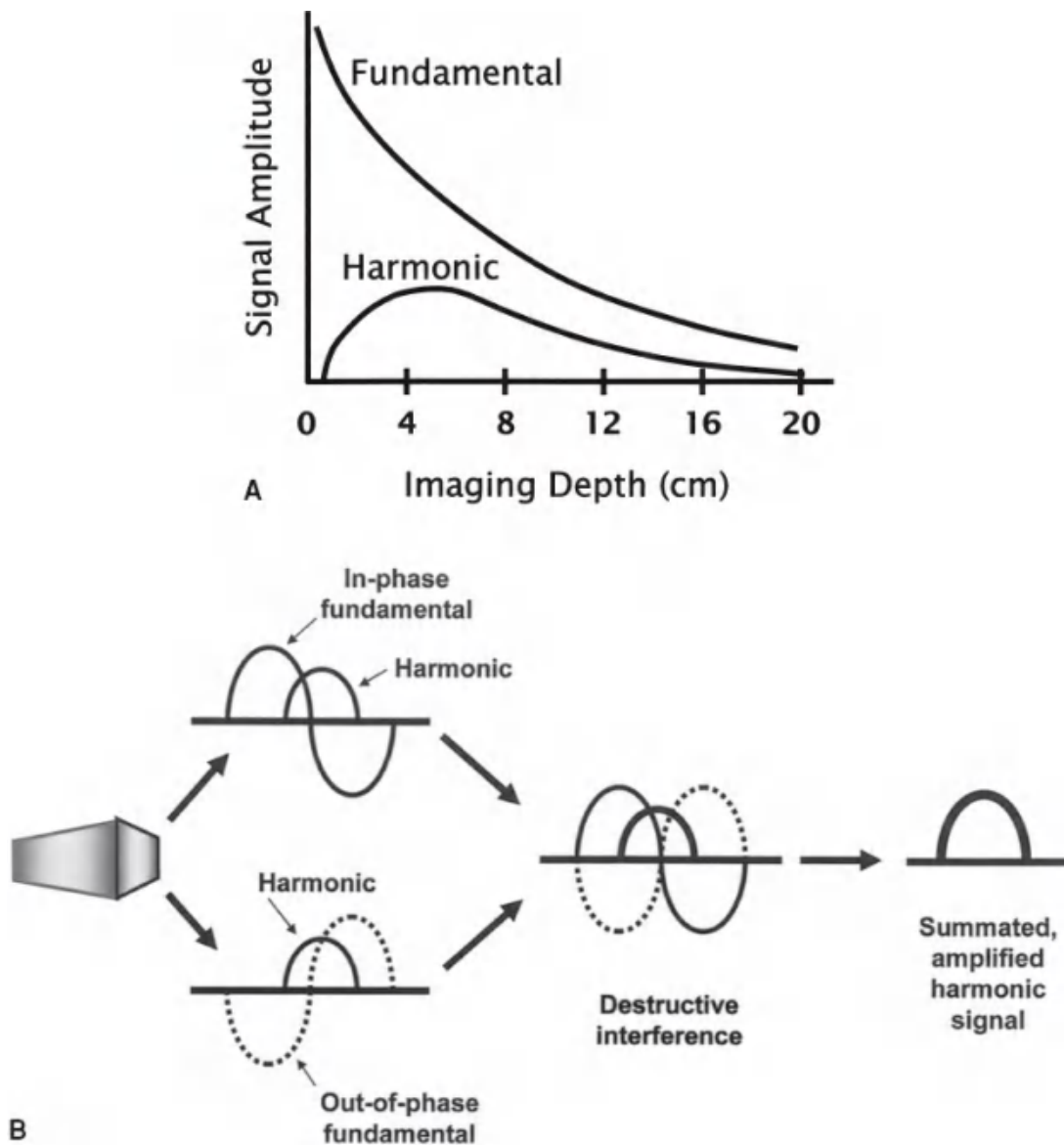


**FIGURE 2.28. A:** Unlike fundamental frequencies, harmonic frequencies increase in strength as the wave penetrates the body. At the chest wall, where many artifacts are generated, very little harmonic signal is present. At useful imaging depths (4 to 8 cm), the relative strength of the harmonic signal 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.

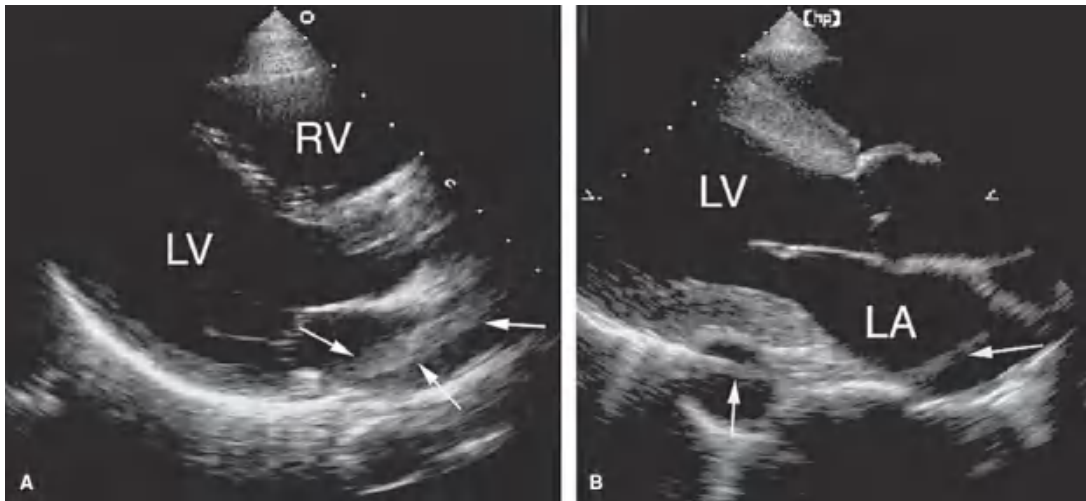


**FIGURE 2.29.** Two examples of side lobes are shown (*arrows*). **A:** The strong echoes produced by the posterior mitral annulus and atrioventricular groove produce a side lobe artifact that appears as a mass within the left atrium. **B:** Bright echoes within the pericardium produce a linear artifact that appears within the descending aorta and left atrium.

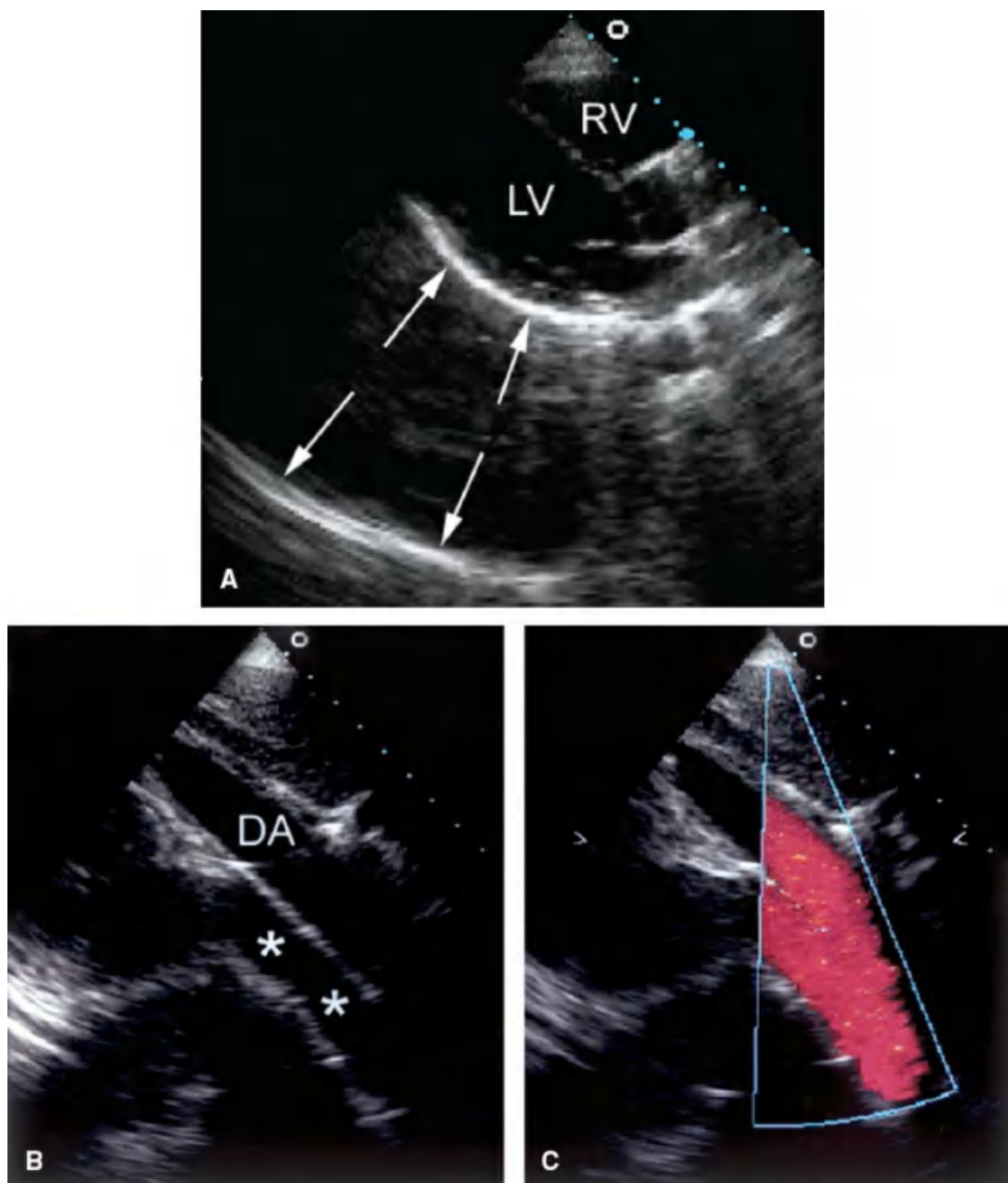


**FIGURE 2.30.** Reverberation artifacts are demonstrated. **A:** The source of the artifact is the posterior pericardium, which is a very strong reflector. This creates the illusion of a second structure behind the heart. In this case, the second line of echoes (*far arrows*) is twice the distance from 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**). 

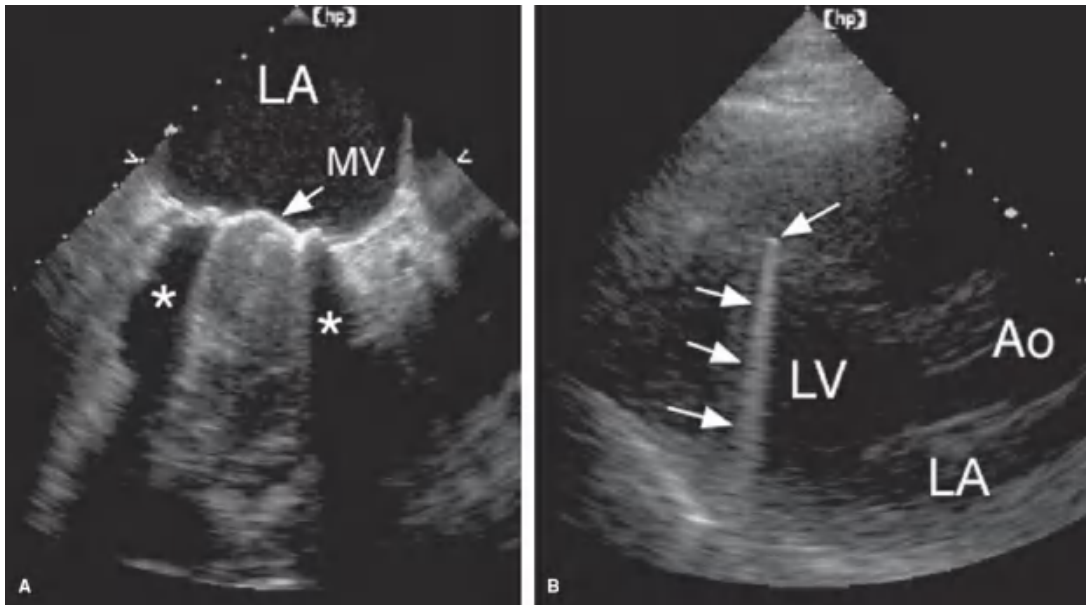


**FIGURE 2.31.** The concept of shadowing is demonstrated and compared with reverberations. **A:** A St. Jude mitral prosthesis (MV) is present. The echo-free space beyond the sewing ring (\*) represents shadowing behind the strong echo-reflecting sewing ring. The cascade of echoes directly beyond the prosthetic valve itself that extend into the LV represent reverberations. **B:** A shotgun pellet within the heart (*upper arrow*) casts a series of reverberations into the left ventricle (*lower arrows*). 

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.

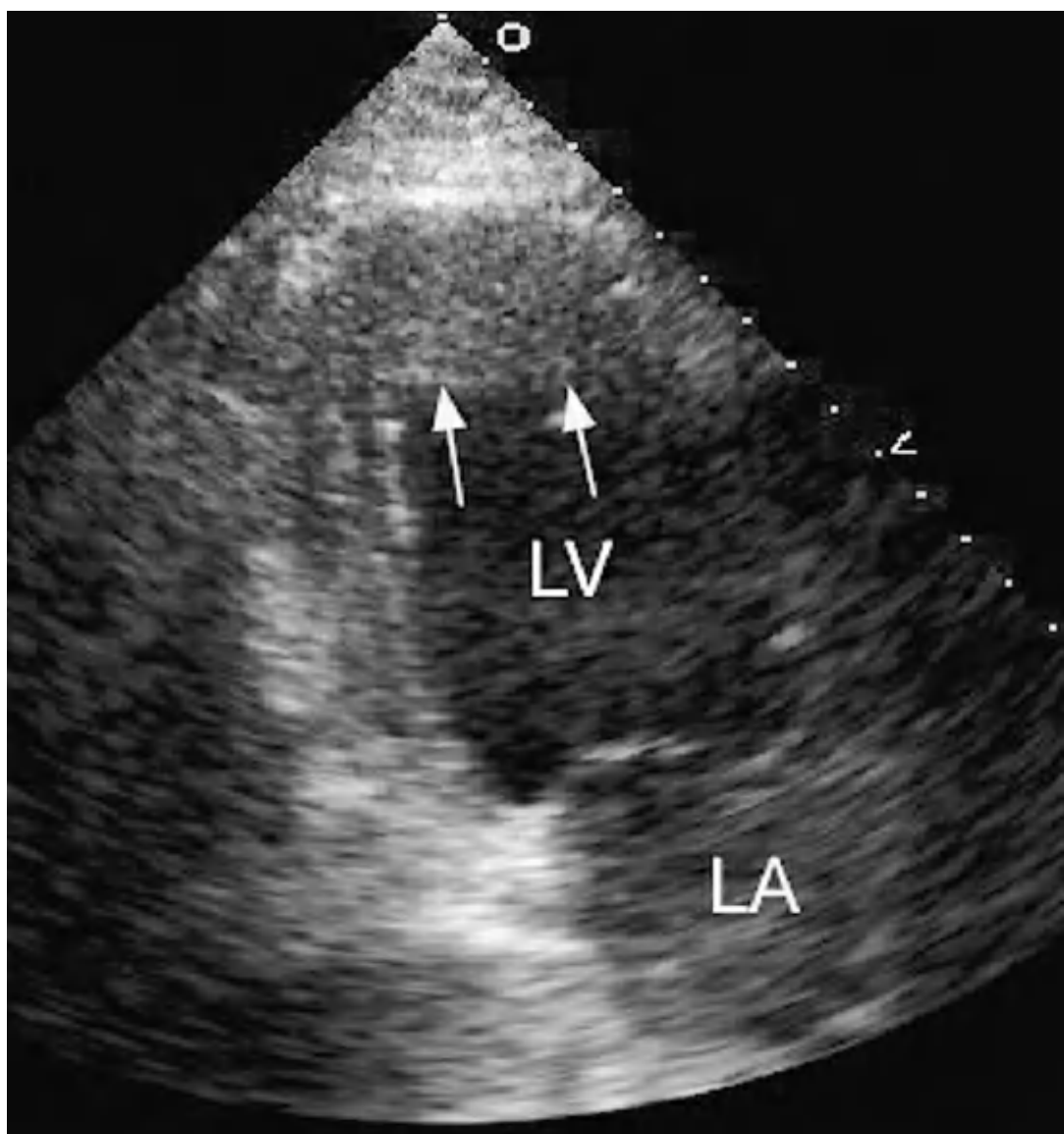


**FIGURE 2.32.** This apical two-chamber view demonstrates an artifact called near-field clutter (*arrows*). This is the result of high-amplitude oscillations emitted by the transducer and is a common source of misinterpretation. 📺

**Table 2.4**

**A COMPARISON OF TWO-DIMENSIONAL ECHOCARDIOGRAPHY  
AND DOPPLER**

|                   | Two-dimensional<br>Echocardiography | Doppler    |
|-------------------|-------------------------------------|------------|
| Ultrasound target | Tissue                              | Blood      |
| Goal of diagnosis | Anatomy                             | Physiology |

|                                           |               |            |
|-------------------------------------------|---------------|------------|
| Type of information                       | Structural    | Functional |
| Optimal alignment between beam and target | Perpendicular | Parallel   |
| Preferred transducer frequency            | High          | Low        |

## 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*.

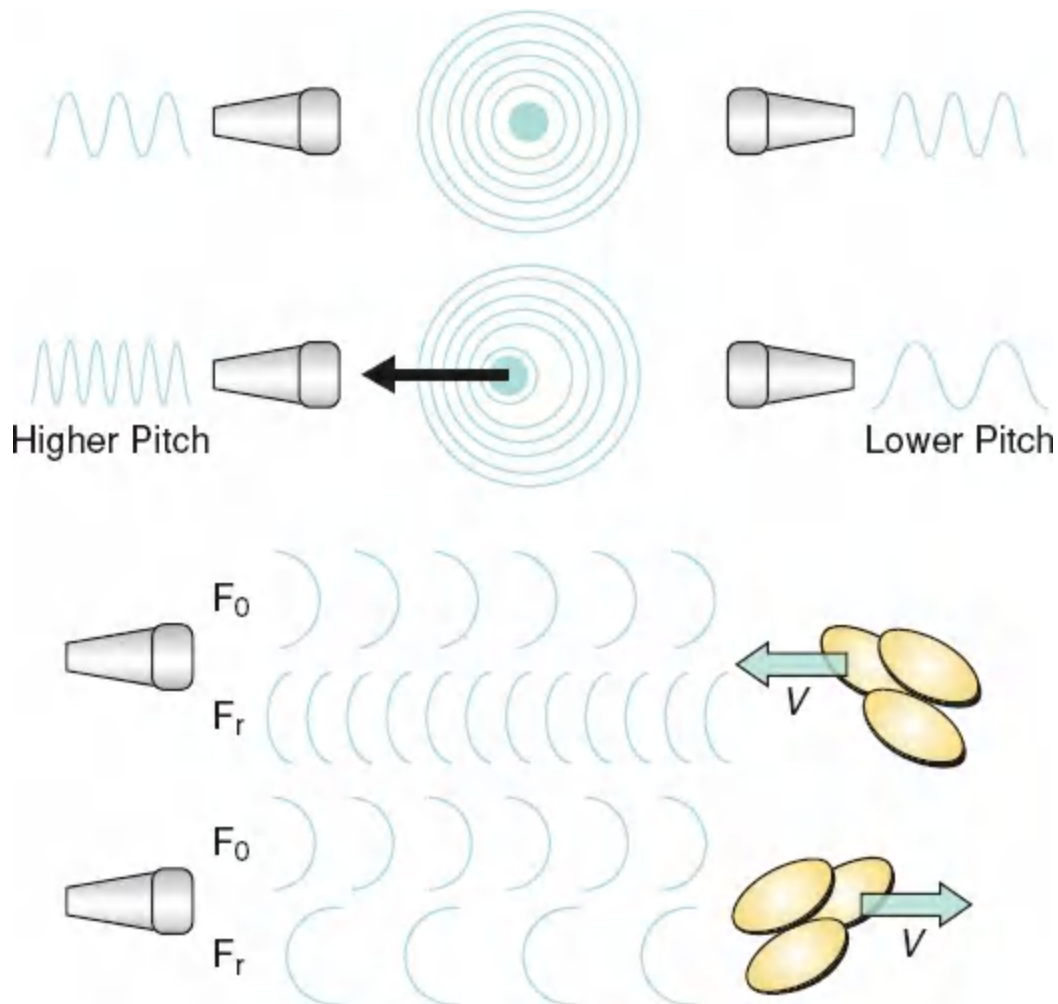


**FIGURE 2.33.** The basic principles of the Doppler phenomenon are illustrated. **Top:** A stationary source of sound produces a given pitch or frequency. If the sound is moving toward a recorder, the pitch appears increased and if the sound is moving away from a recorder, the pitch appears decreased. **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.

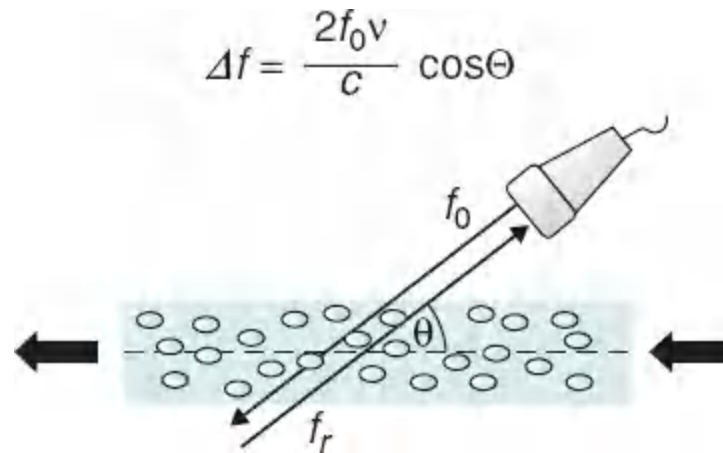


**FIGURE 2.34.** Calculation of the Doppler shift requires knowledge of the transmitted frequency ( $f_0$ ), the reflected frequency ( $f_r$ ), the angle of incidence ( $\theta$ ), and the speed of sound. See text for details.

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.

$$\Delta F = \frac{2f_0 v}{c} \times \cos \theta \quad [\text{Eq. 2.3}]$$

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,  $\theta$ .

$$\Delta f \propto v \times \cos \theta \quad [\text{Eq. 2.4}]$$

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  $\theta$  decreases from 1 to 0. The relationship between  $\theta$  and cosine  $\theta$  is shown in [Figure 2.35A](#). For any angle other than 0, multiplying by the cosine  $\theta$  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  $\theta$  equals 10 degrees, cosine  $\theta$  equals approximately 0.98 and the degree of underestimation is trivial. As  $\theta$  increases to 30 degrees, cosine  $\theta$  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  $\theta$  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 ( $\theta$ ) of 30 degrees, the calculated gradient will be approximately 75 mm Hg, a significant underestimation.

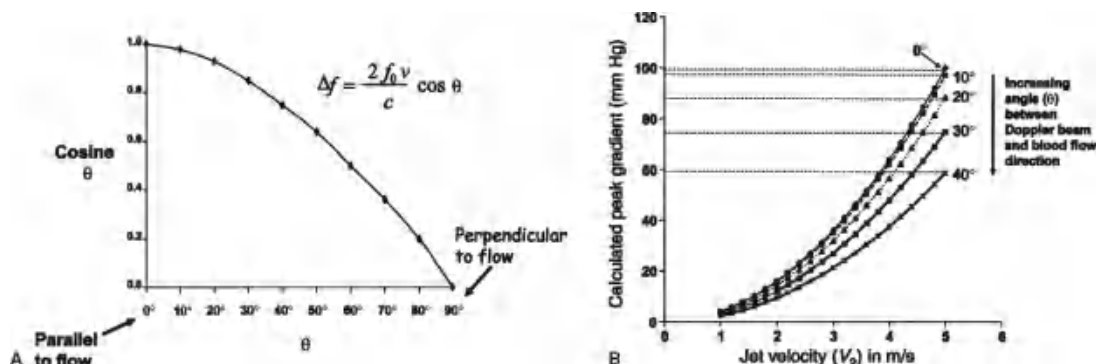


**FIGURE 2.35. A:** The effect of intercept angle on the Doppler equation is shown. See text for details. **B:** The intercept angle has an important effect on the accuracy of velocity measurement. This effect is magnified at higher velocity and becomes increasingly important as the intercept angle increases 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

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.

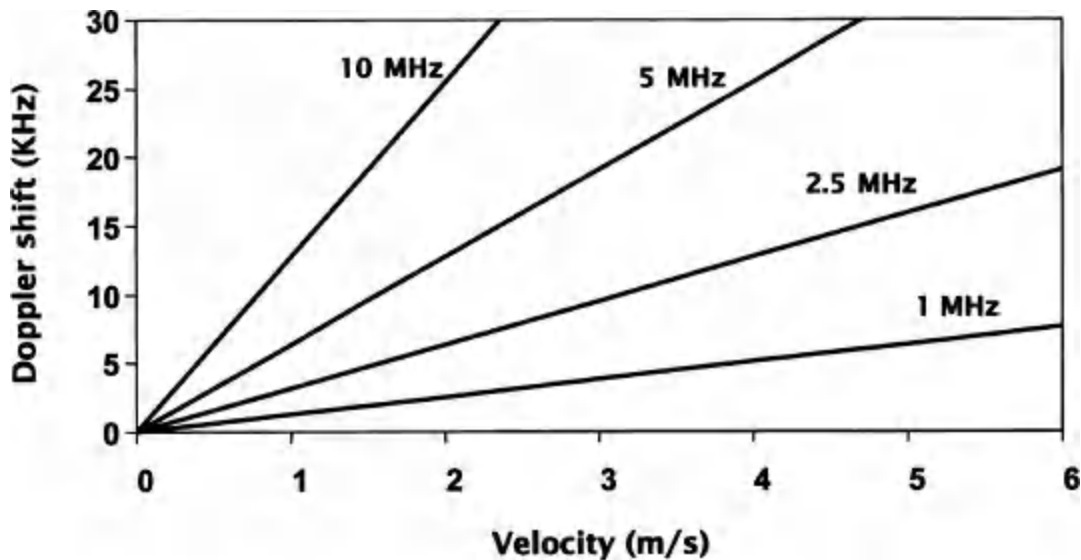


**FIGURE 2.36.** The relationship between the Doppler shift and blood flow velocity for four different transducers is shown. The graph demonstrates that lower-frequency transducers are capable of resolving higher-velocity flow. See text for details.

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:

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

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.

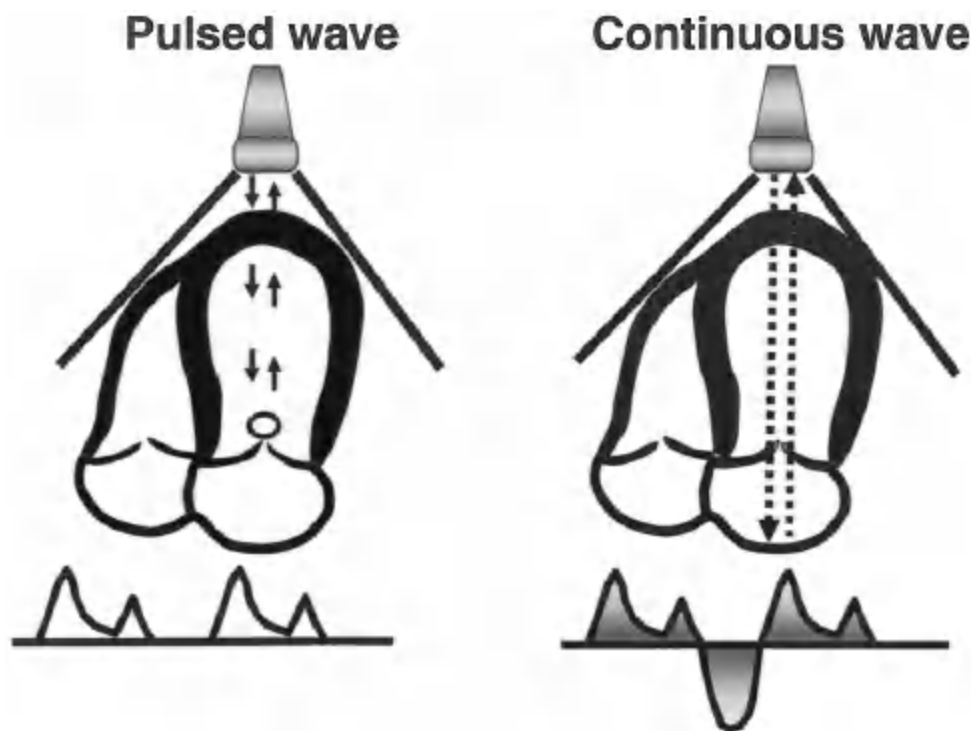


**FIGURE 2.37.** The differences between pulsed- and continuous-wave Doppler imaging are illustrated. See text for details.

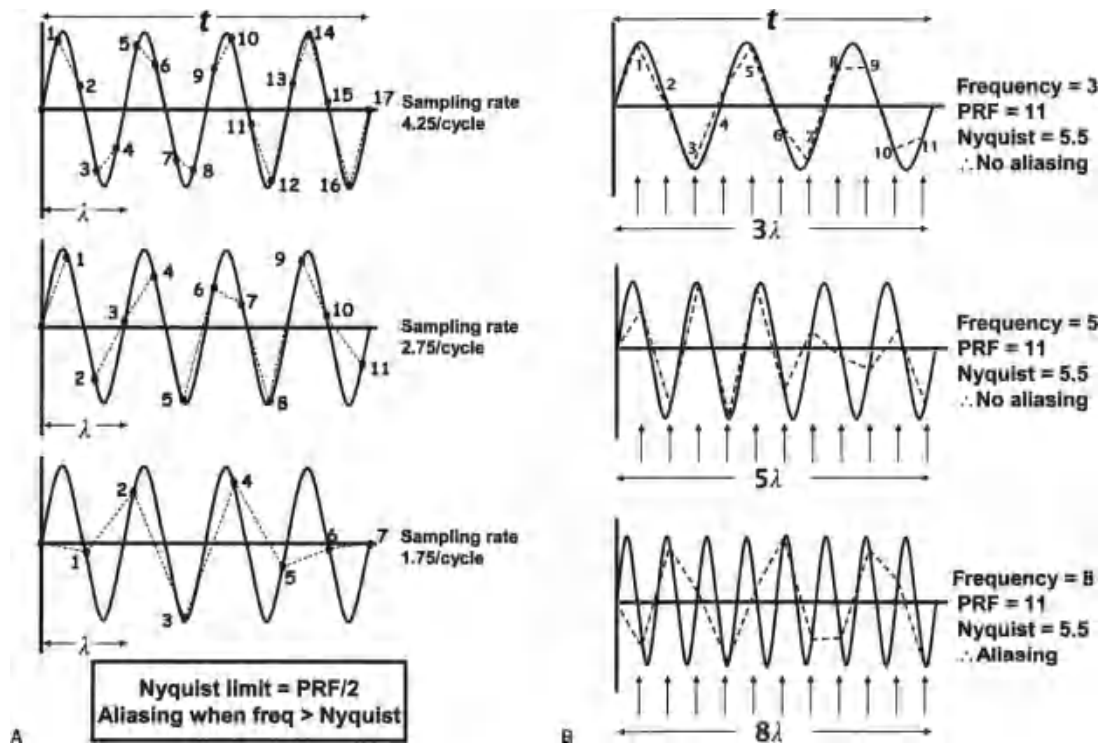


**FIGURE 2.38.** The concept of aliasing is demonstrated graphically in this schematic. See text for details.

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.

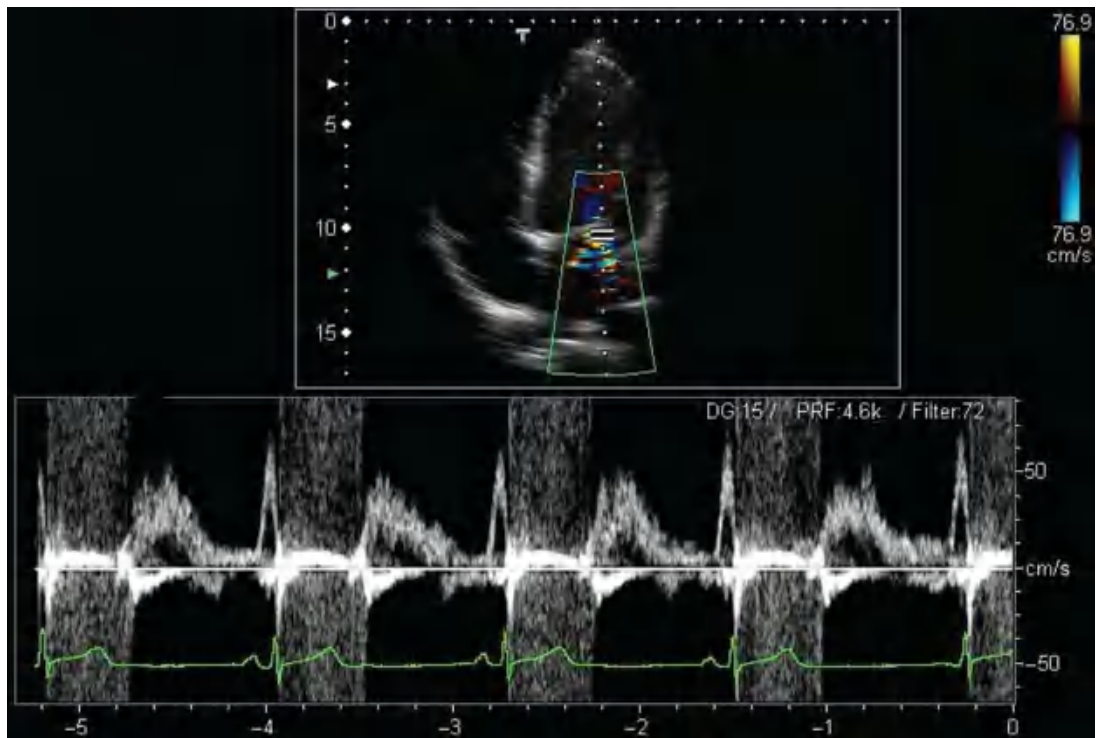


**FIGURE 2.39.** An example of aliasing is provided. Using pulsed-wave Doppler imaging, the sample volume is placed in the left atrium, just beyond the mitral valve. In systole, mitral regurgitation produces a high-velocity jet that cannot be resolved with the pulsed-wave Doppler technique. Aliasing of the jet is the result.

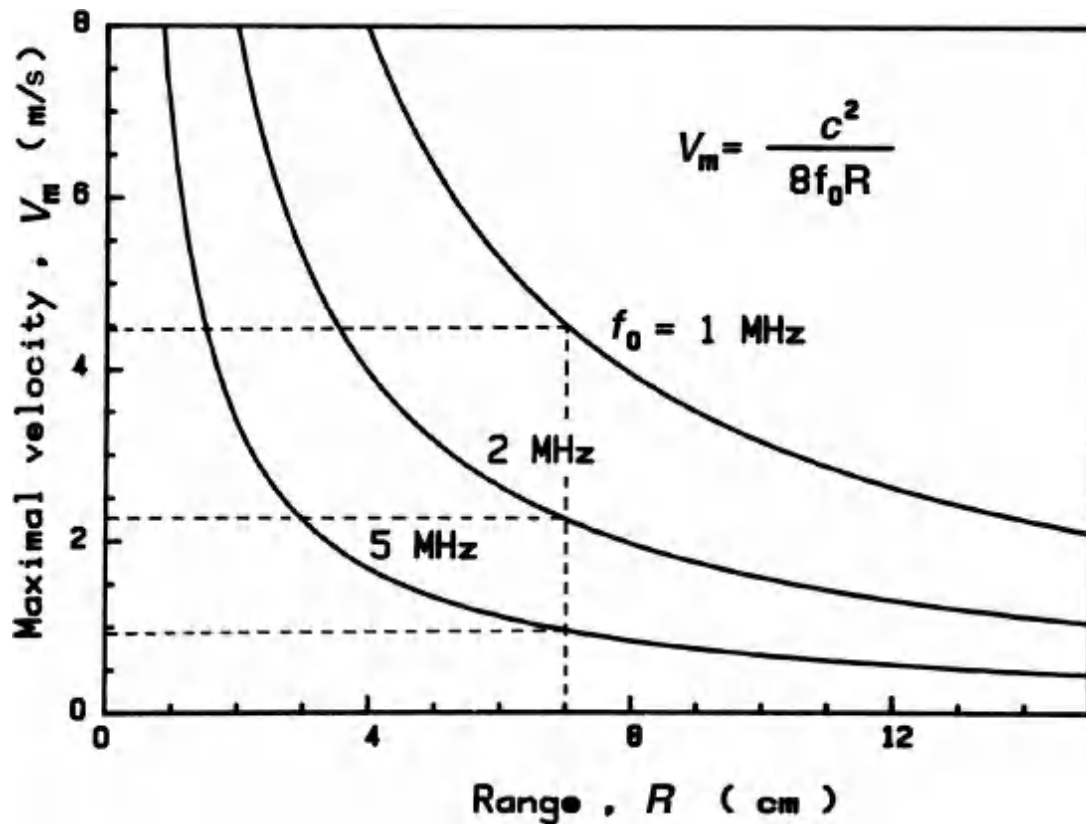


**FIGURE 2.40.** This graph demonstrates the relationship between range, or depth, and the maximal velocity that can be resolved, using two different transducer frequencies. The relationship is given by the equation. In both cases, as depth increases, the maximal velocity that can be recorded decreases. However, for any given depth, the lower-frequency transducer is capable of resolving higher velocities compared with the higher-frequency transducer.  $V_{\max}$ , maximal velocity;  $c$ , velocity of ultrasound;  $f_0$ , transducer frequency;  $R$ , range. (From Hatle L, Angelsen B. *Doppler Ultrasound in Cardiology: Physical Principles and Clinical Applications*. 2nd ed. Philadelphia, PA: Lea & Febiger; 1985, with permission.)

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.

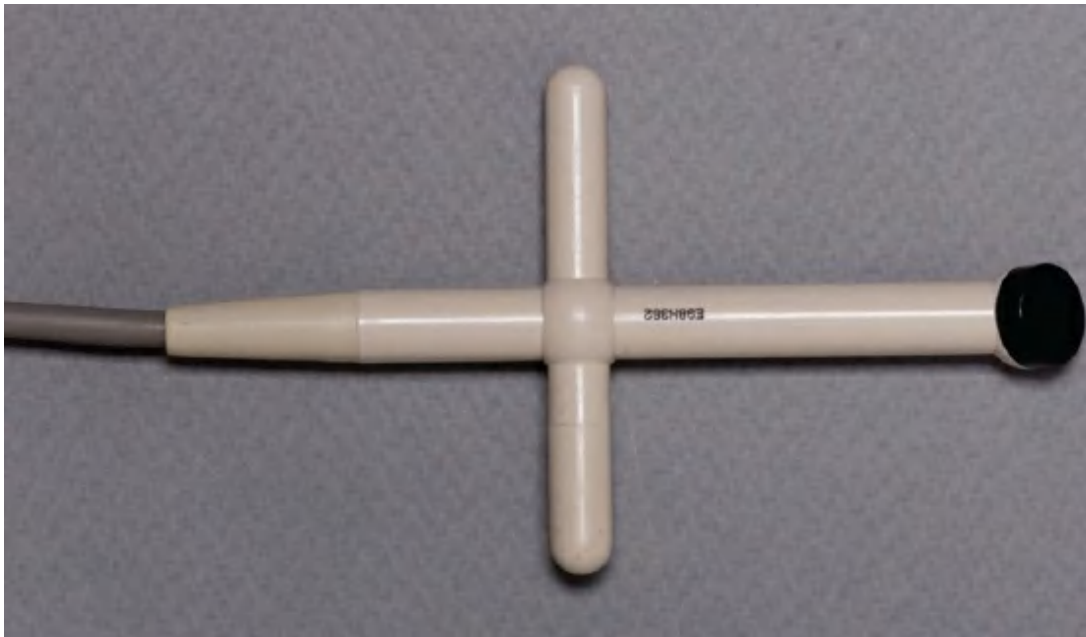


**FIGURE 2.41.** A nonimaging, or Pedoff, continuous-wave Doppler transducer is shown. The transducer contains two elements: one for transmitting and one for receiving.

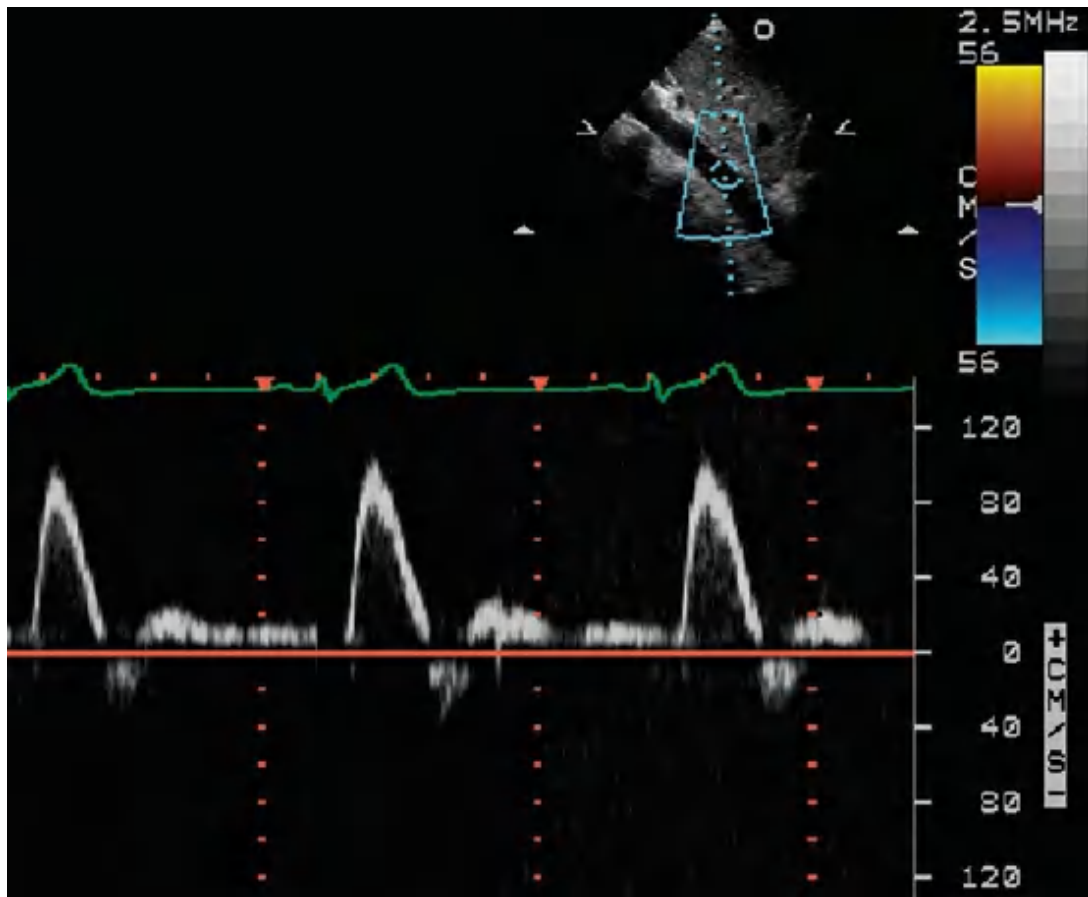


**FIGURE 2.42.** Laminar, pulsatile flow in the abdominal aorta is recorded with pulsed-wave Doppler imaging. The signal demonstrates a narrow envelope during systole. The maximal velocity of the flow is approximately 100 cm/s. Note, however, that the ultrasound beam is not parallel to the flow direction.

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.

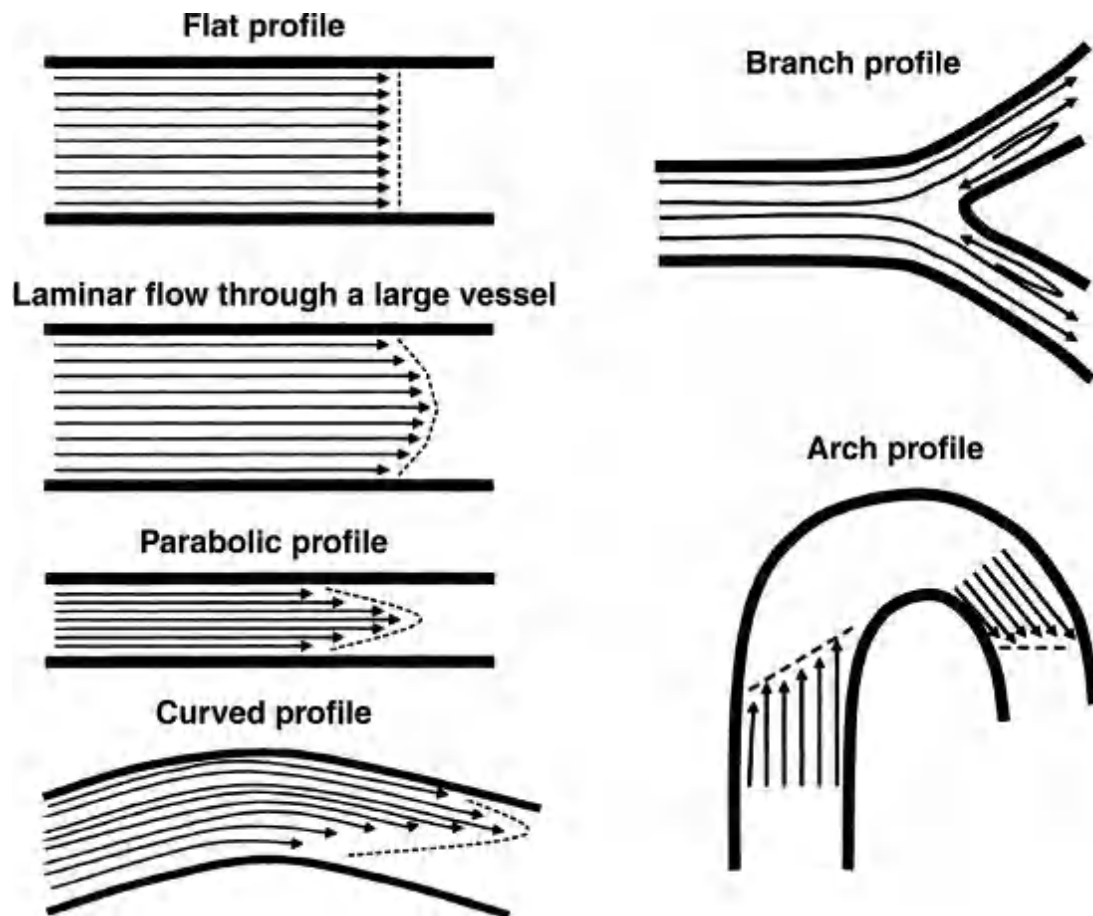


**FIGURE 2.43.** Various types of flow patterns are shown. See text for details.



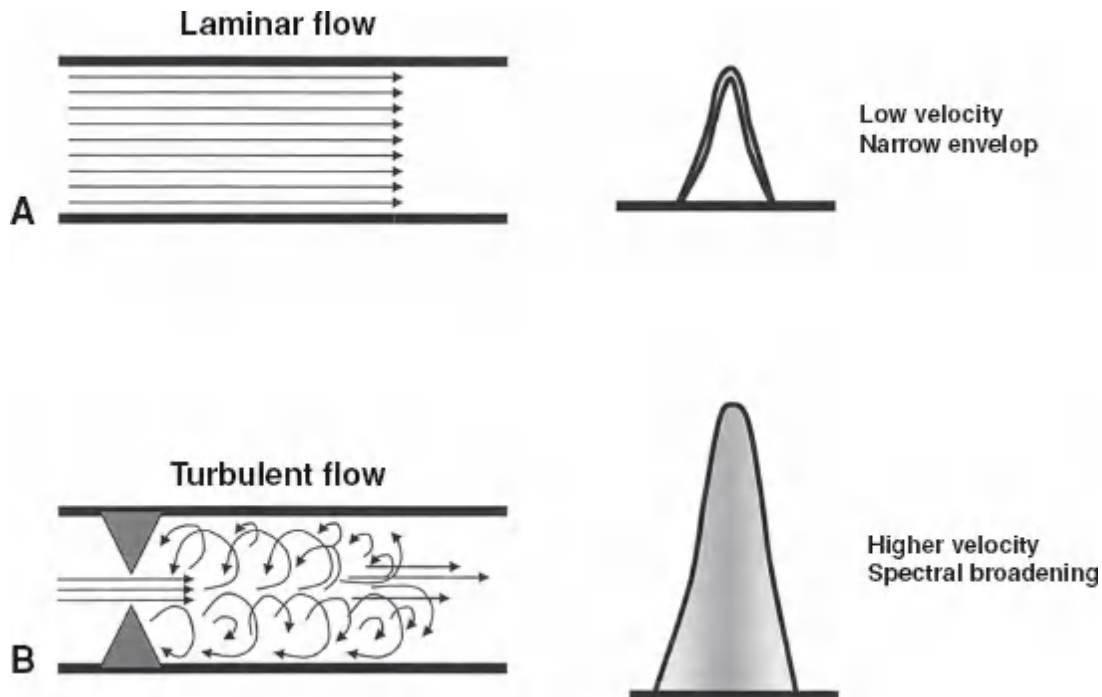


**FIGURE 2.44. A:** A laminar flow profile occurs when most of the red blood cells are traveling in approximately the same direction at approximately the same velocity. In a pulsatile system, this results in a Doppler signal that has a narrow envelope, as shown on the right. This would be typical of 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.

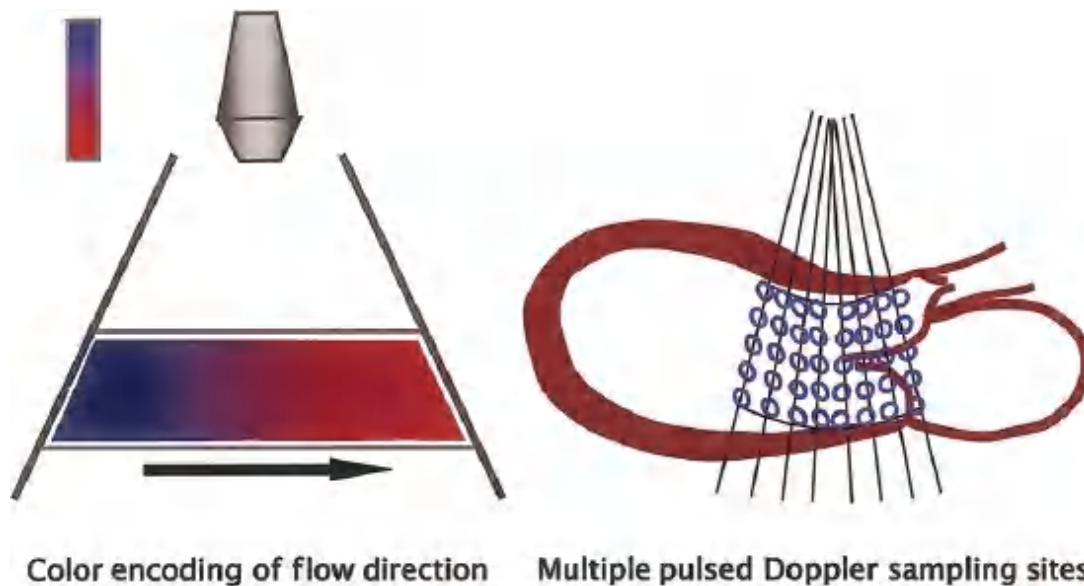


**FIGURE 2.45.** The basic principles of color flow imaging are illustrated. See text for details.

## 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 (ROA) ( $A$ ). 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 2.5**

**FACTORS THAT AFFECT COLOR DOPPLER JET AREA**

|             | Factor                                                                                                                         | Impact on Jet Area                              | Reason                                                                                                                                                                                                       |
|-------------|--------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Physiologic | Increase in blood pressure<br>Increased jet velocity<br>Eccentric jet (wall hugging)<br>Slit-like orifice<br>Multiple orifices | Larger<br>Larger<br>Smaller<br>Larger<br>Larger | May result in increased jet velocity<br>Higher velocity leads to higher momentum<br>Wall constraint causes more rapid loss of momentum<br>2D imaging aligned to the long axis of the slit                    |
| Technical   | Higher pulse repetition frequency (PRF)<br>Decrease in Nyquist limit<br>Increase in color Doppler gain<br>Transducer frequency | Smaller<br>Larger<br>Larger<br>Variable         | Results in encoding of lower velocities<br>Color encoding increases with increasing gain<br>Higher frequency encodes lower velocities (larger jet) but also suffers from far-field attenuation (smaller jet) |



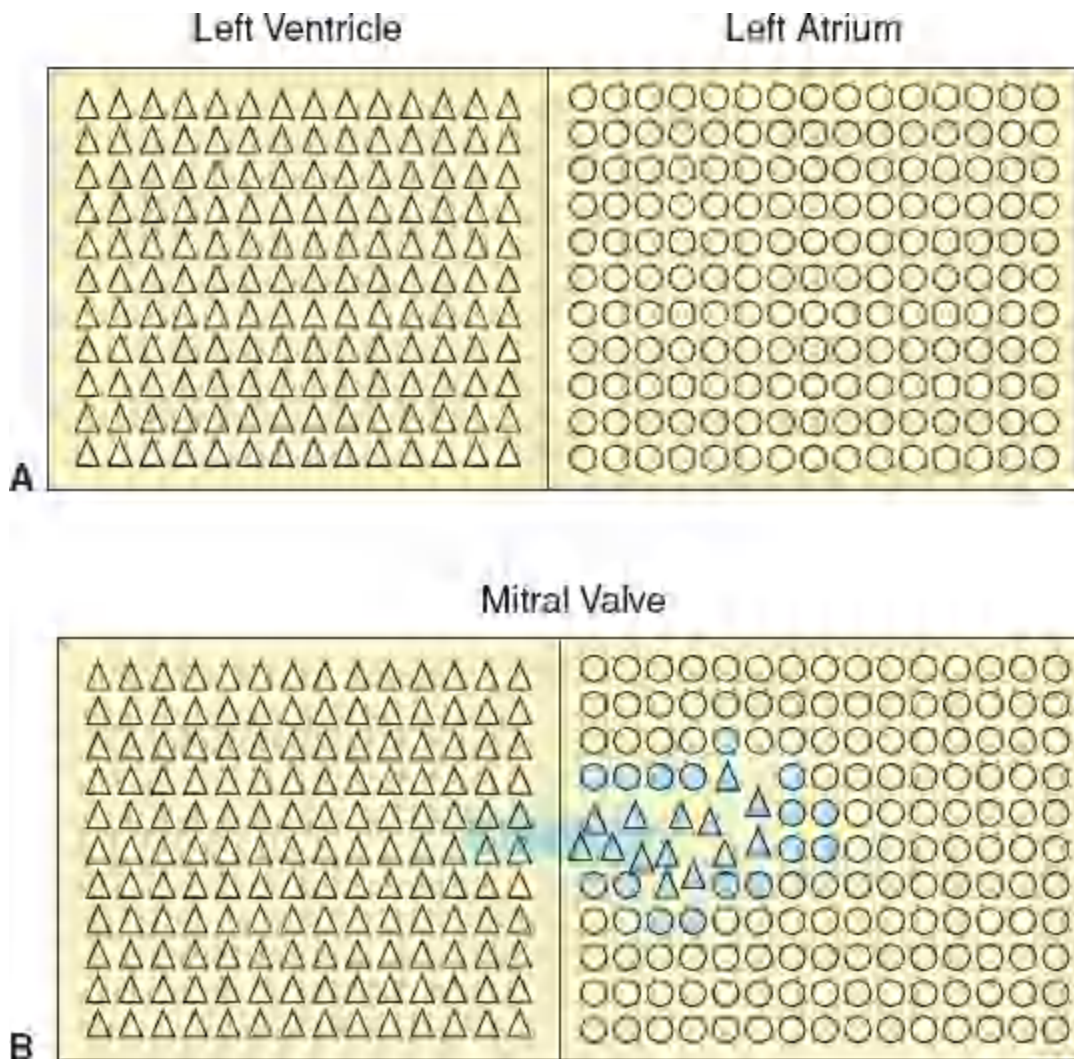


**FIGURE 2.46. A:** This is a schematic depiction of mitral regurgitation, with the triangles representing blood within the left ventricle, and the circles indicating left atrial blood. **B:** Mitral regurgitation is demonstrated by some of the triangles moving through the orifice into the left atrium. 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.

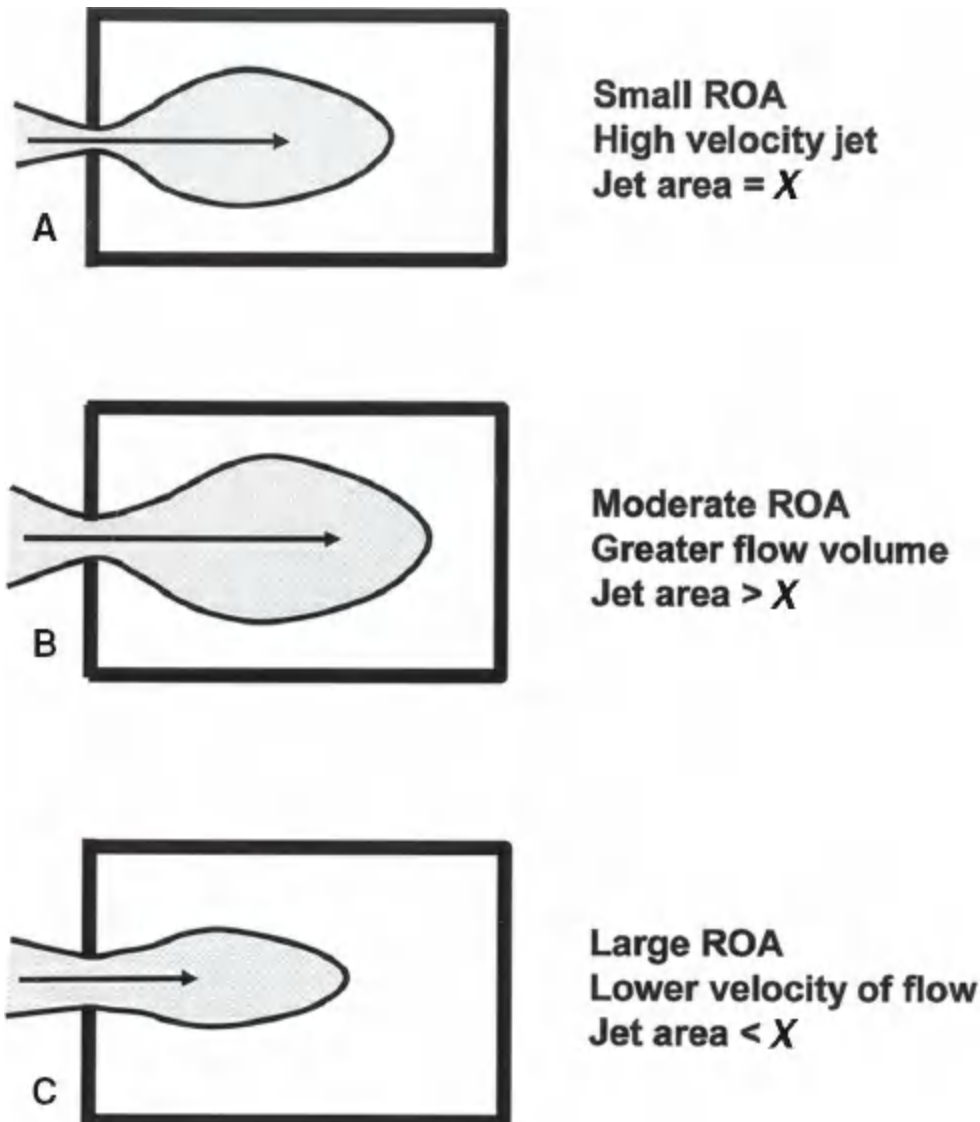


**FIGURE 2.47.** This diagram demonstrates how turbulent flow through a regurgitant orifice relates to color Doppler signal. See text for details. ROA, regurgitant orifice area.

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.

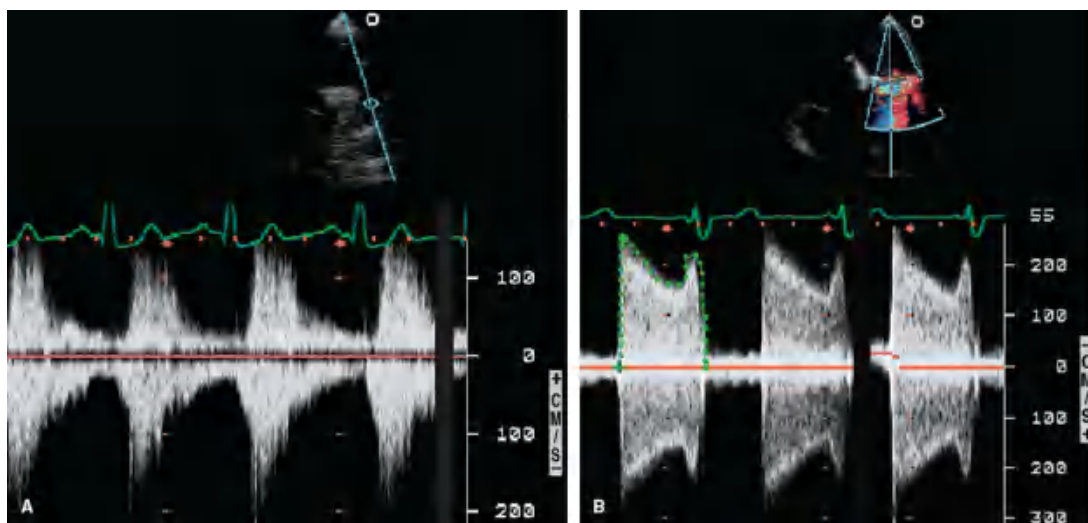


**FIGURE 2.48.** Two examples of mirror image artifact are demonstrated. **A:** Descending aortic flow appears to occur both above and below the baseline. **B:** A

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.

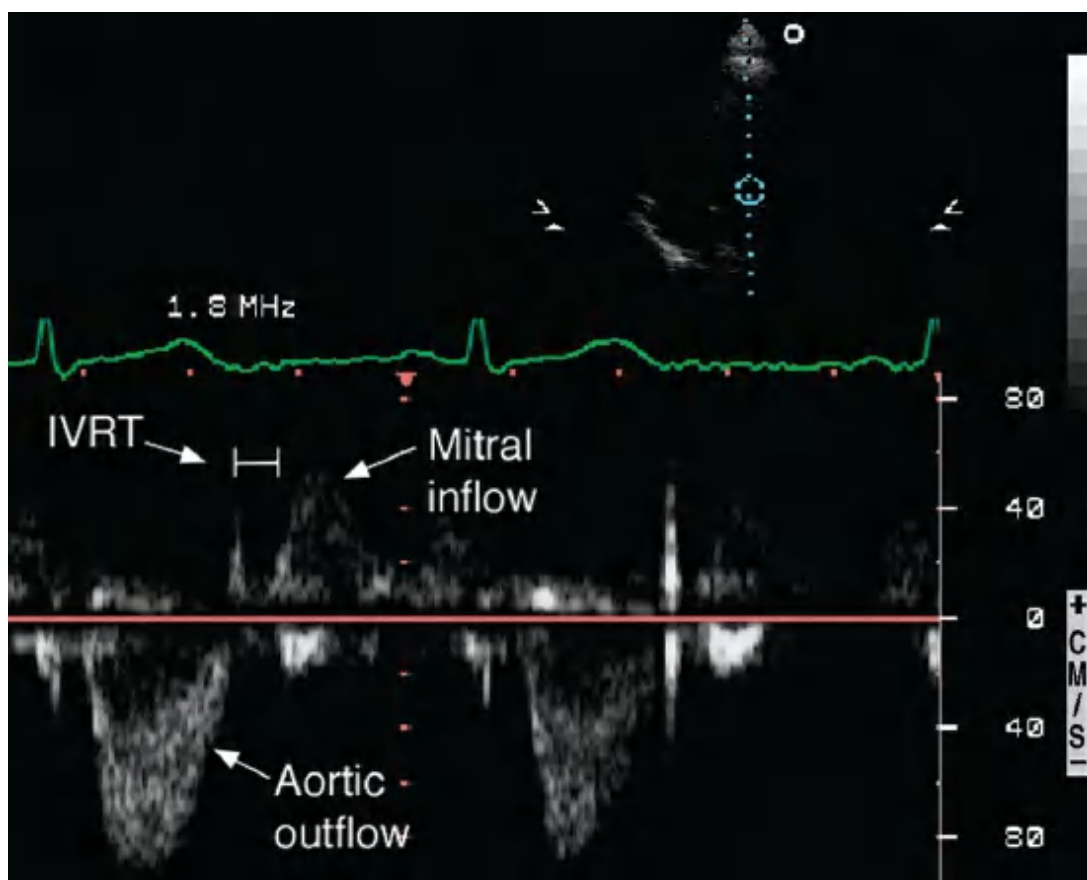


**FIGURE 2.49.** Beam width artifacts in Doppler imaging can be clinically useful. In this case, the thickness of the Doppler beam allows simultaneous recording of both aortic outflow and mitral inflow. This permits the isovolumic relaxation time to

be determined. IVRT, isovolumic relaxation time.

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.

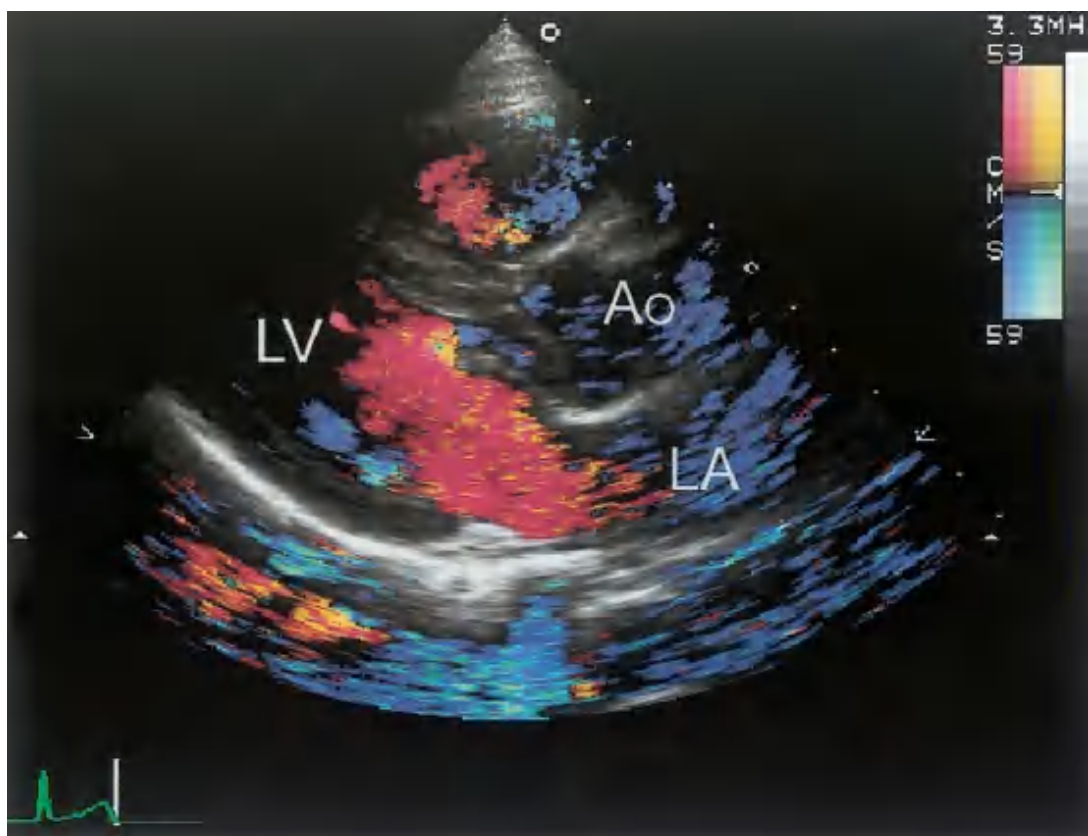


**FIGURE 2.50.** Ghosting occurs when brief displays of color are painted over

regions of tissue, as shown in the illustration. See text for details.

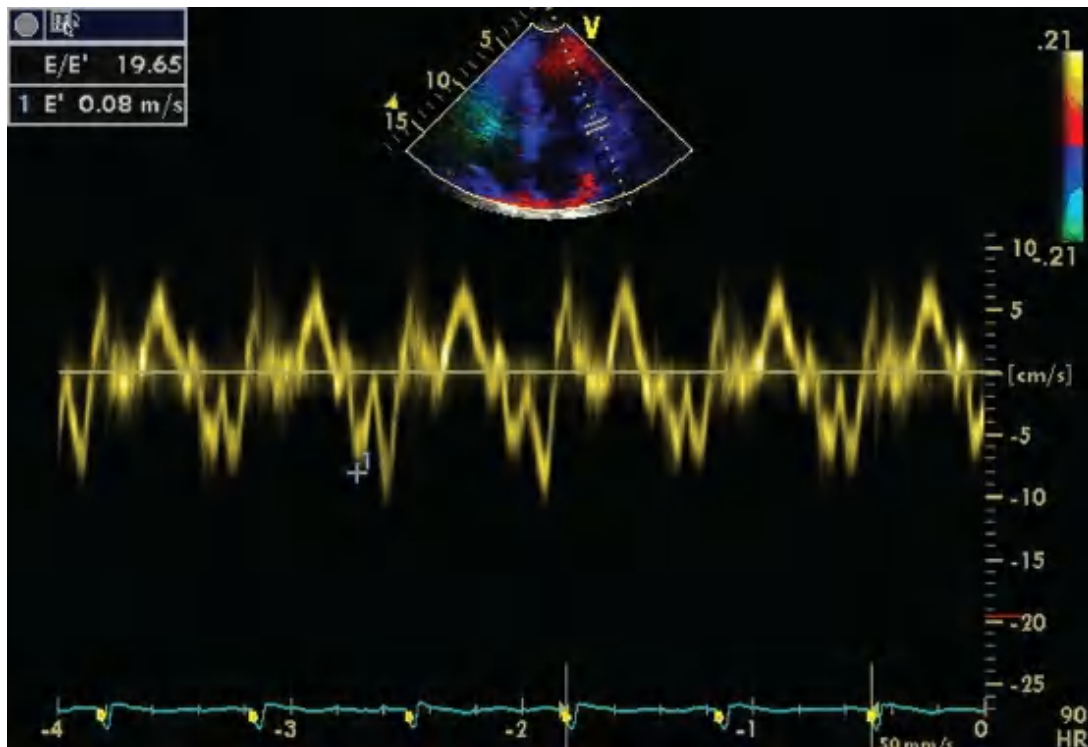


**FIGURE 2.51.** An example of tissue Doppler imaging is provided. The sample volume is positioned within the mitral annulus and velocity of the annulus is recorded throughout the cardiac cycle.

## 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 ( $\text{W}/\text{m}^2$ ) or in milliwatts per centimeter squared ( $\text{mW}/\text{cm}^2$ ). 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  $1.0 \text{ cm}^2$ , then SA intensity would be  $2.0 \text{ mW}/\text{cm}^2$ . 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  $\mu\text{s}$  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<sup>2</sup>. Pulsed-Doppler imaging, however, may have an SP, temporal average as high as 1,900 mW/cm<sup>2</sup>, considerably greater than 100 mW/cm<sup>2</sup> 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.

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## 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.

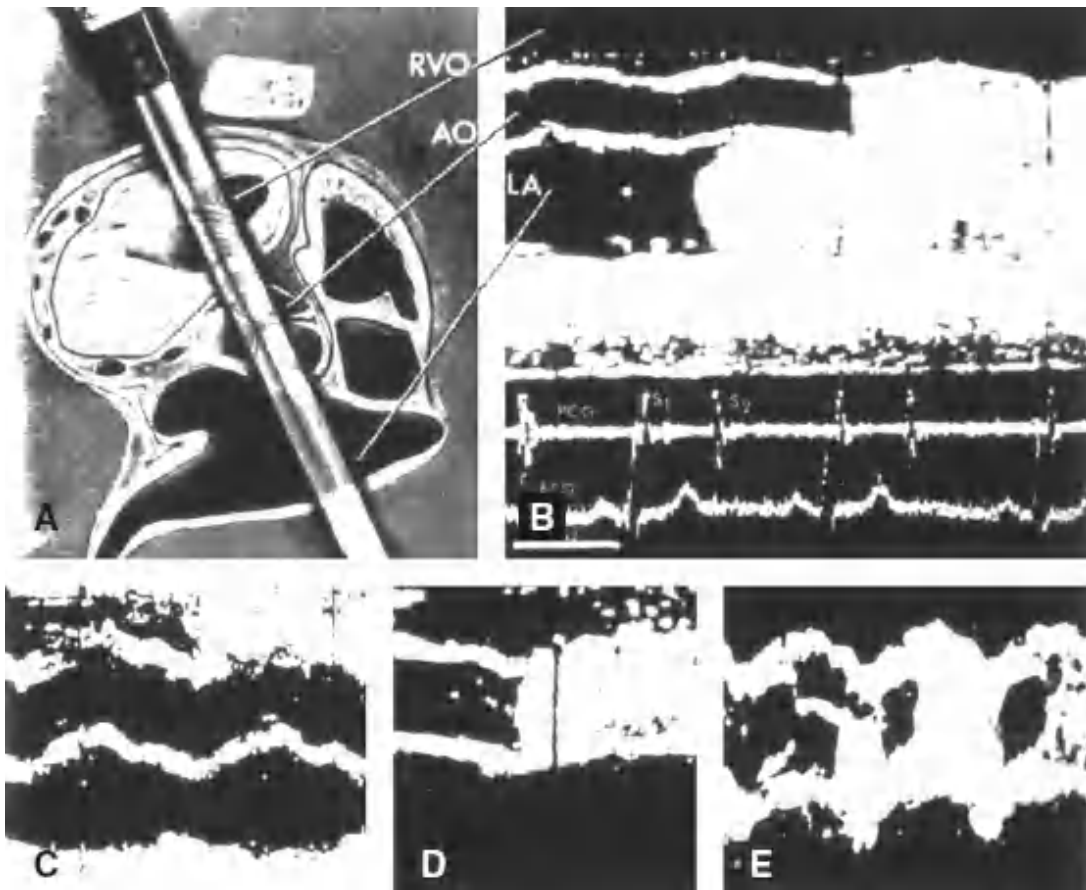


**FIGURE 3.1.** Early M-mode contrast echocardiograms recorded in the cardiac catheterization laboratory. **A:** The orientation of the M-mode ultrasound beam. **B:** Image was recorded after injection of contrast into the left atrium and shows subsequent appearance of contrast in the aorta. **C:** Contrast 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 supraventricular 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.

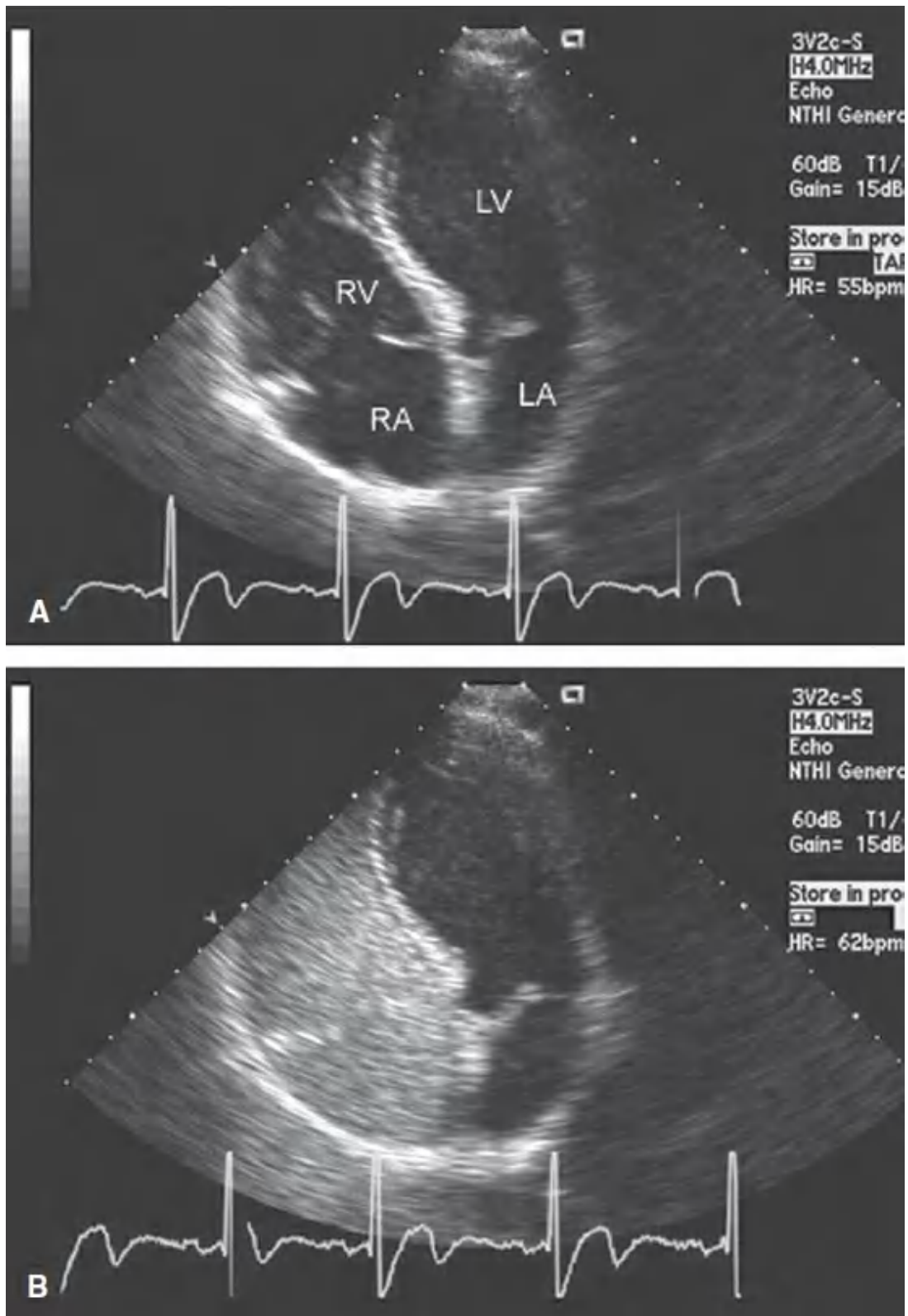
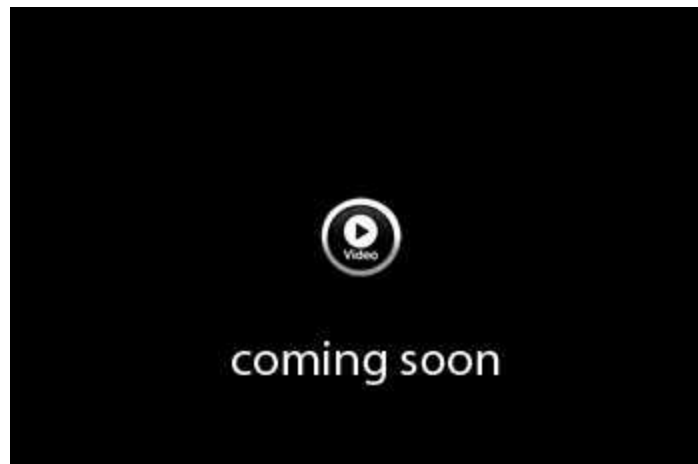


**FIGURE 3.3.** **A:** Apical four-chamber view recorded in a patient after injection of saline into a left upper extremity vein. **B:** After injection of intravenous contrast, there is uniform opacification of the right atrium and right ventricle with no



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  $\mu\text{m}$  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.

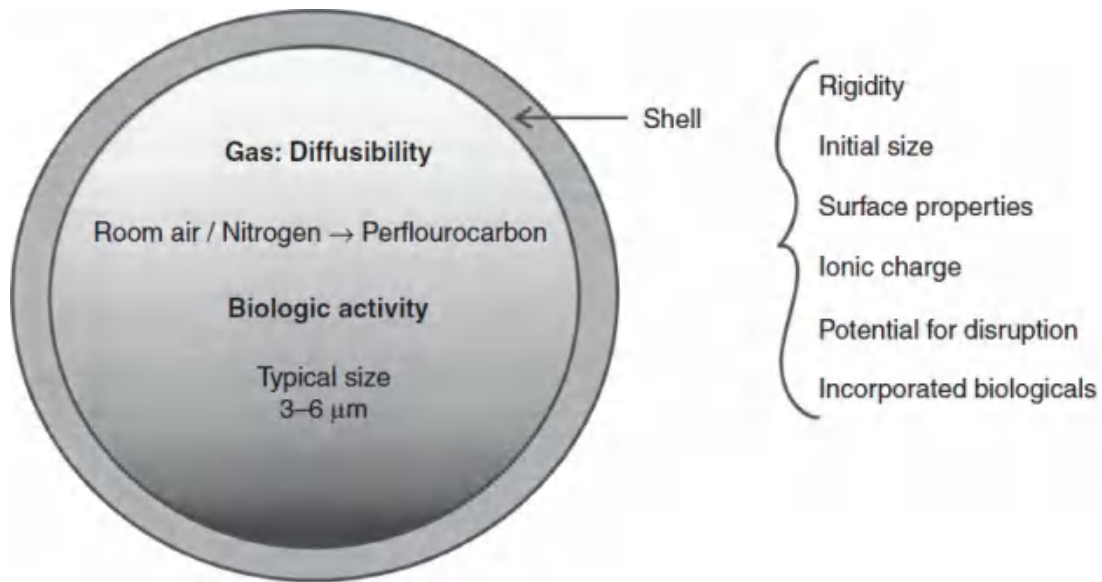


**FIGURE 3.4.** Schematic representation of a microbubble depicts its contents and various shell characteristics. See text for details.

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  $\geq 0.3$  MI), microbubbles are not pure reflectors but begin to resonate. A resonating bubble will reflect ultrasound not only at the fundamental insonating frequency ( $f_i$ ) 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.

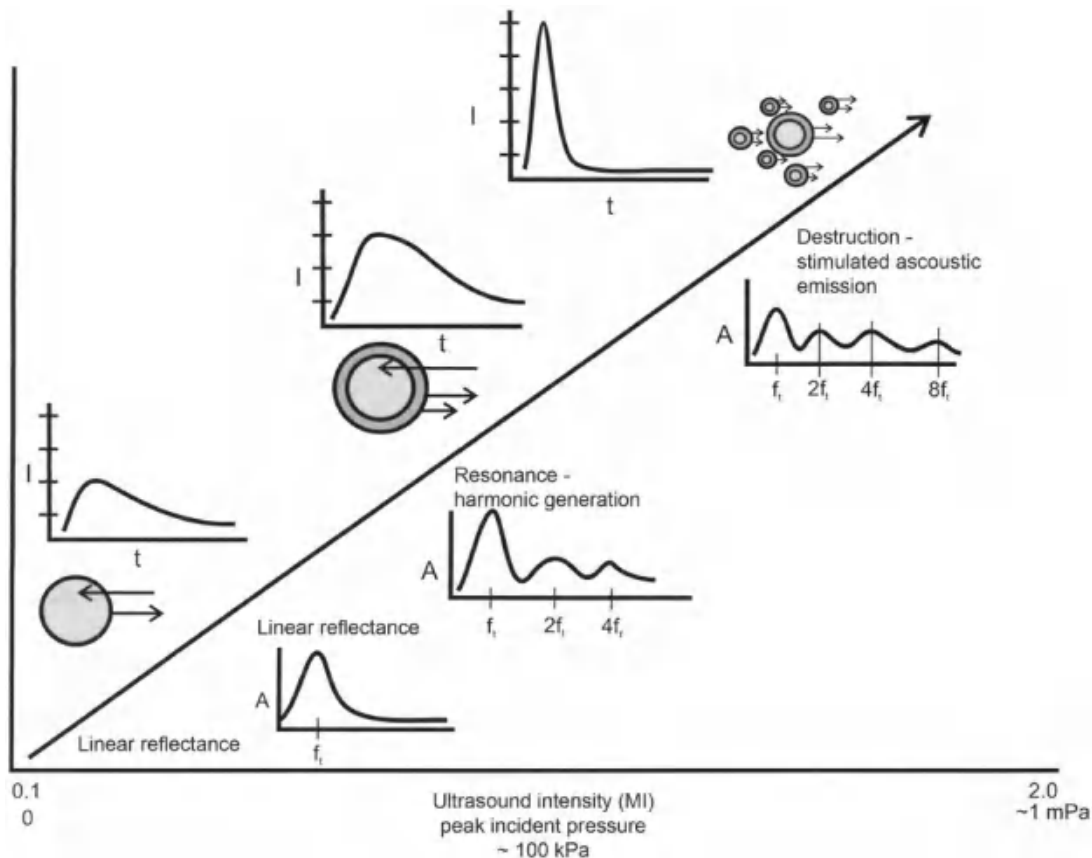


**FIGURE 3.5.** Schematic representation of various microbubble responses to increasing ultrasound intensity. Above the diagonal line, depicting increasing intensity, is a graphic representation of reflected image intensity at each level of ultrasound intensity and below the line a stylized depiction 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).

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

### 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/ $\sqrt{f_t}$ , where  $f_t$  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.

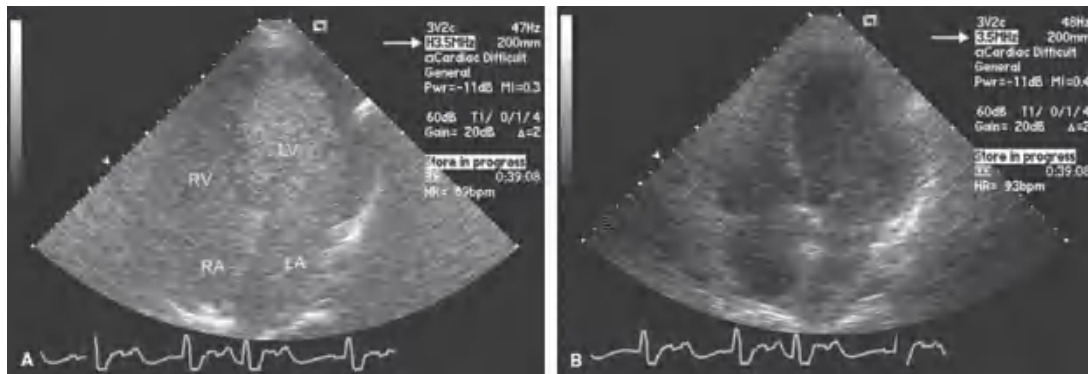
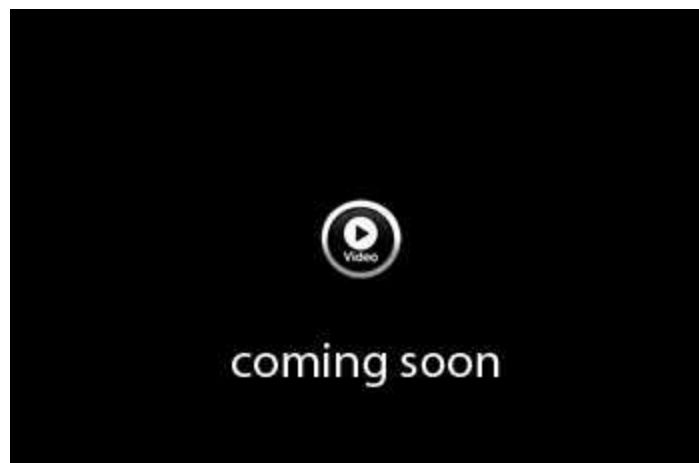


**FIGURE 3.6.** Four-chamber view recorded in a patient during harmonic (A) and fundamental (B) imaging often injection of a commercial contrast agent for left ventricular opacification. Note that with harmonic imaging, there is smooth opacification of the cavity and detection of contrast in all four 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**

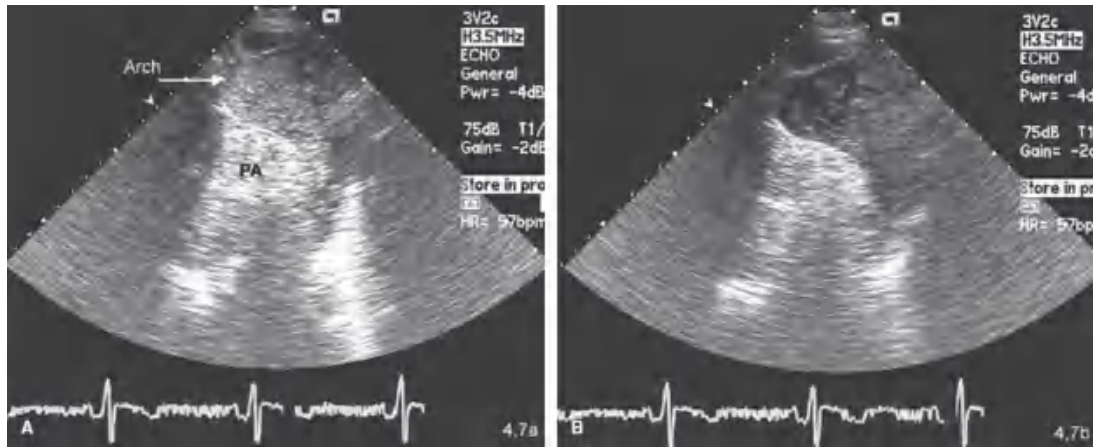
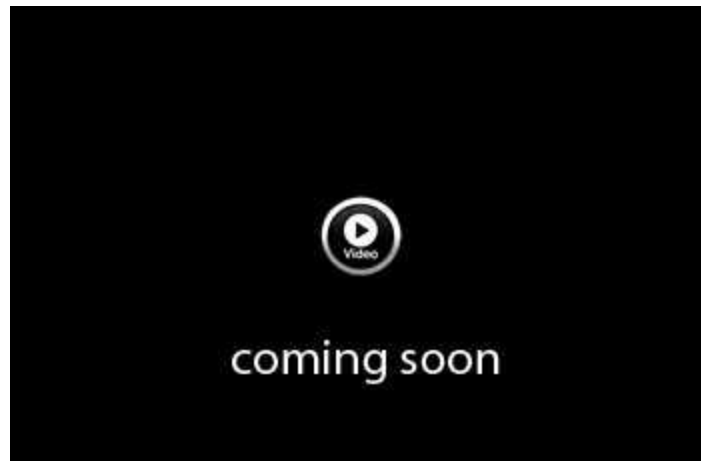


**FIGURE 3.7.** Suprasternal view of a normal aorta after intravenous injection of ultrasound contrast. The electrocardiogram is provided for timing. **A:** A systolic frame in which contrast is clearly identified in the arch of the aorta. **B:** The diastolic portion of the same cardiac cycle, in which 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. 📺



**Video 3-7**

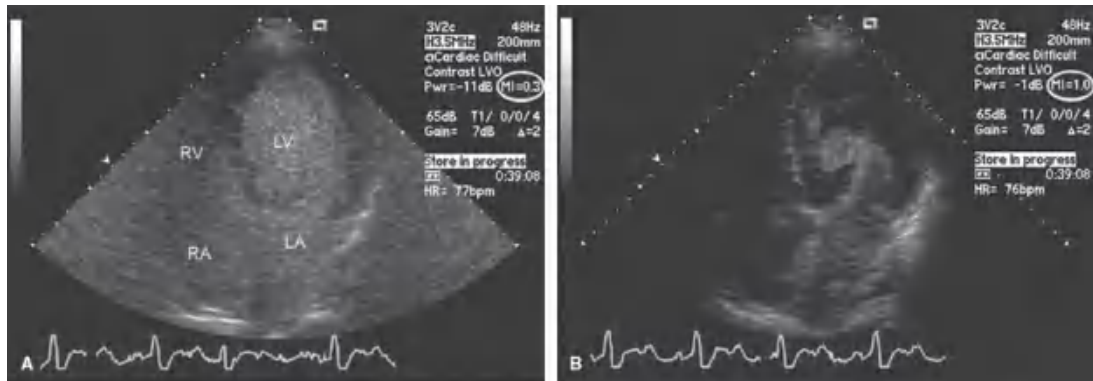
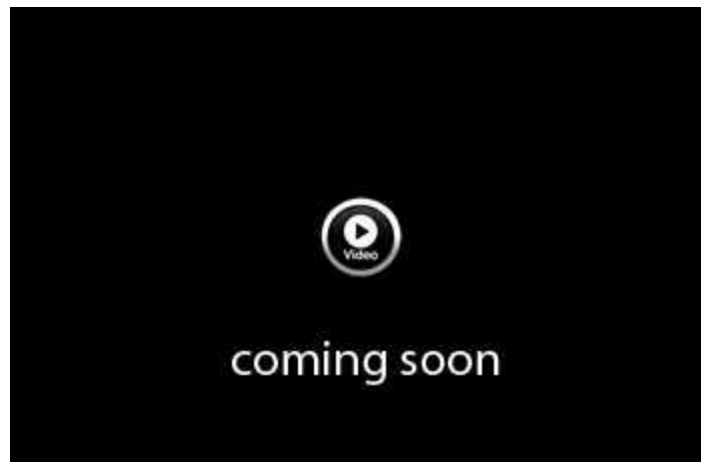
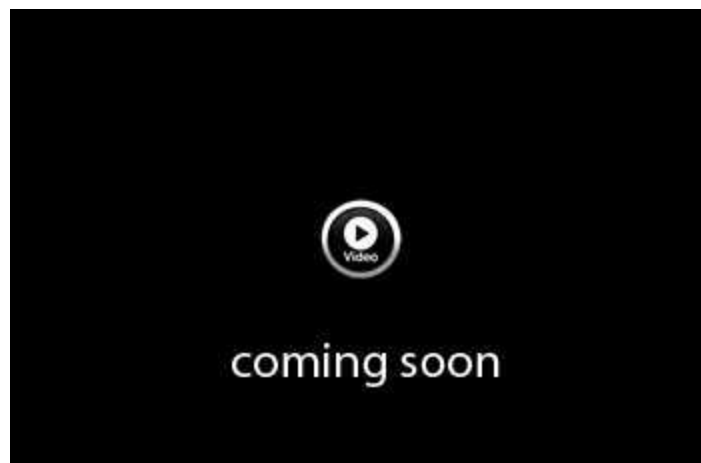


**FIGURE 3.8.** Apical four-chamber view recorded in a patient demonstrating the impact of mechanical index on contrast appearance. **A:** Image was recorded with a mechanical index of 0.3 and reveals smooth opacification of all four cardiac chambers. **B:** Image was recorded 10 seconds later with a 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**



### Video 3-8b

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.

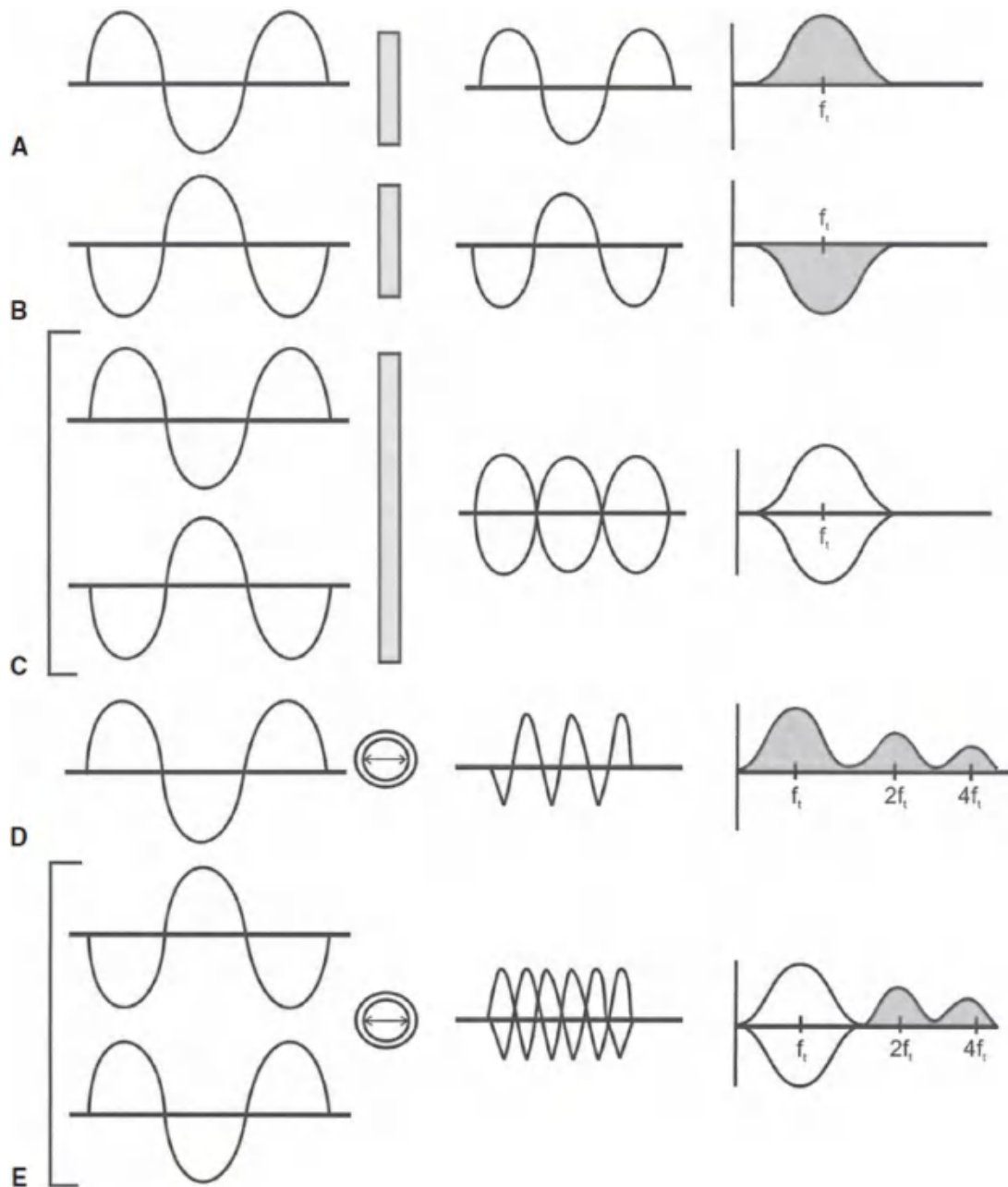


**FIGURE 3.9.** A simplified version of phase analysis is presented in which only sequential pulses 180 degrees out of phase are diagrammed. In practice, both phase and amplitude may be altered in the sequential pulses. **A:** A transmitted wave interacts with a linear reflector (*solid bar*). The received 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.

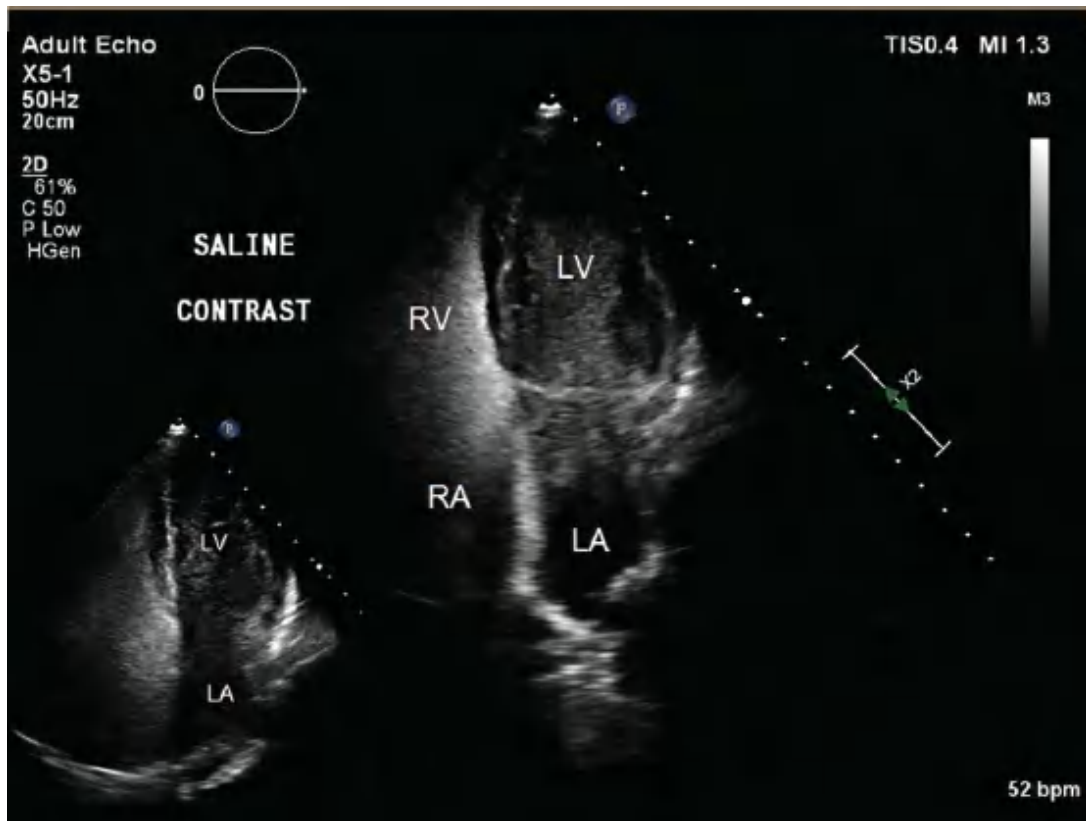
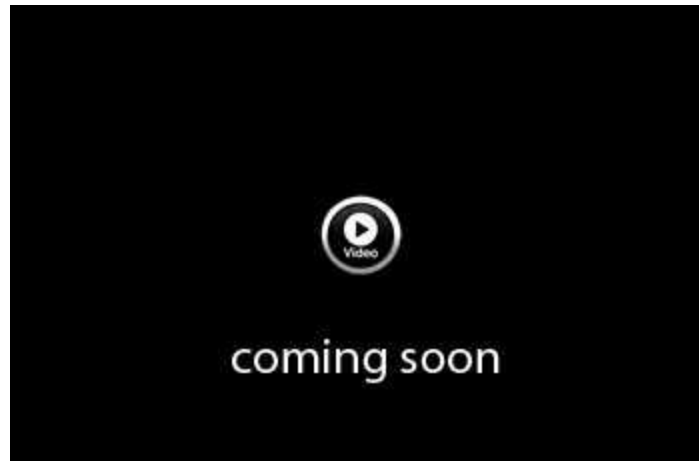


**FIGURE 3.10.** Apical four-chamber view recorded in a patient with a secundum atrial septal defect. The central image was recorded after injection of agitated saline and demonstrates a significant right-to-left shunt which was phasic with the respiratory cycle. Note the contrast in the left ventricle 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.







**Video 3-10**

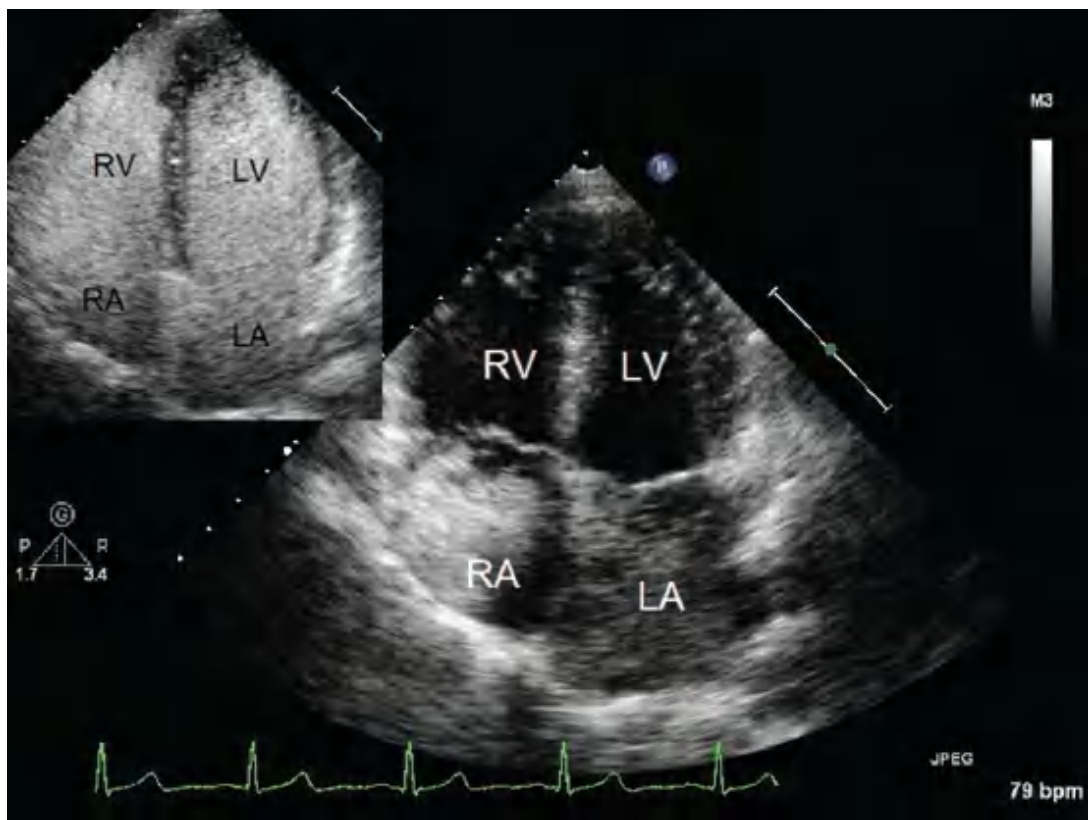
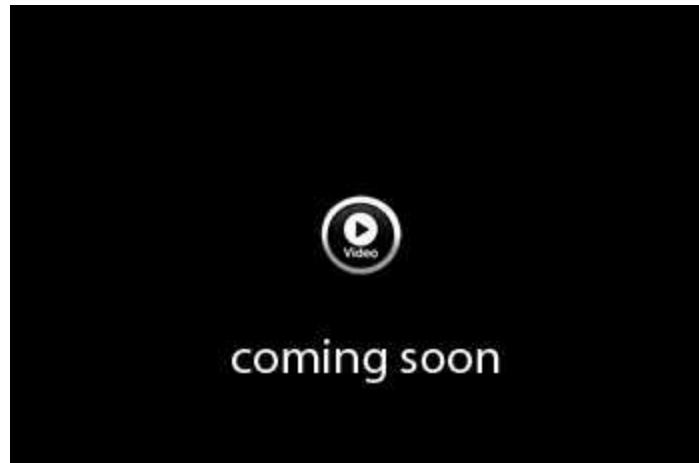


**FIGURE 3.11.** Apical four-chamber view recorded after saline contrast injection in a patient with a sinus venosus atrial septal defect. The central image was recorded immediately after appearance of contrast in the right atrium and shows concurrent early appearance in the left atrium, prior to its 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. 



### **Video 3-11**

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.

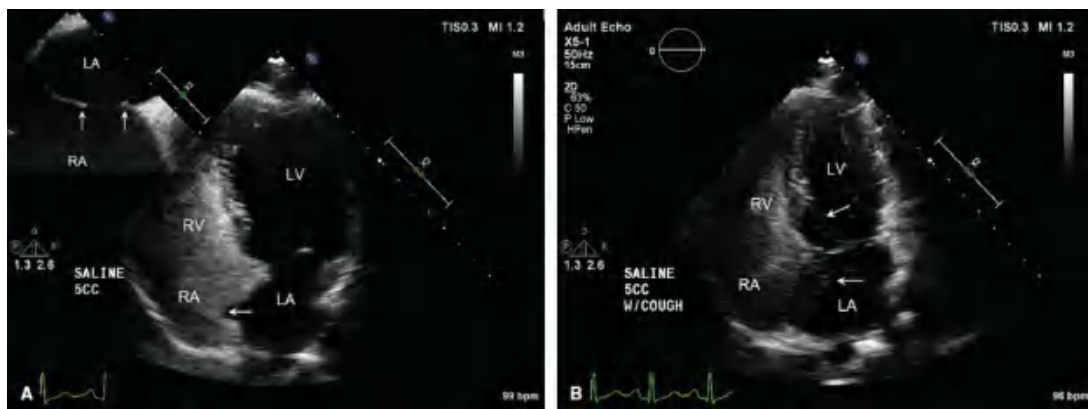
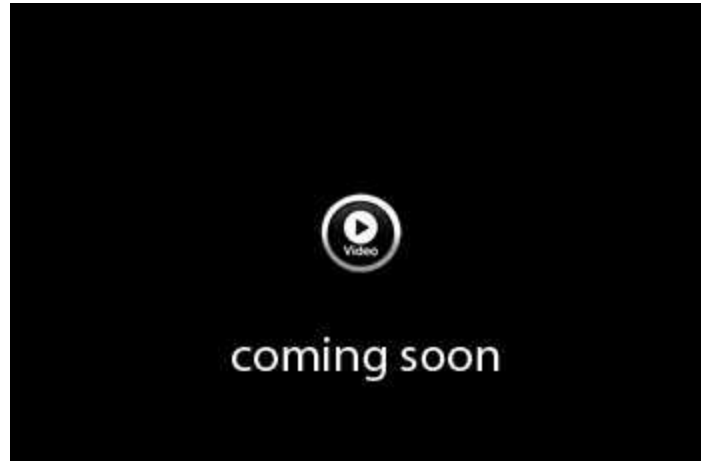


**FIGURE 3.12.** Saline contrast echocardiogram recorded in a patient with a secundum atrial septal defect. **A:** Four-chamber view with saline contrast recorded during quiet respiration. Note the opacification of the right atrium and right ventricle and the negative contrast jet from the left atrium 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.

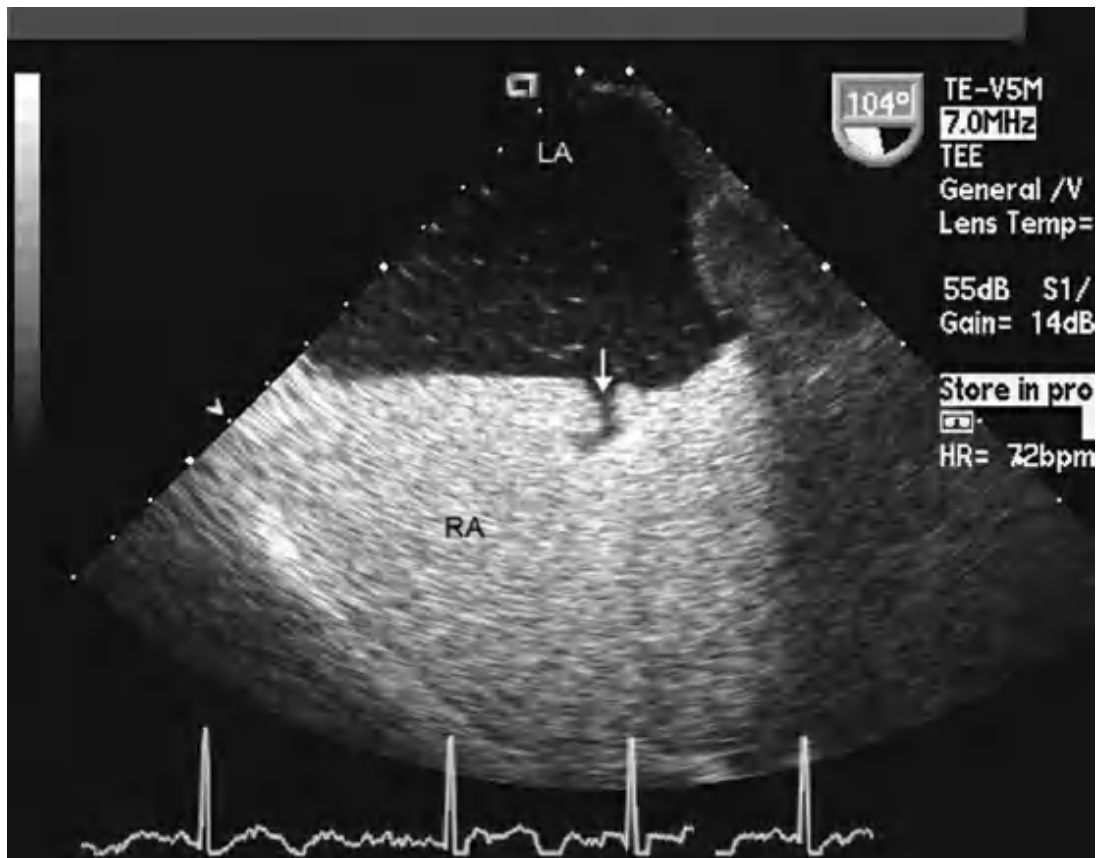
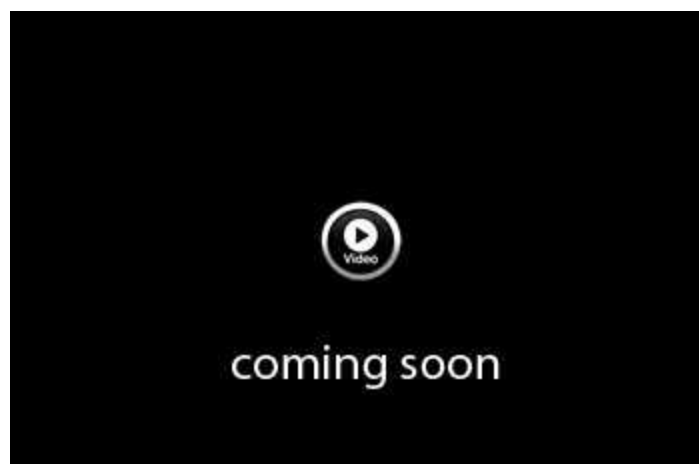


**FIGURE 3.13.** Transesophageal echocardiogram recorded in a longitudinal view of the atrial septum. Agitated saline has been injected into an upper extremity vein and has completely filled the right atrium. Note a small number of individual contrast targets in the left atrium consistent with a limited 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. 🇧🇷



### 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).

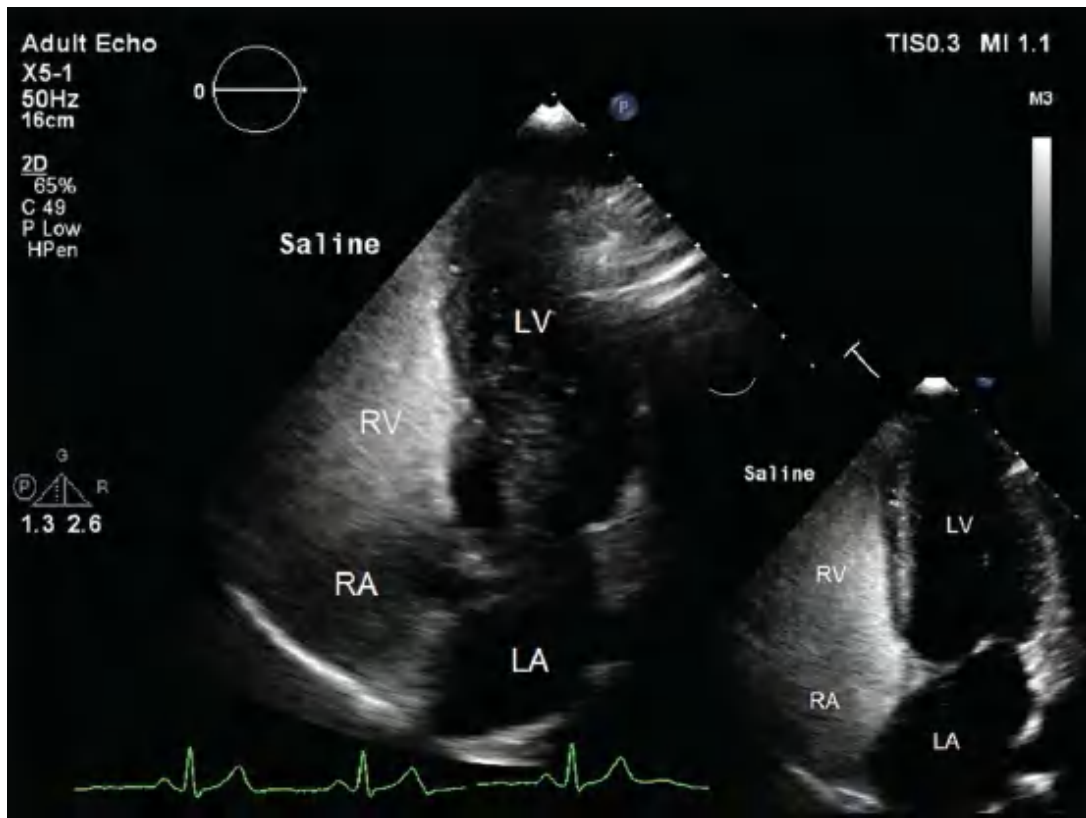
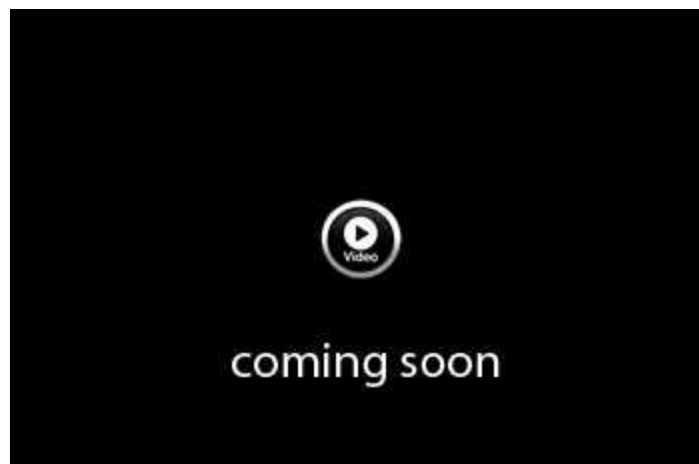


**FIGURE 3.14.** Apical four-chamber view recorded in a young patient with a neurologic event. Saline contrast has been injected to evaluate the possibility of a patent foramen ovale. The presented central image was recorded approximately five cycles after appearance in the right atrium and shows definite 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. 📺





### Video 3-14

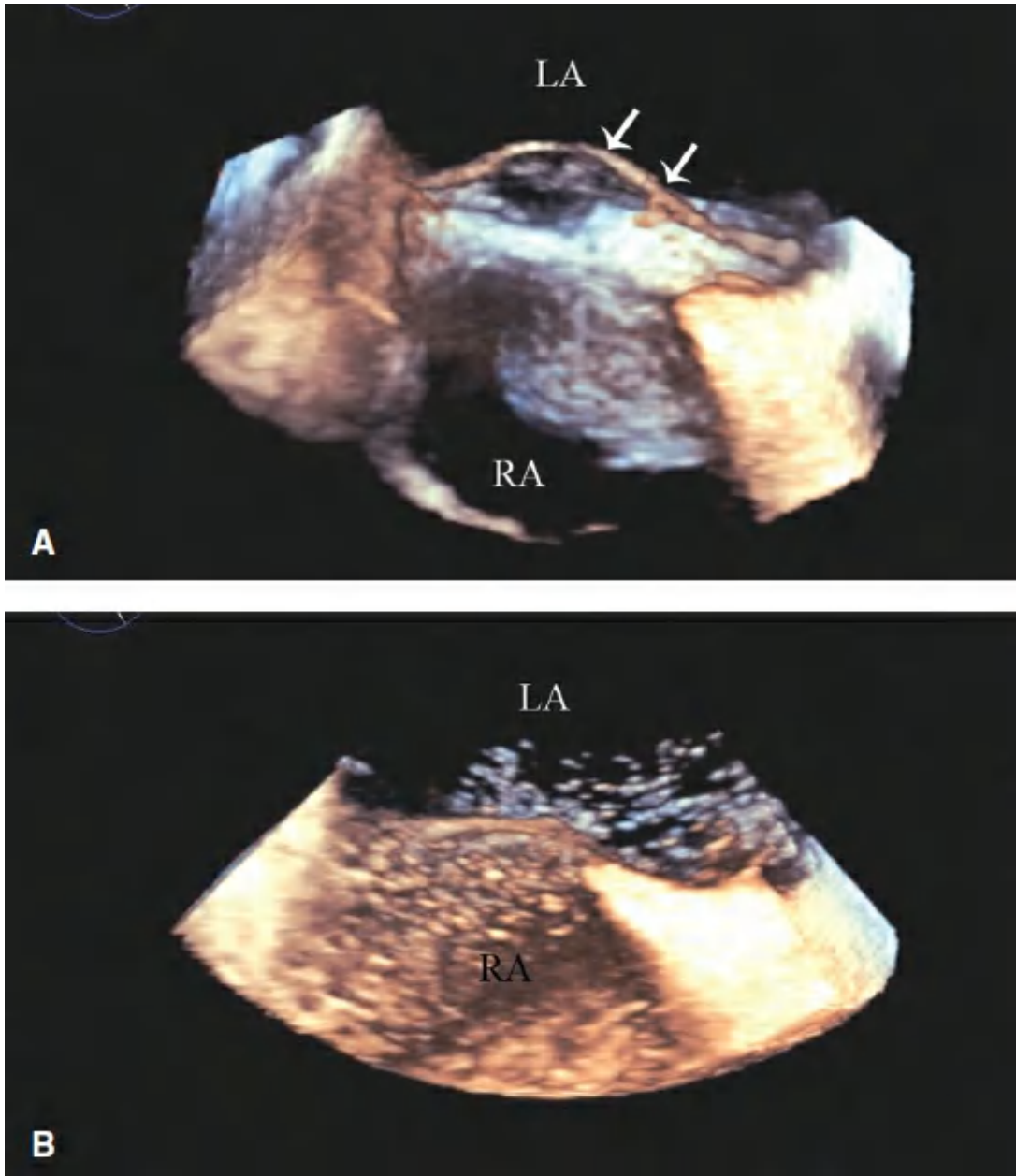
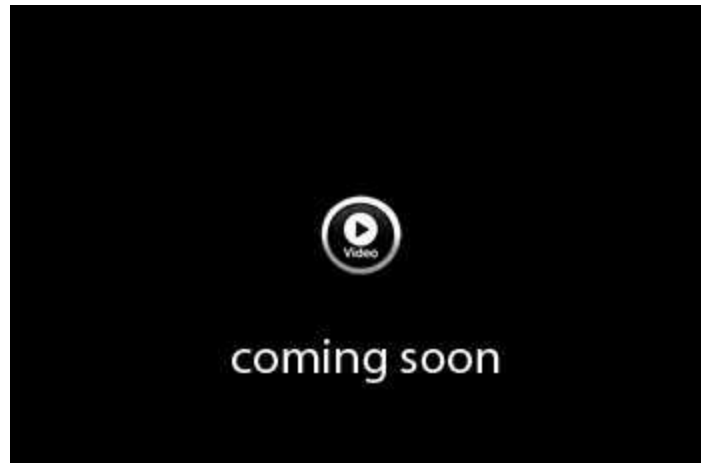
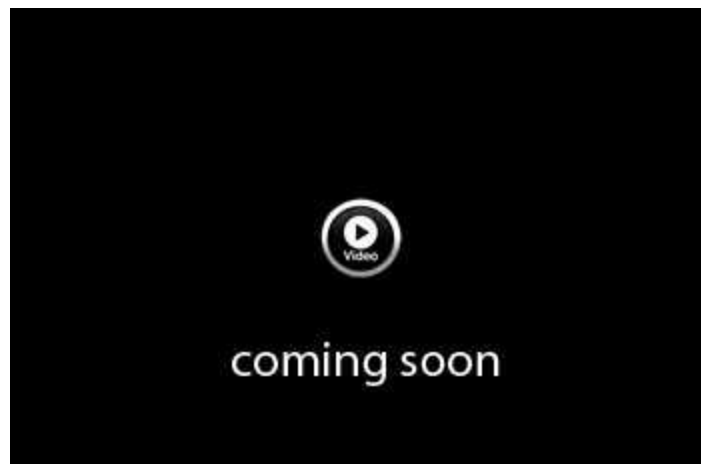


**FIGURE 3.15.** Real-time three-dimensional transesophageal echocardiogram of the atrial septum in a patient with a patent foramen ovale and a right-to-left shunt on saline contrast injection (**B**). **A**: Note the mobility of the foraminal tissue (*arrows*) in the real-time image. 





**Video 3-15a**



**Video 3-15b**

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 with azygos 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 latter 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.

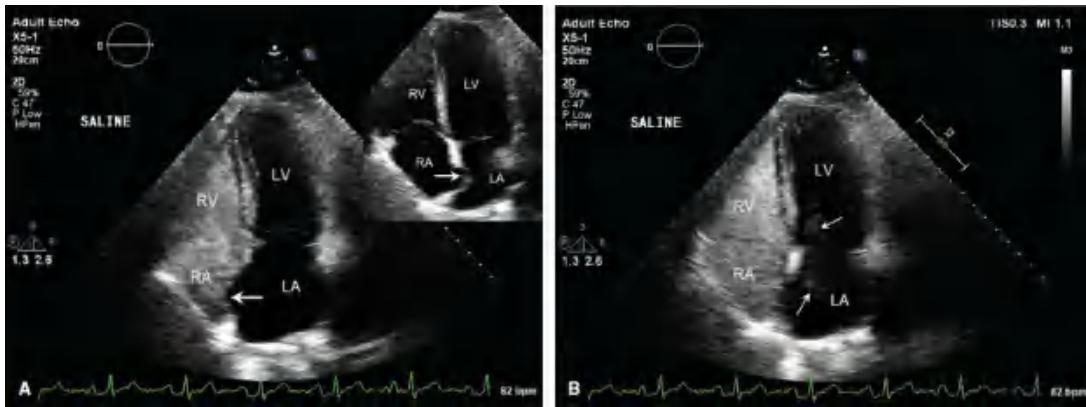
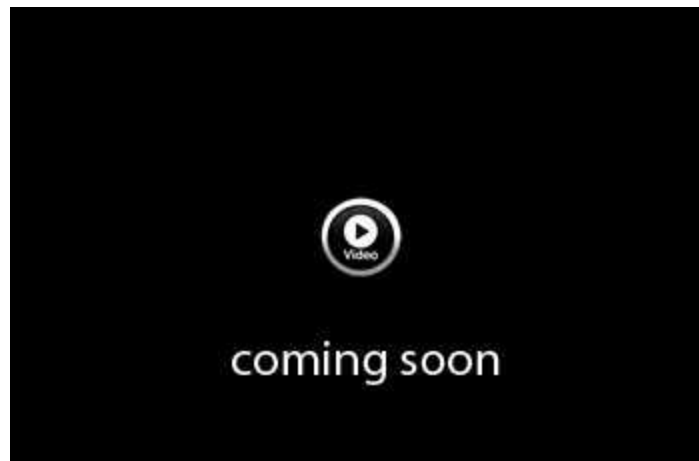


**FIGURE 3.16.** Apical four-chamber view recorded after intravenous injection of agitated saline in a patient with an atrial septal aneurysm. **A:** In the central image, note the complete opacification of the right atrium and right ventricle and the bulging of the atrial septal aneurysm (*arrow*) into 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.



**Video 3-16**

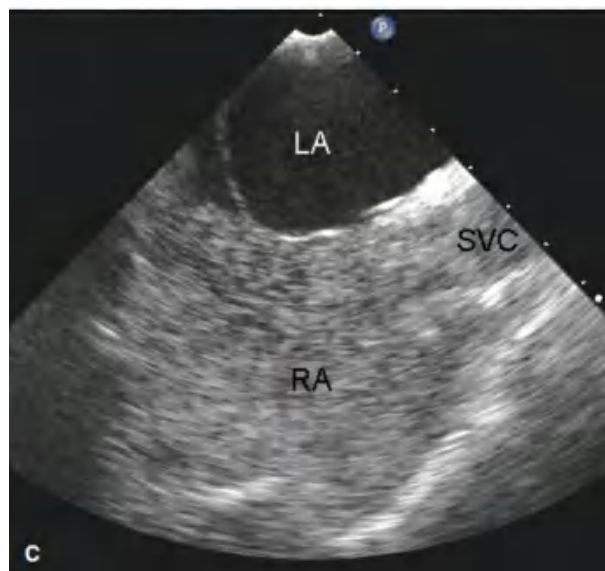
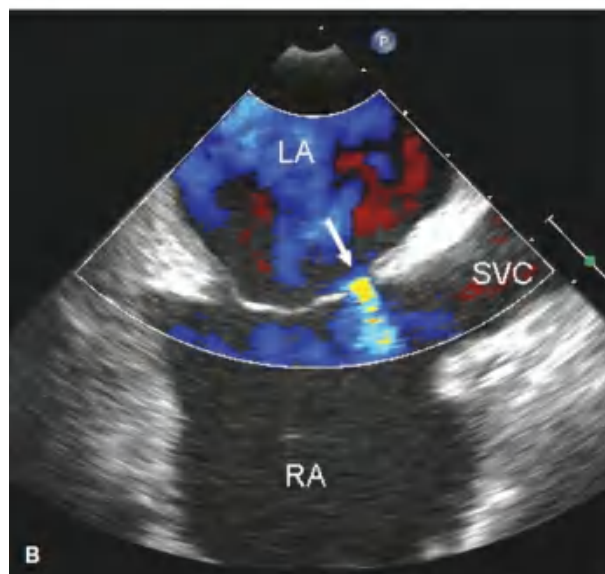
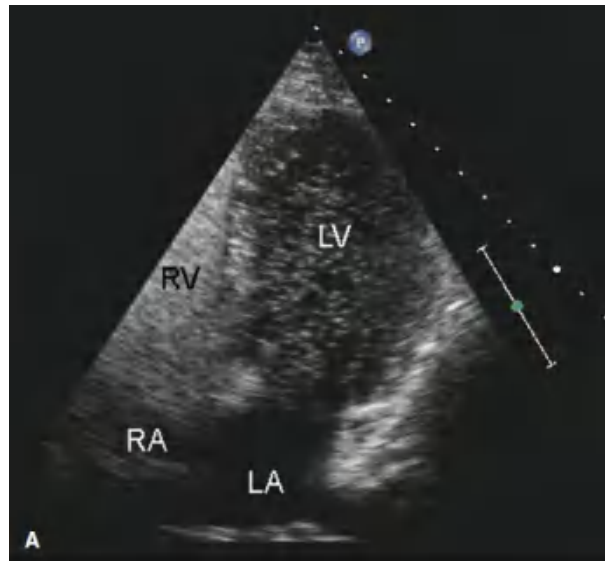
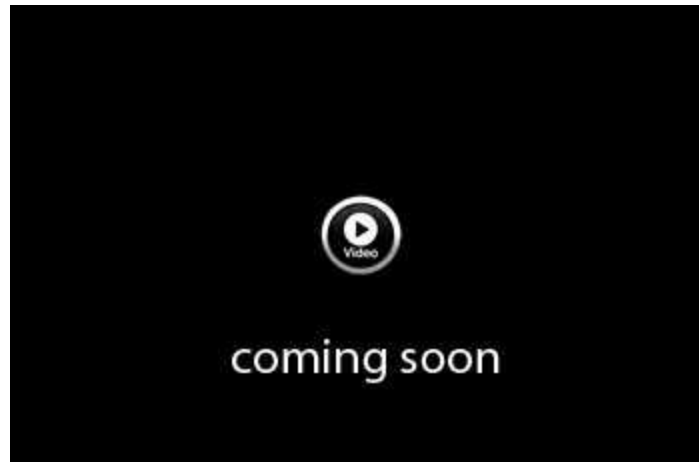
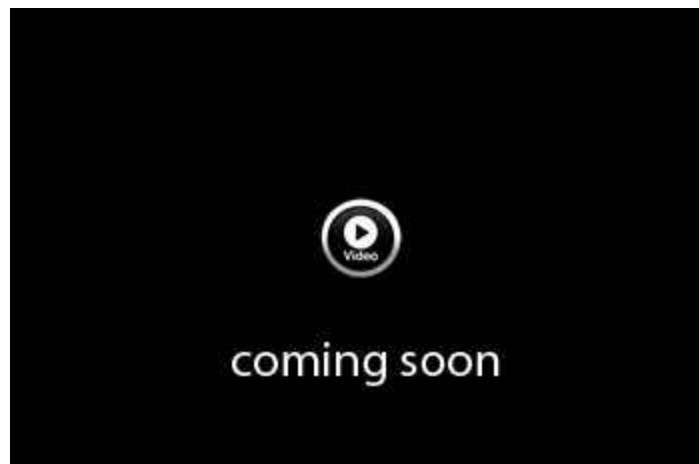


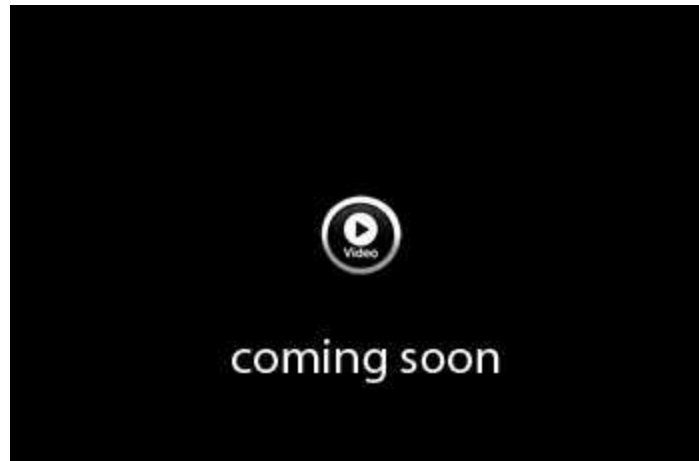
**FIGURE 3.17.** Transthoracic and transesophageal echocardiogram recorded in the same patient on two separate days. **A:** The upper panel was recorded under basal, nonfasting conditions and reveals a definite right-to-left shunt. **B, C:** The lower panels (transesophageal study) were recorded after 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**



**Video 3-17b**



**Video 3-17c**

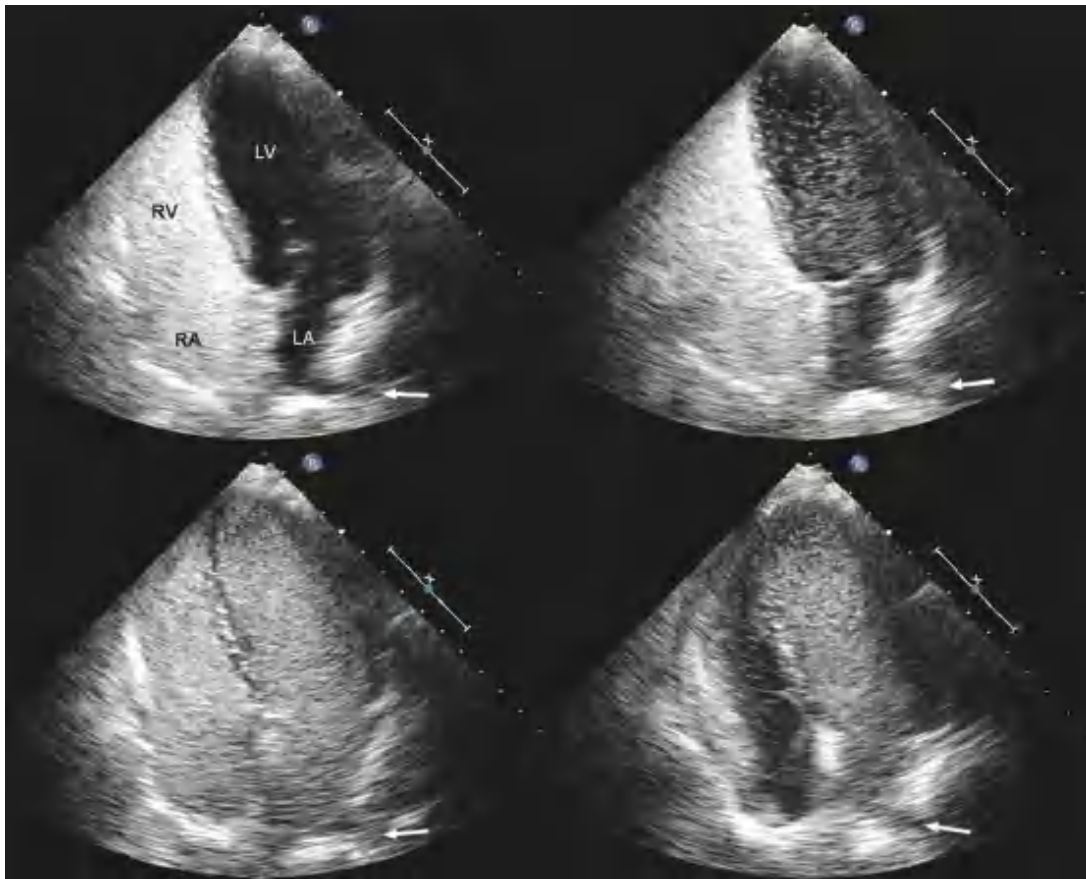
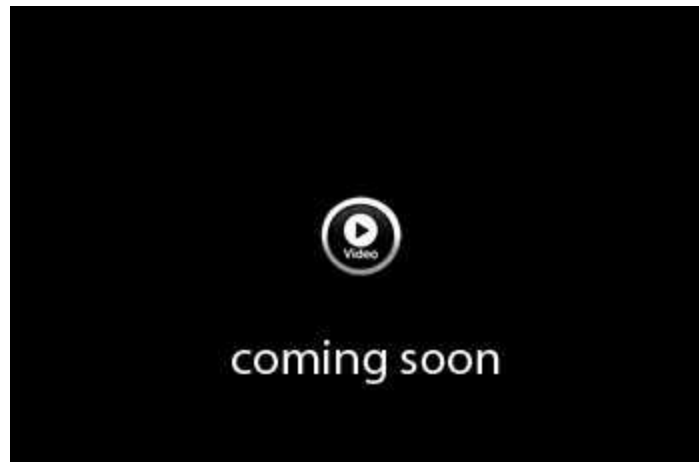


**FIGURE 3.18.** Apical four-chamber view recorded over a prolonged imaging period in a patient with a large pulmonary venous malformation after injection of saline contrast into an upper extremity vein. The upper left panel was recorded immediately after filling of the right atrium and right ventricle. 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). 📺



**Video 3-18**

## 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 caval* 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 caval* blood 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.



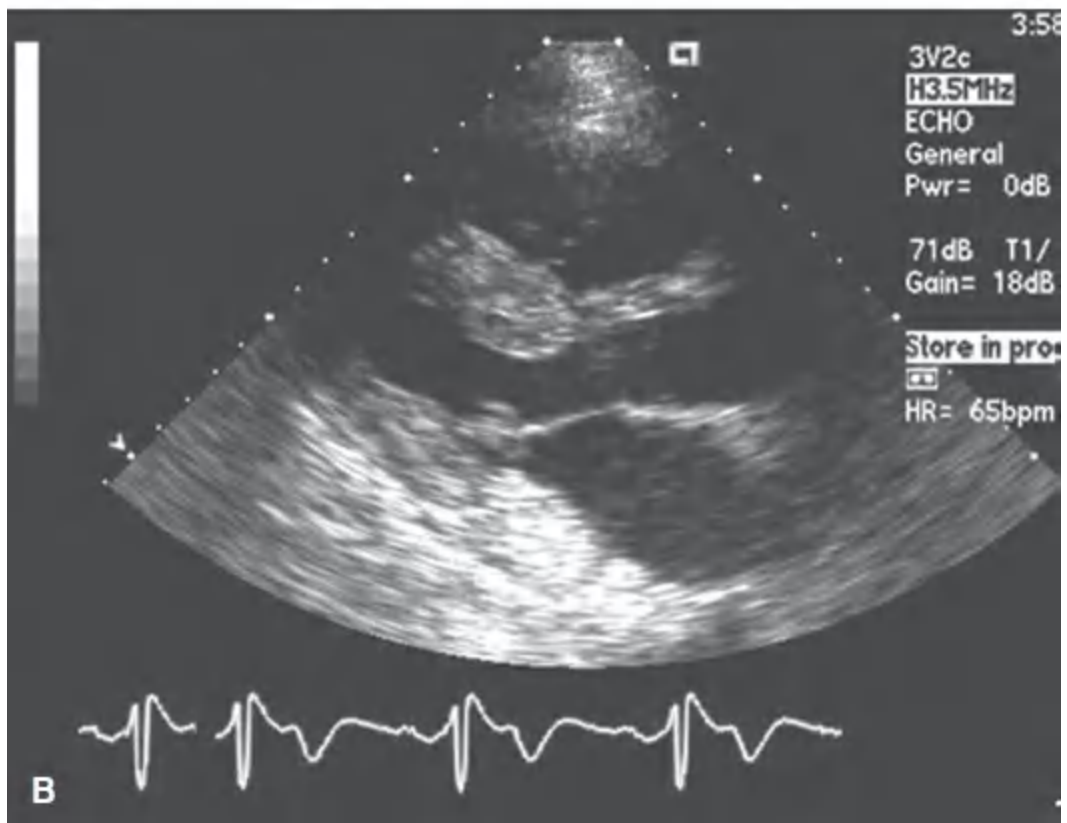
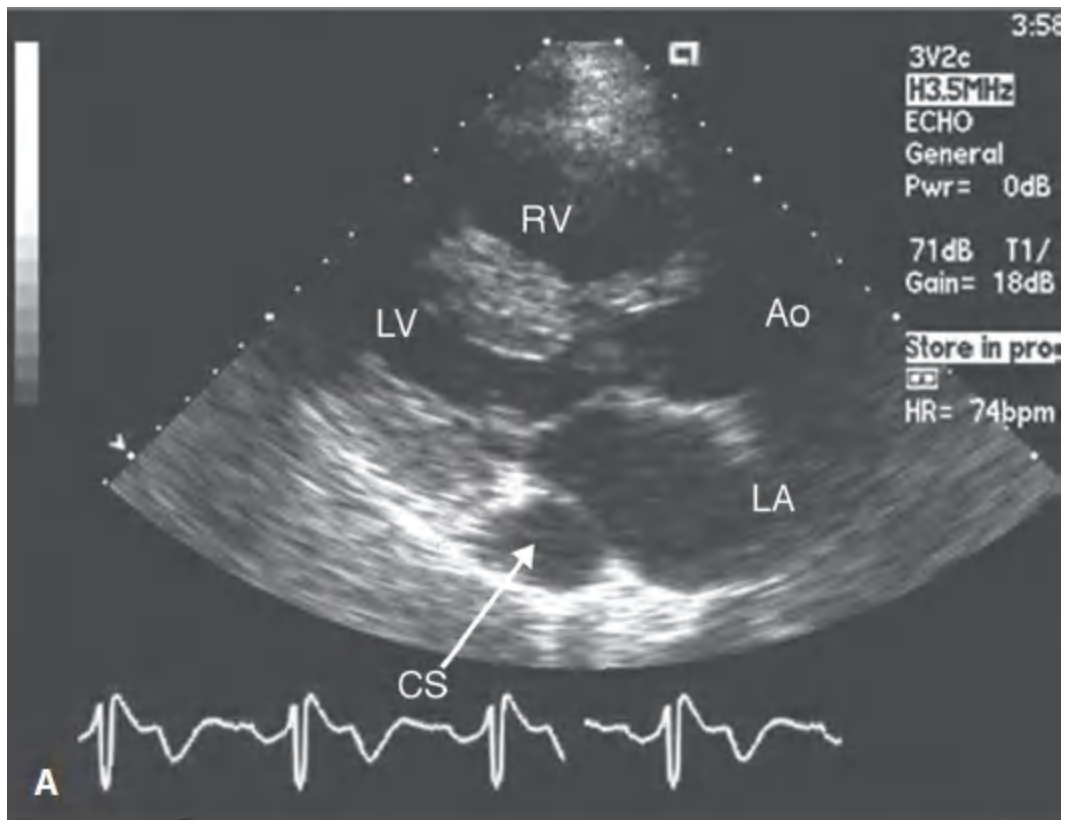
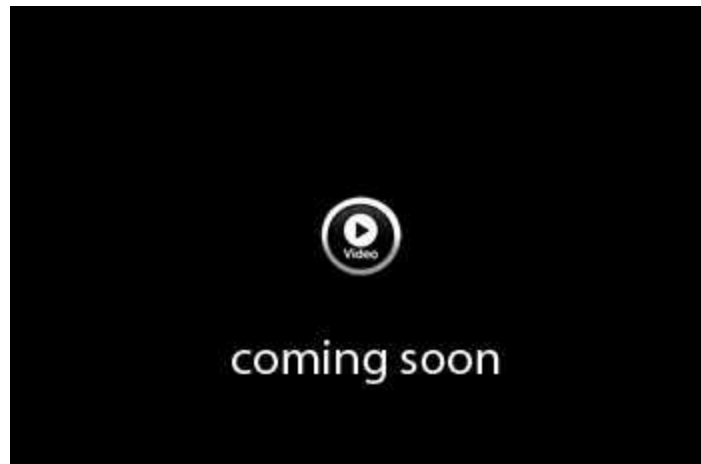
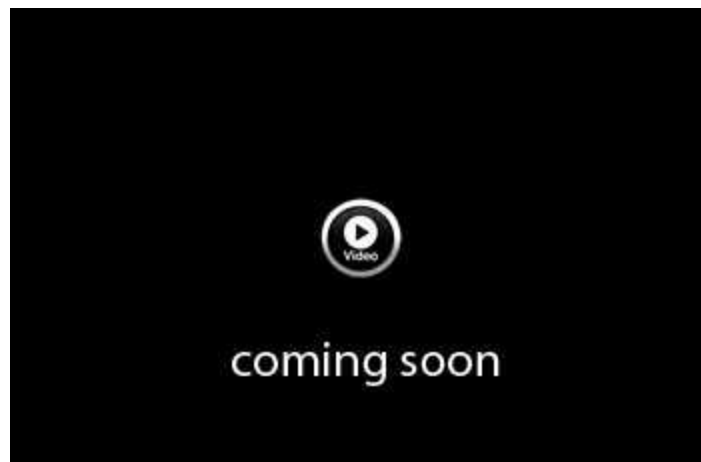


**FIGURE 3.19. A:** Parasternal long-axis view recorded in a healthy young patient.

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. 



**Video 3-19a**



**Video 3-19b**

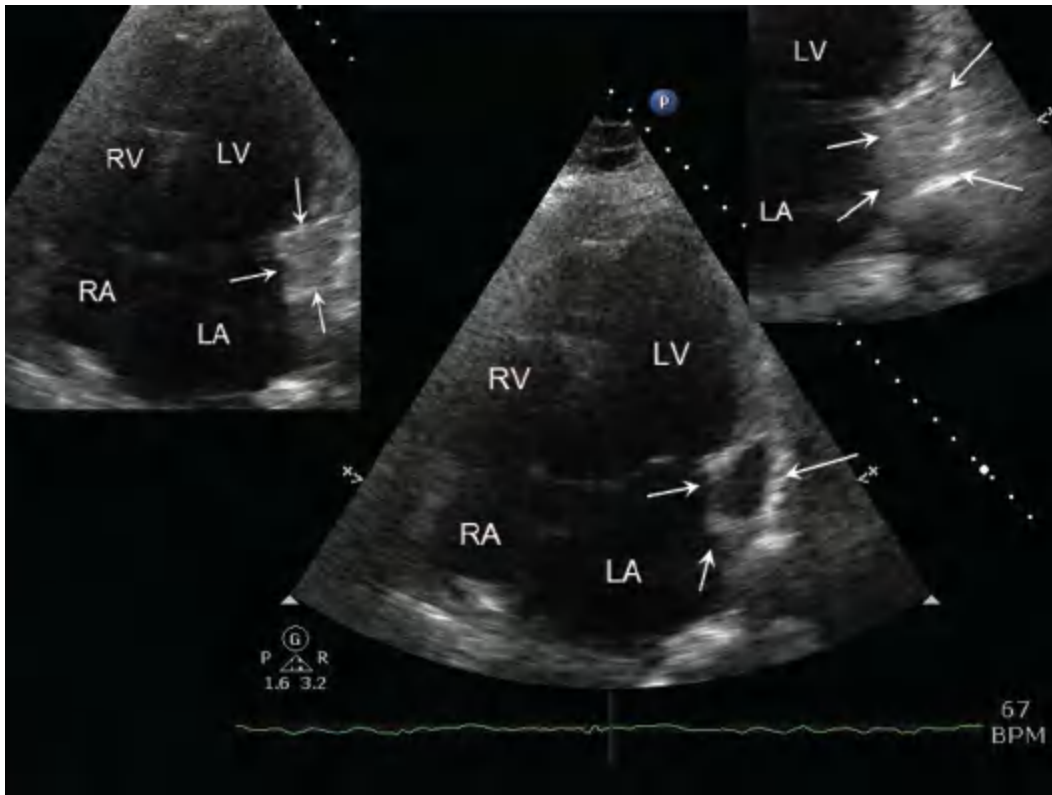
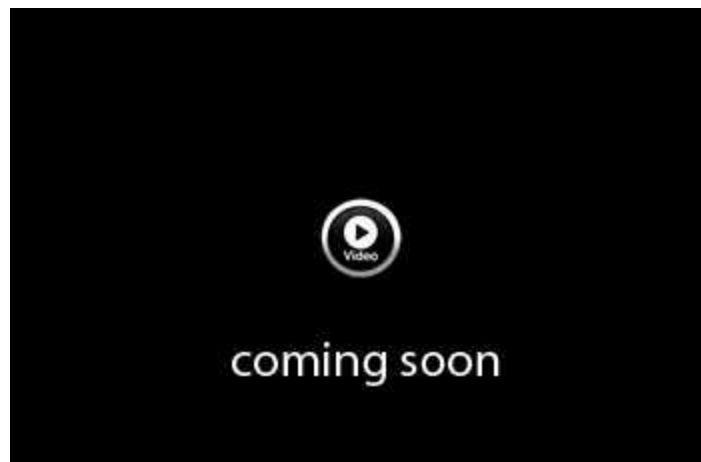


**FIGURE 3.20.** Apical four-chamber view recorded in a patient with a persistent left superior vena cava to coronary sinus communication. The central image was recorded without contrast. Note the oval-shaped echo-free space in the lateral atrioventricular groove representing a dilated coronary sinus (*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. 📺



**Video 3-20**

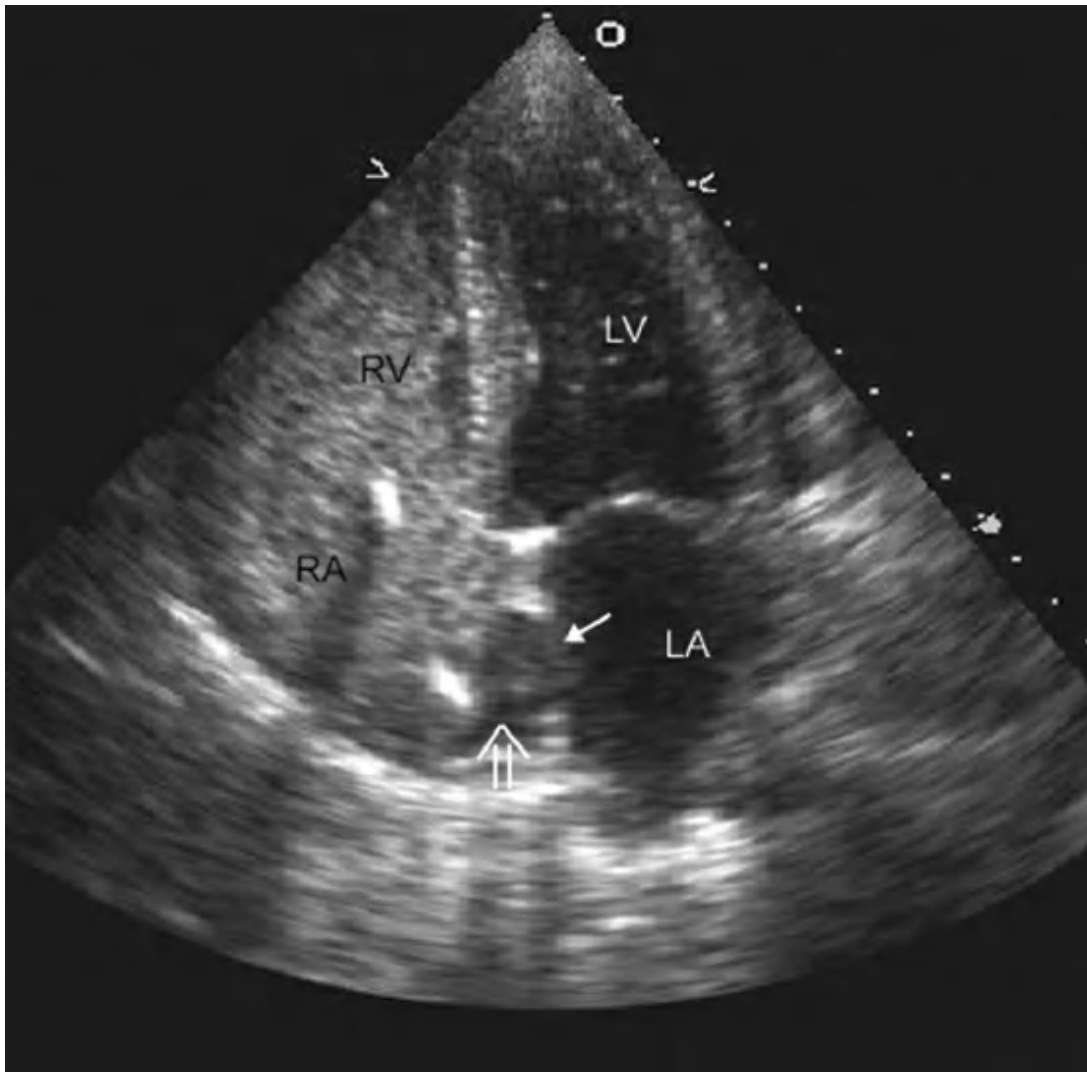
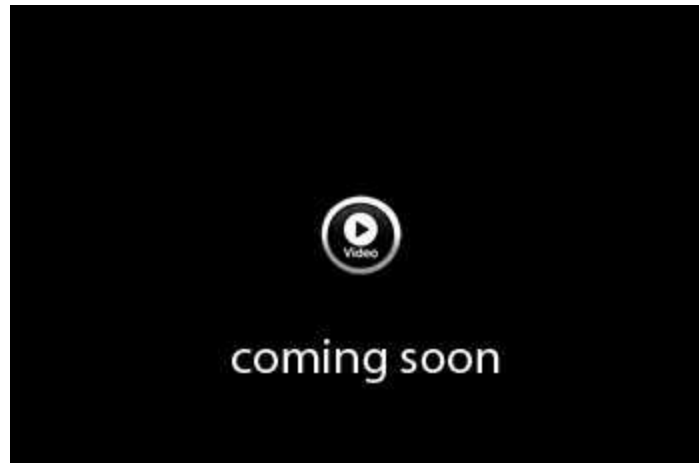


**FIGURE 3.21.** Apical four-chamber view recorded in a patient after injection of agitated saline into an upper extremity vein. Note the area of absent contrast effect (*large arrow*) along the most superior portion of the atrial septum, which is due to competitive flow from noncontrast-enhanced inferior *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*. 



**Video 3-21**

## **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.

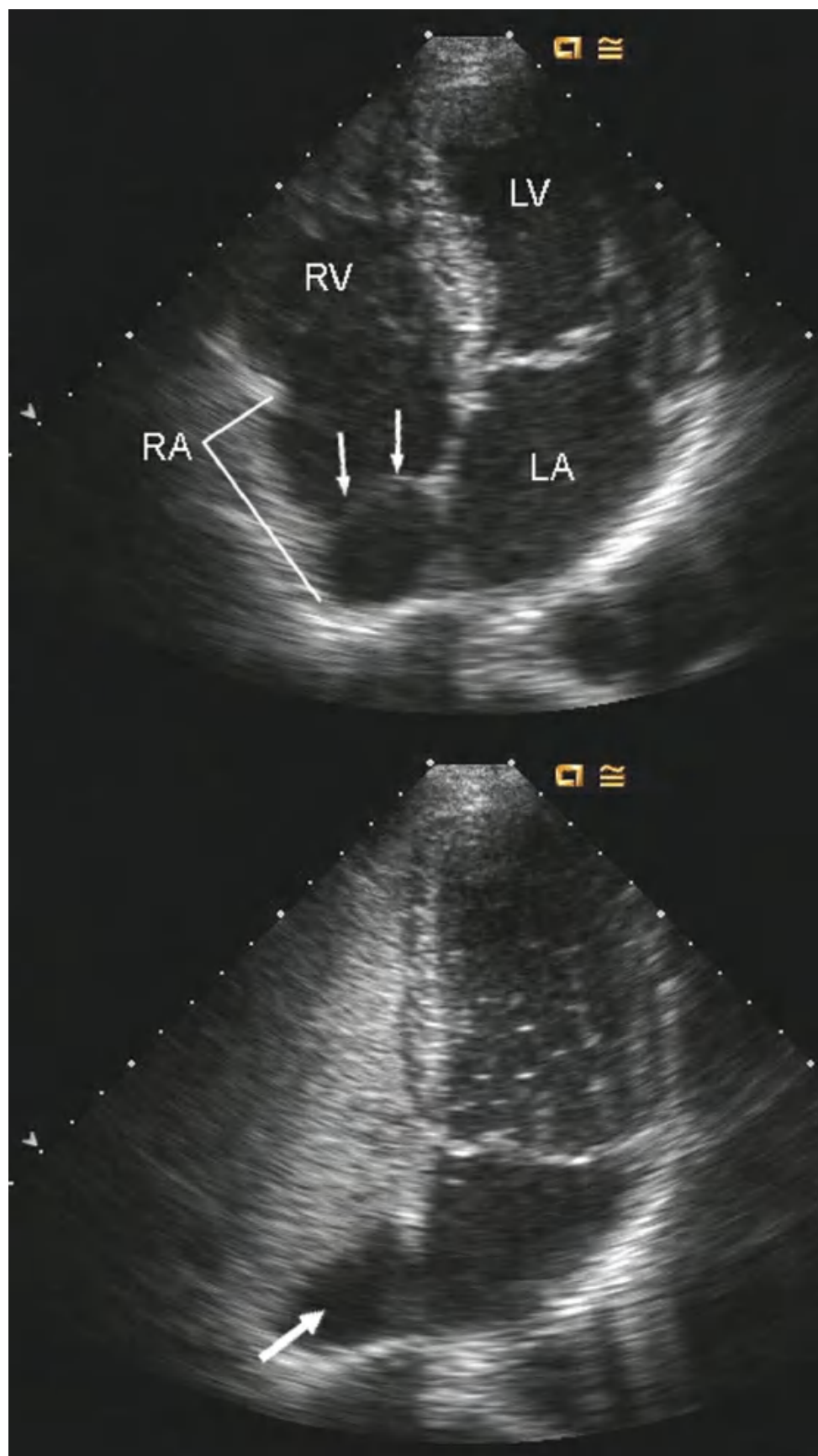
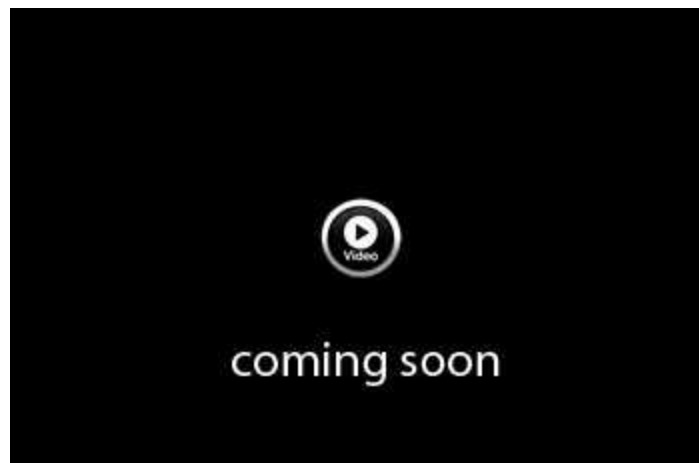
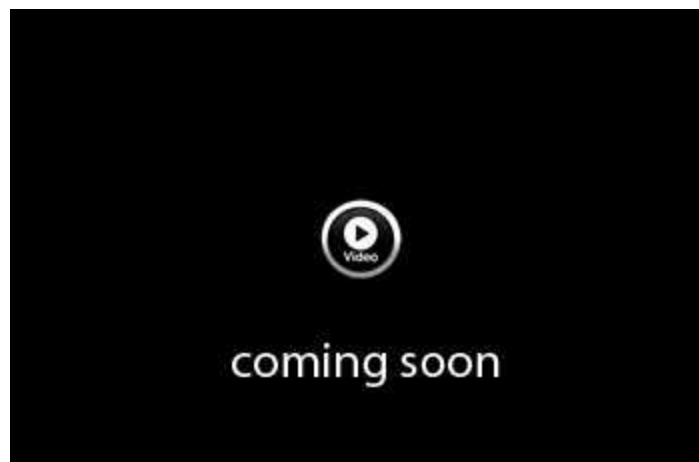


**FIGURE 3.22.** Apical four-chamber view recorded in a patient with a vague linear echo traversing the right atrium (*arrows*). This represents a complete eustachian valve which effectively subdivides the right atrium into two segments (upper panel). After injection of saline contrast, (lower panel) 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. 📺



**Video 3-22a**



**Video 3-22b**

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.

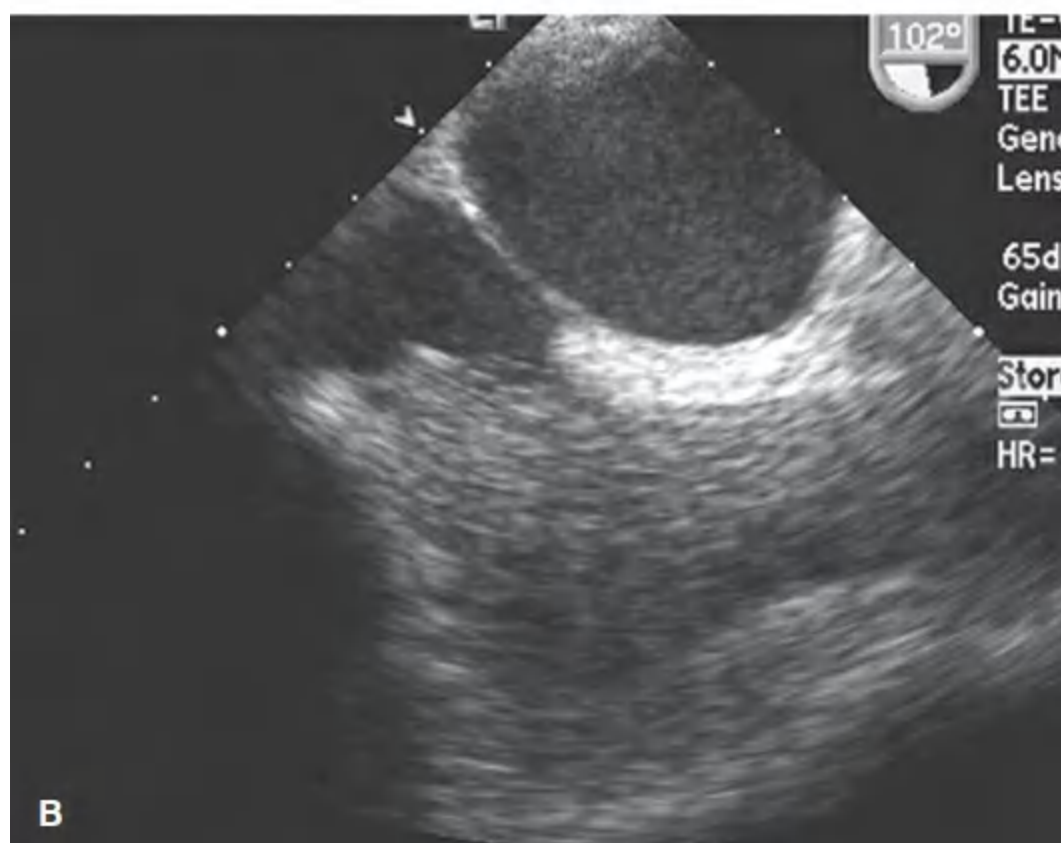
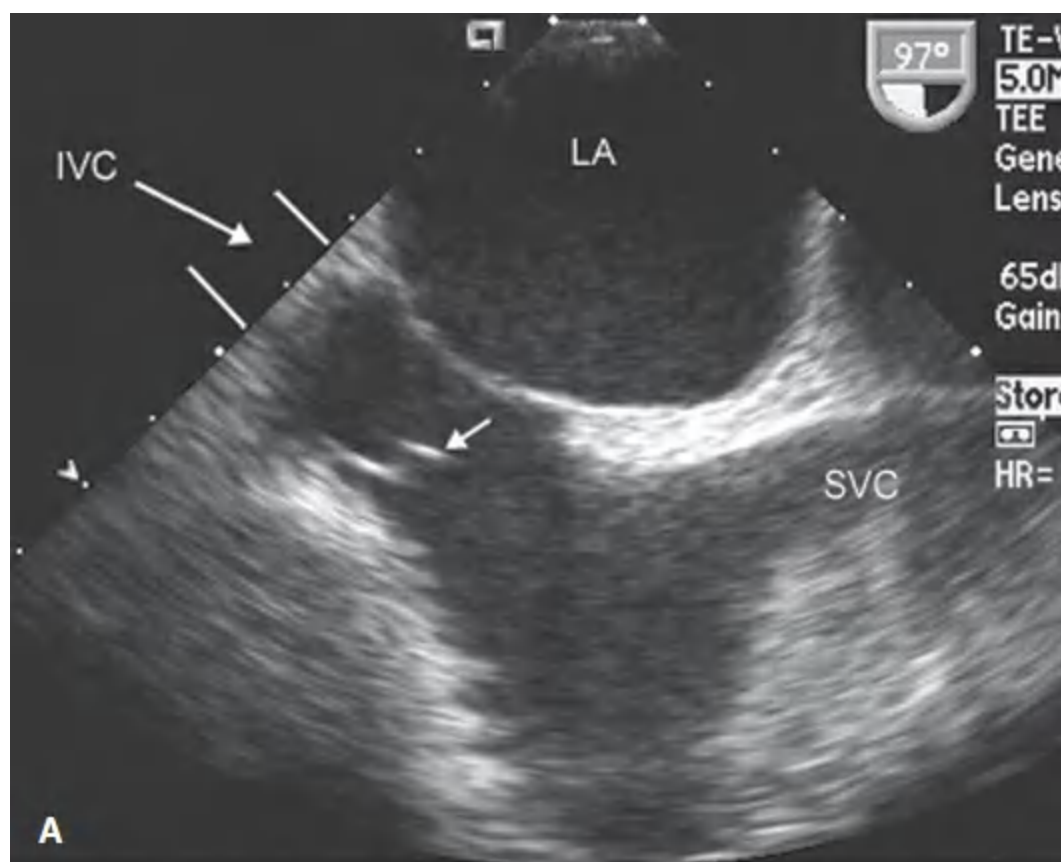
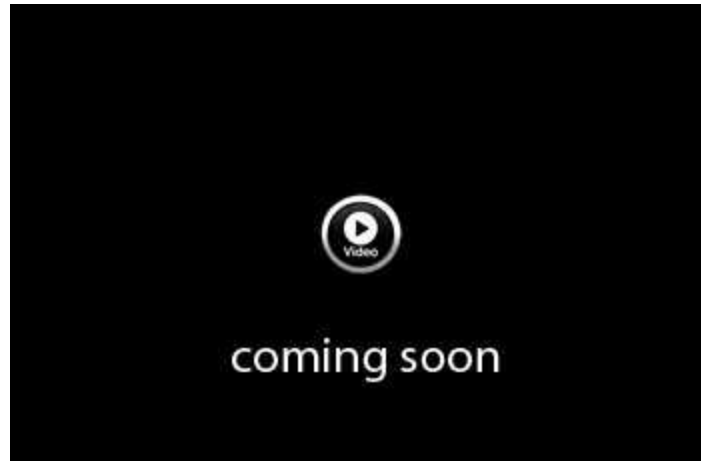
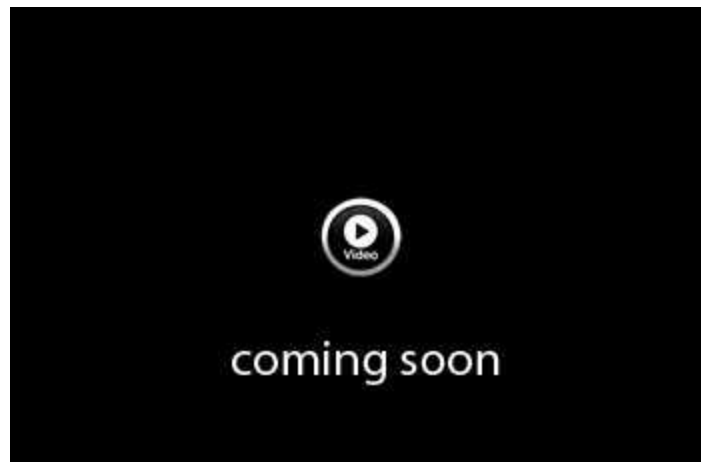


**FIGURE 3.23.** Transesophageal echocardiogram demonstrating a prominent eustachian valve and margination of contrast-enhanced blood flow. **A:** Image was recorded before injection of contrast into an upper extremity vein. Note the prominent eustachian valve adjacent to the inferior *vena cava* (*arrow*). **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**



**Video 3-23b**

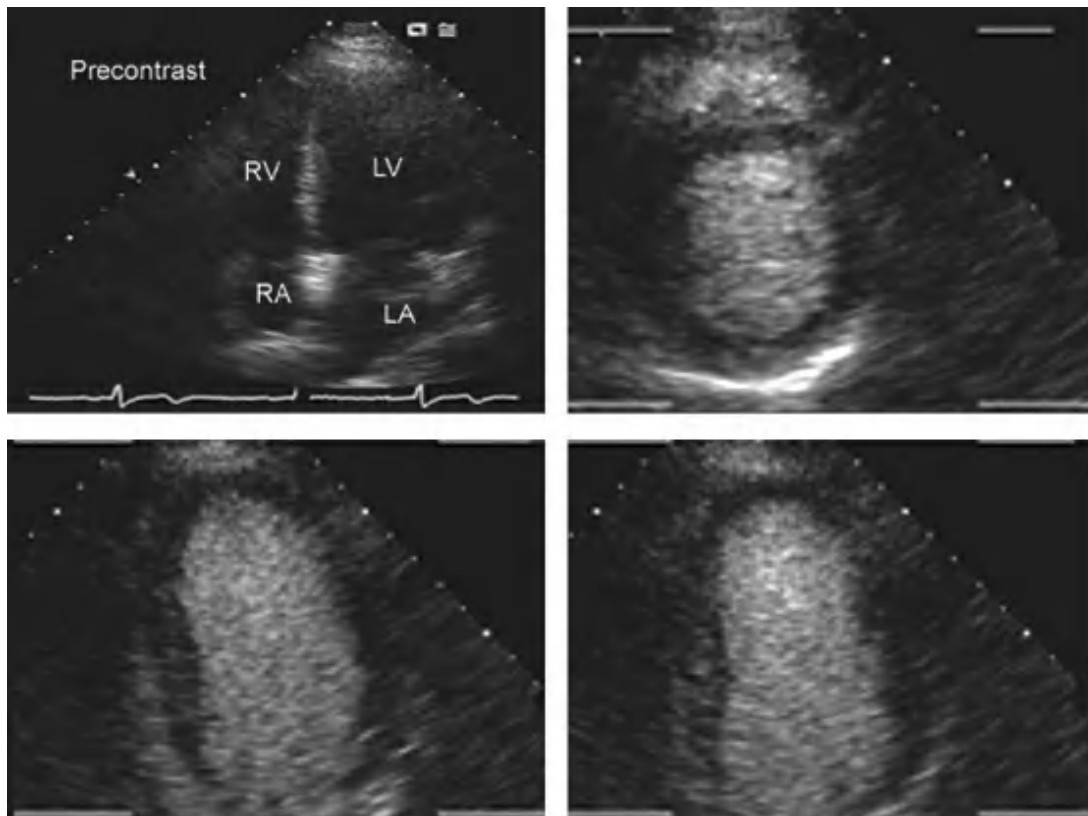
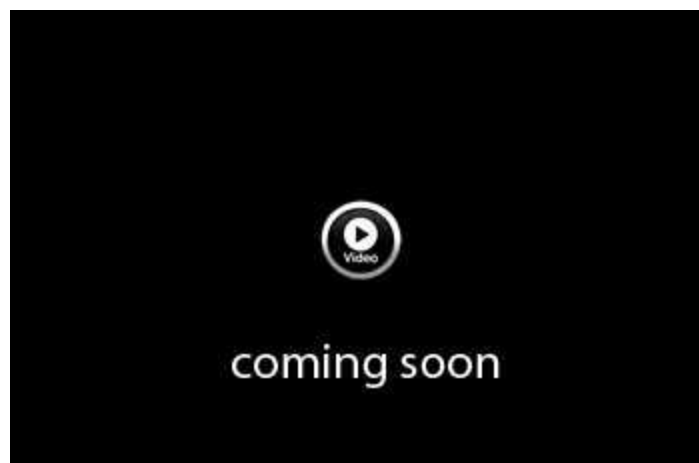
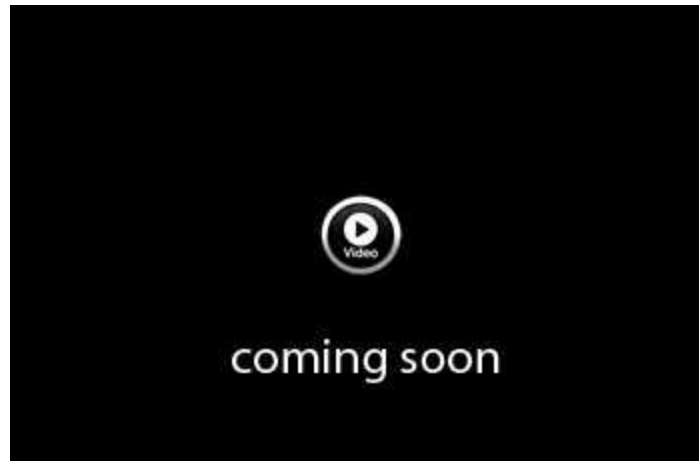


**FIGURE 3.24.** Example of left ventricular opacification after intravenous injection of a perfluorocarbon-based contrast agent. **Top left:** A baseline apical four-chamber view. Note the poor visualization of the apex and lateral wall. The other three panels were recorded after intravenous injection 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**



### Video 3-24abcd

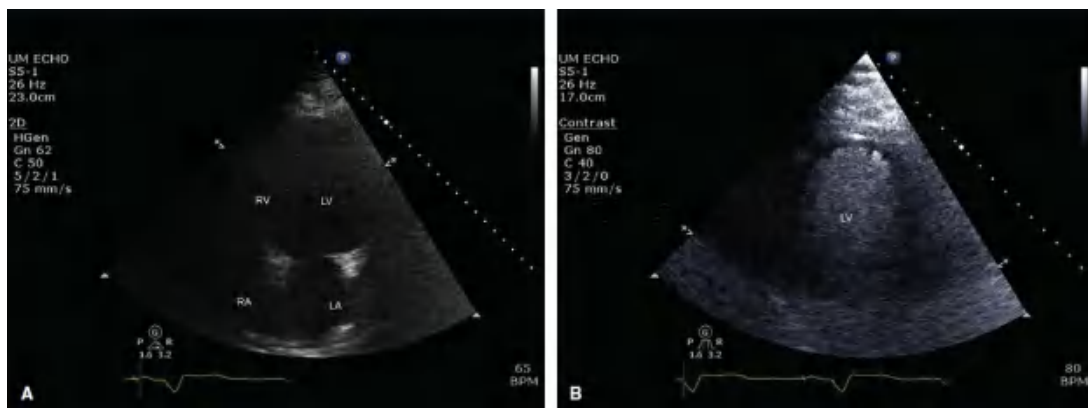
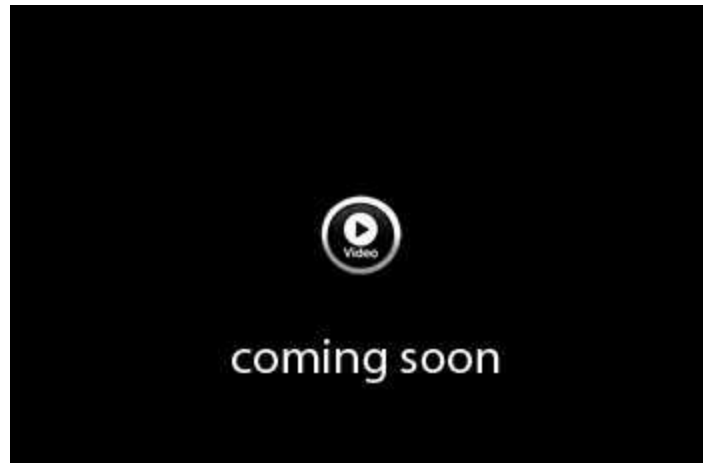
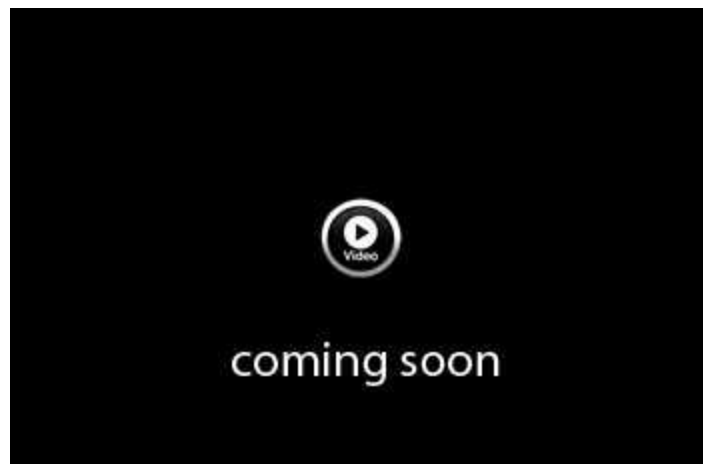


**FIGURE 3.25.** Transthoracic echocardiogram recorded in an obese patient undergoing mechanical ventilation in the surgical intensive care unit. **A:** Apical four-chamber view from which no accurate visualization of cardiac chambers or assessment of ventricular function can be made. Note the approximate 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. 



**Video 3-25a**



**Video 3-25b**

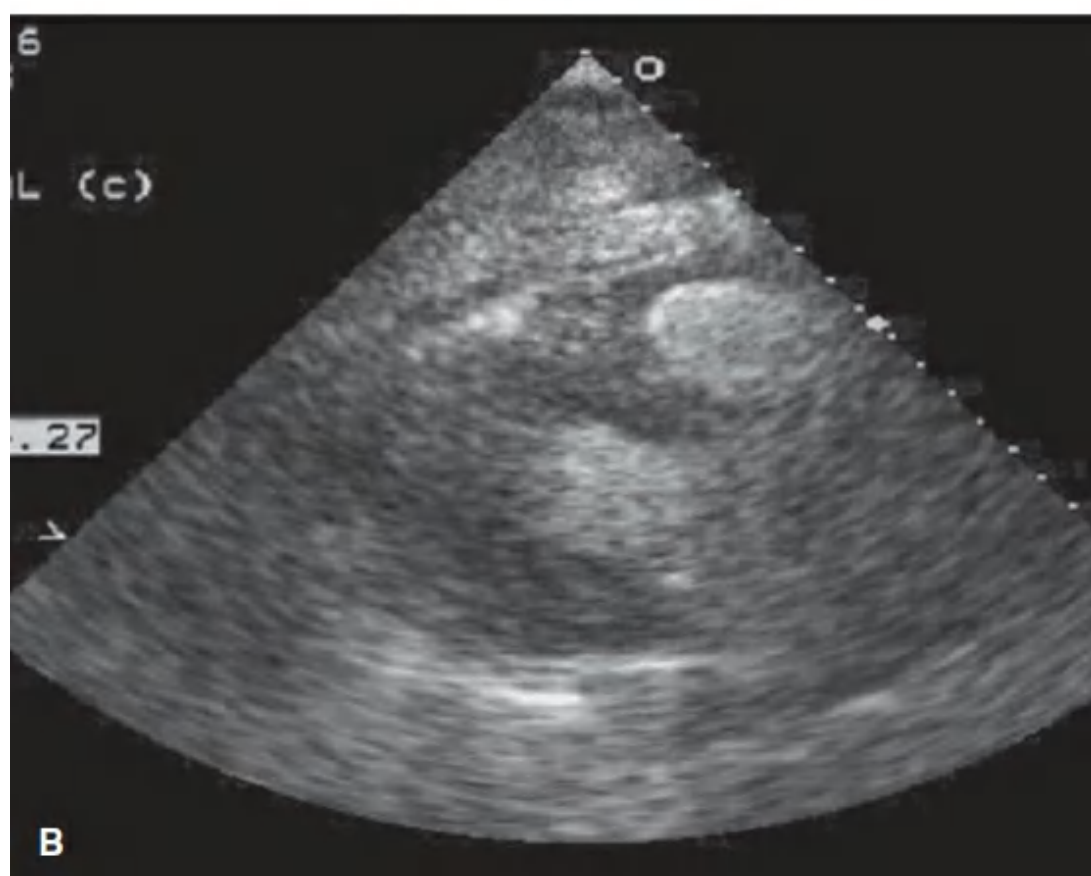
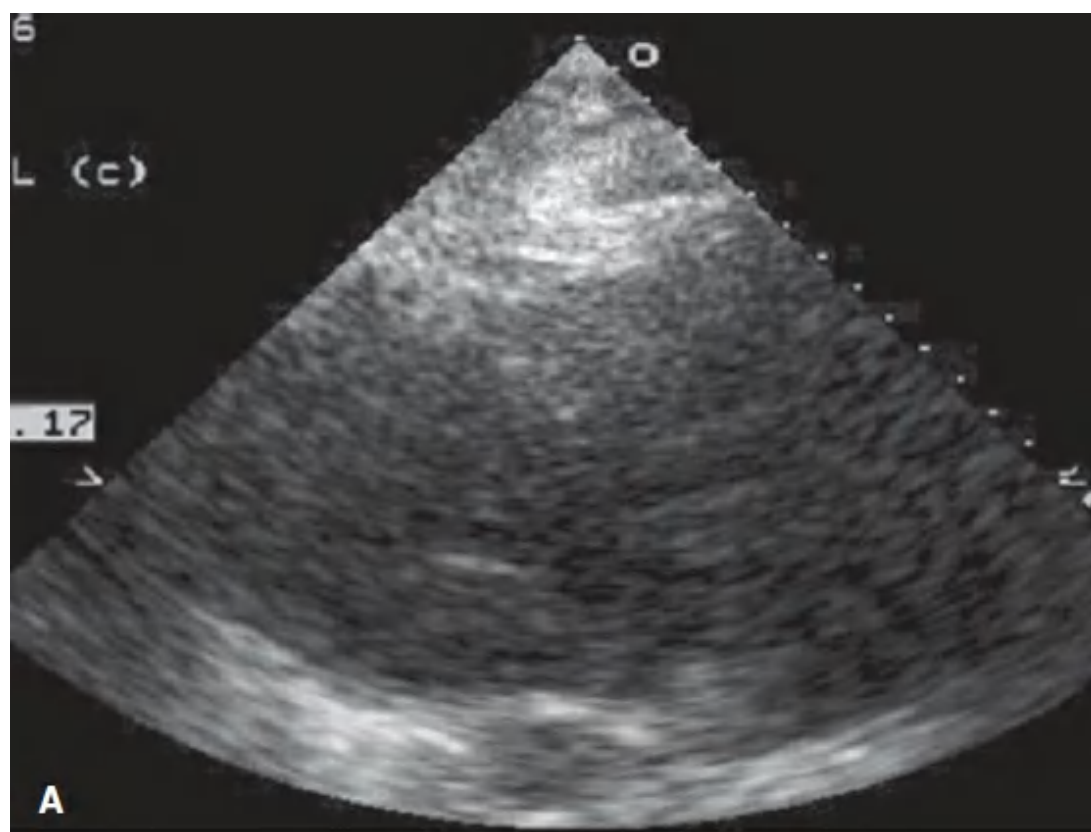
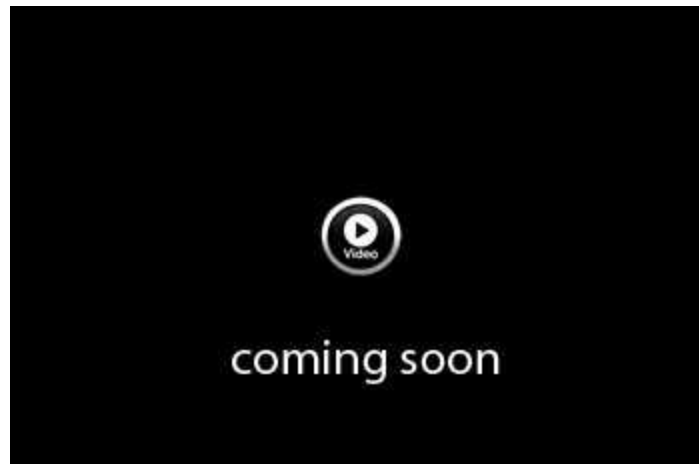
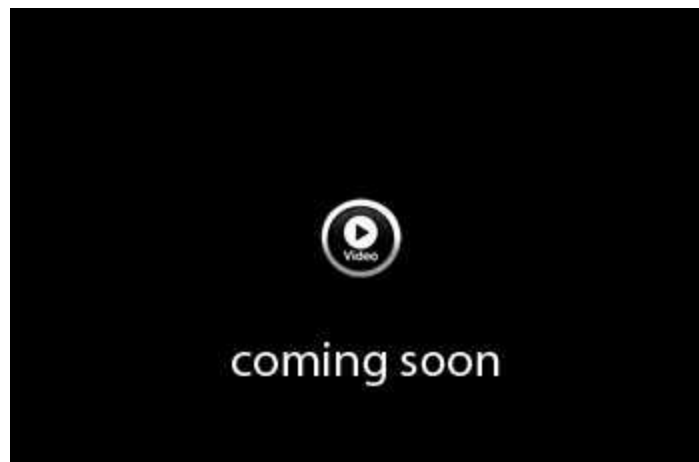


**FIGURE 3.26.** Parasternal long-axis echocardiogram recorded in a morbidly obese patient in an intensive care unit undergoing mechanical ventilation. **A:** Image was recorded before injection of a perfluorocarbon-based contrast agent. Even in the real-time image, it is difficult to identify any cardiac 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. 



**Video 3-26a**



**Video 3-26b**



## **MISCELLANEOUS APPLICATION OF LEFT VENTRICULAR CONTRAST**

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.

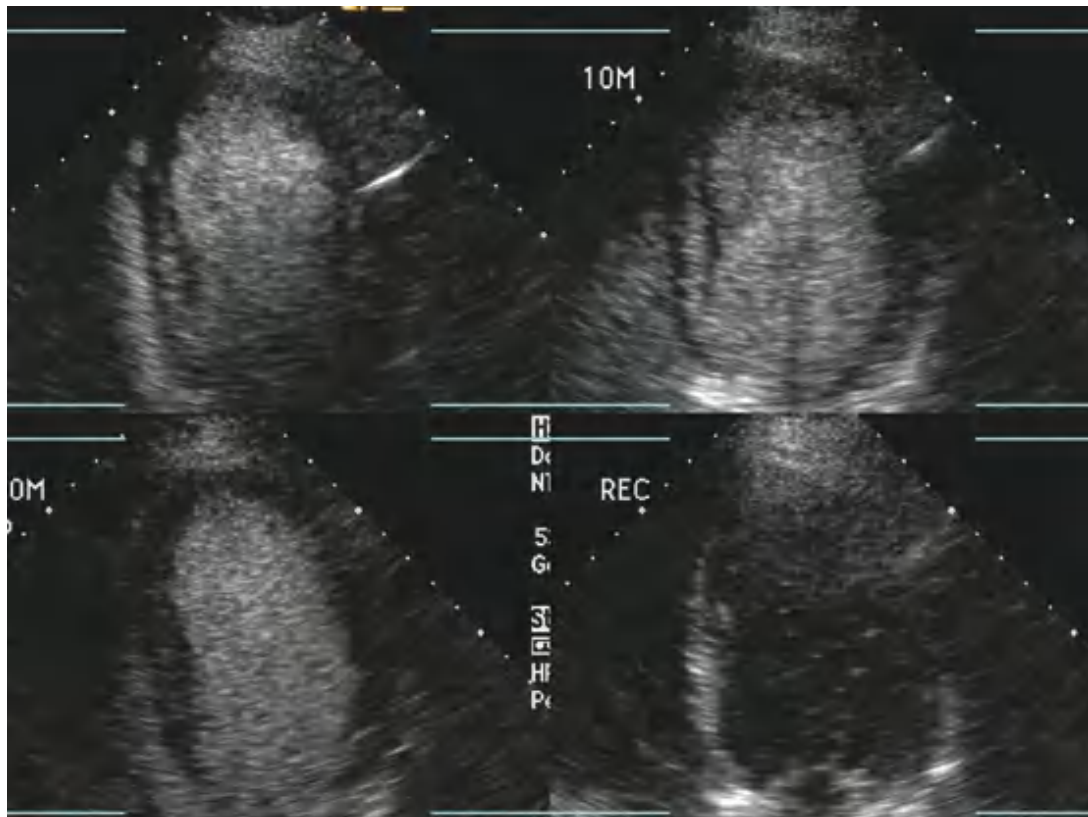
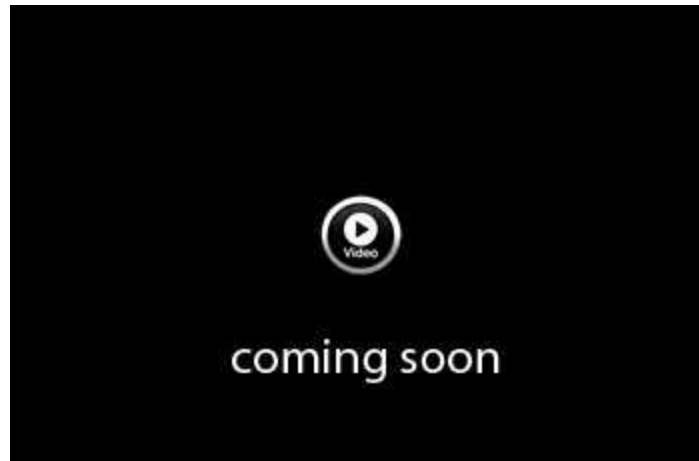


**FIGURE 3.27.** Demonstration of left ventricular contrast during a dobutamine stress echocardiogram. In this quad screen format, the lower right image was recorded during recovery when the majority of contrast is no longer present. Notice the suboptimal image quality with adequate visualization only 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. 



**Video 3-27**

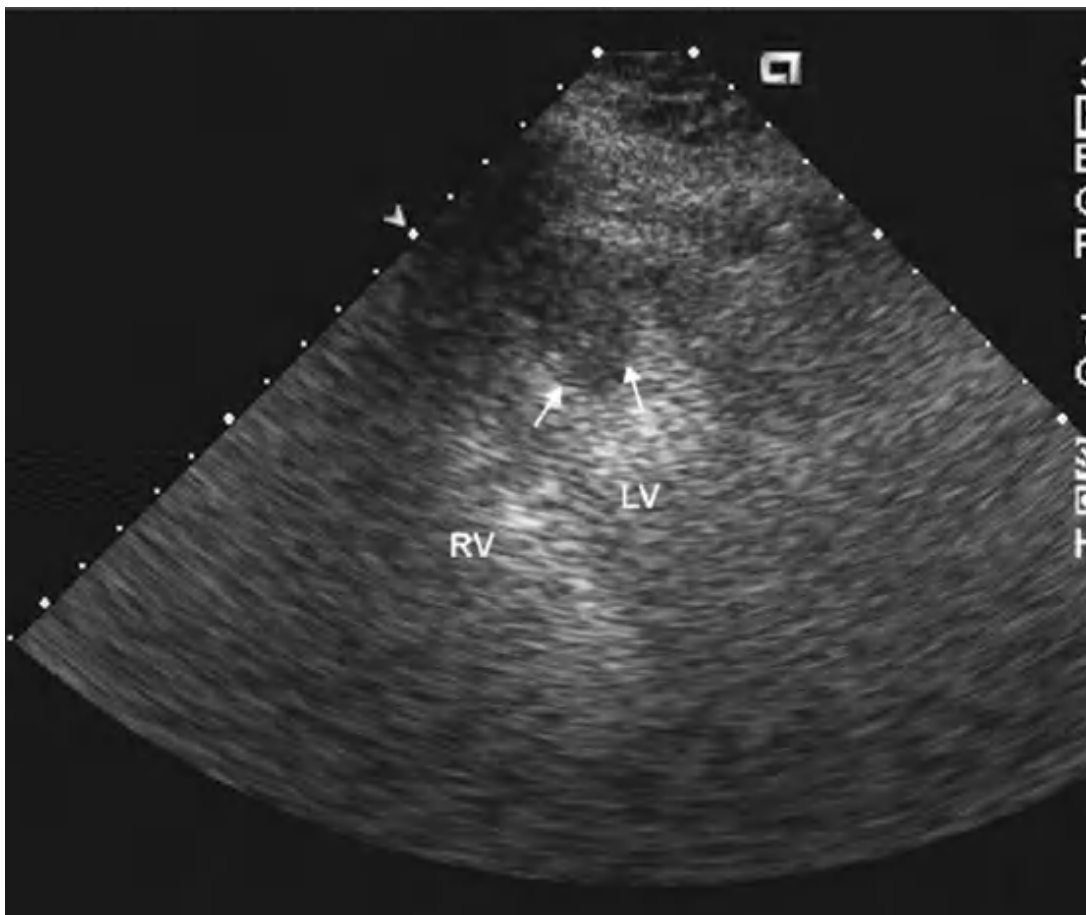
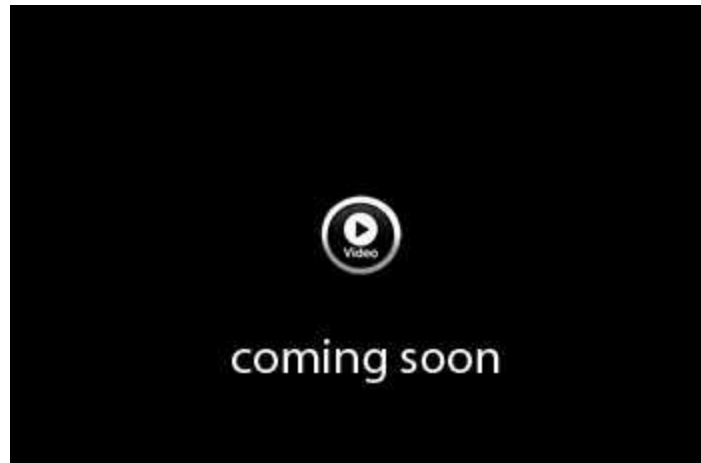


**FIGURE 3.28.** Apical view recorded in a patient with a vague echo density on noncontrast imaging. After intravenous injection of a perfluorocarbon-based agent, a distinct spherical filling defect is noted in the apex, consistent with a pedunculated apical thrombus (arrows). 



### **Video 3-28**

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](#)).

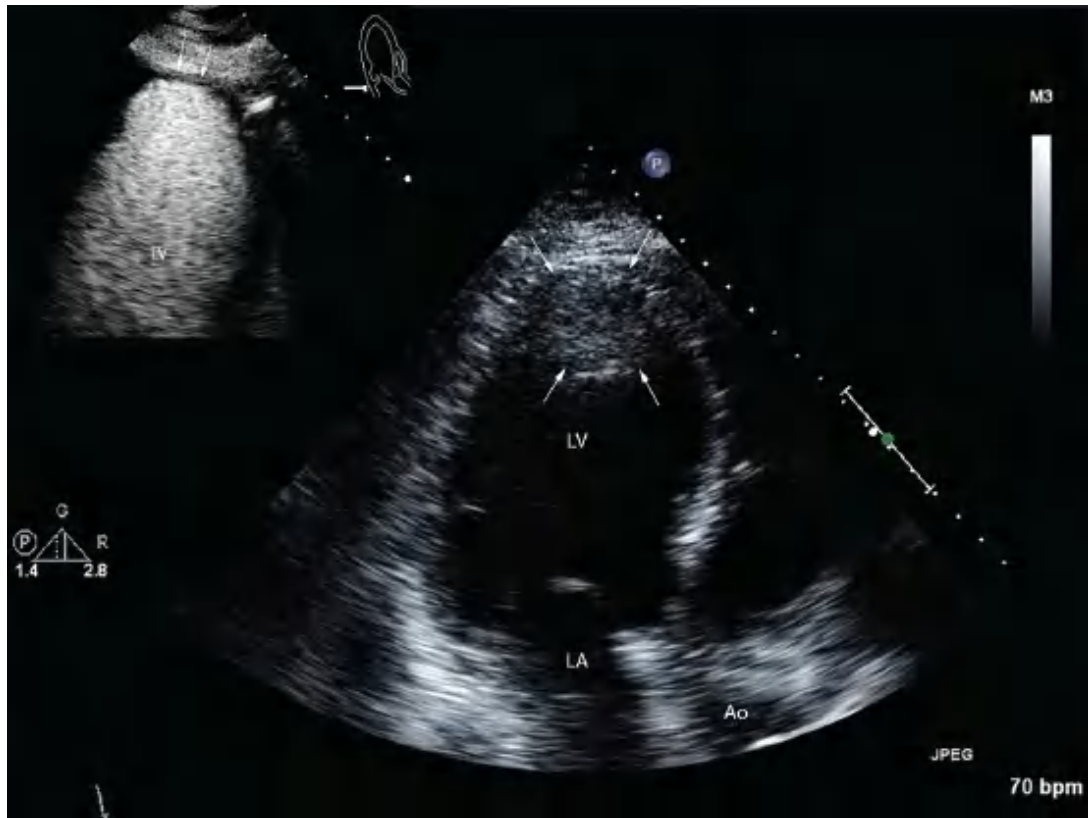
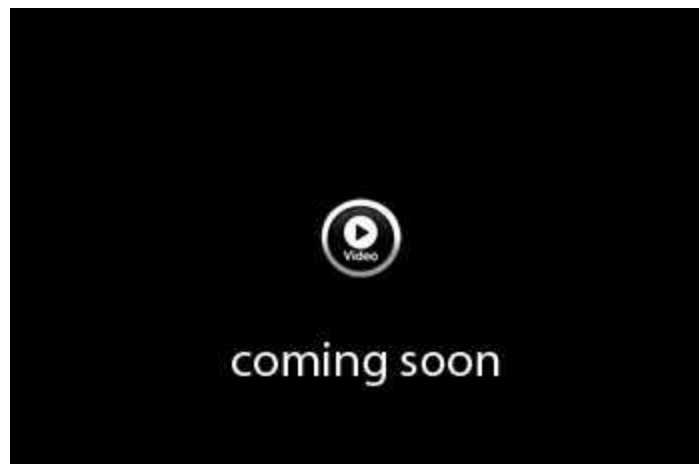
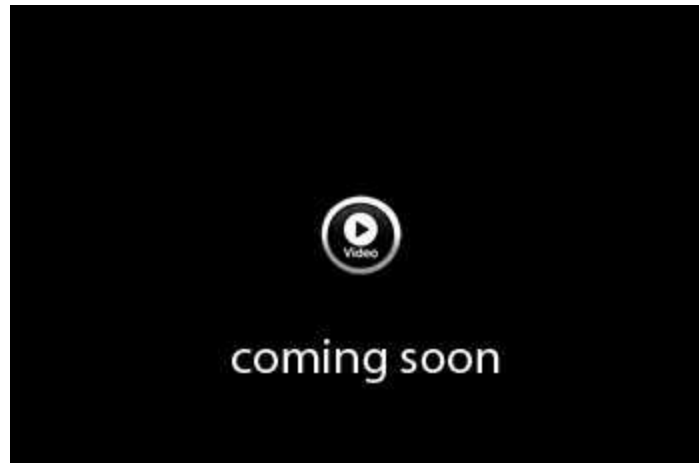


**FIGURE 3.29.** Apical long-axis view recorded in a patient with an ischemic cardiomyopathy and left ventricular systolic dysfunction. In the central image note the dilated hypokinetic left ventricle and the vague echo density at the left ventricular apex (*arrows*). The inset at the upper left was 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**



### **Video 3-29b**

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](#)).

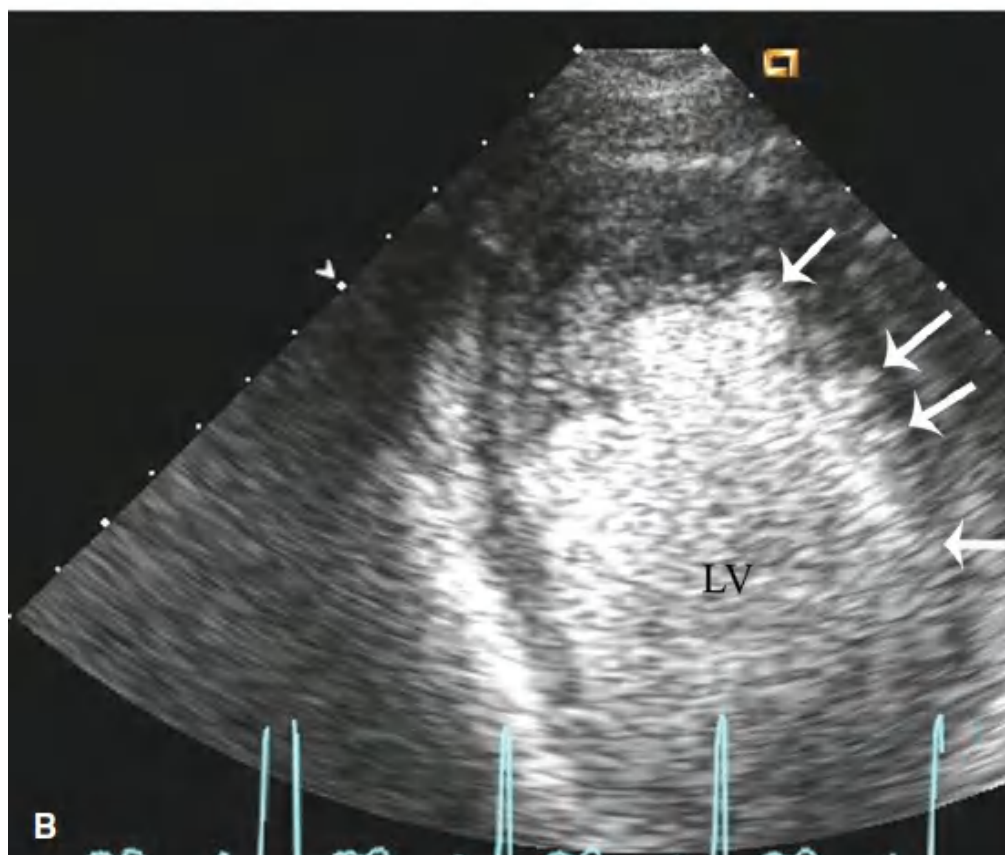
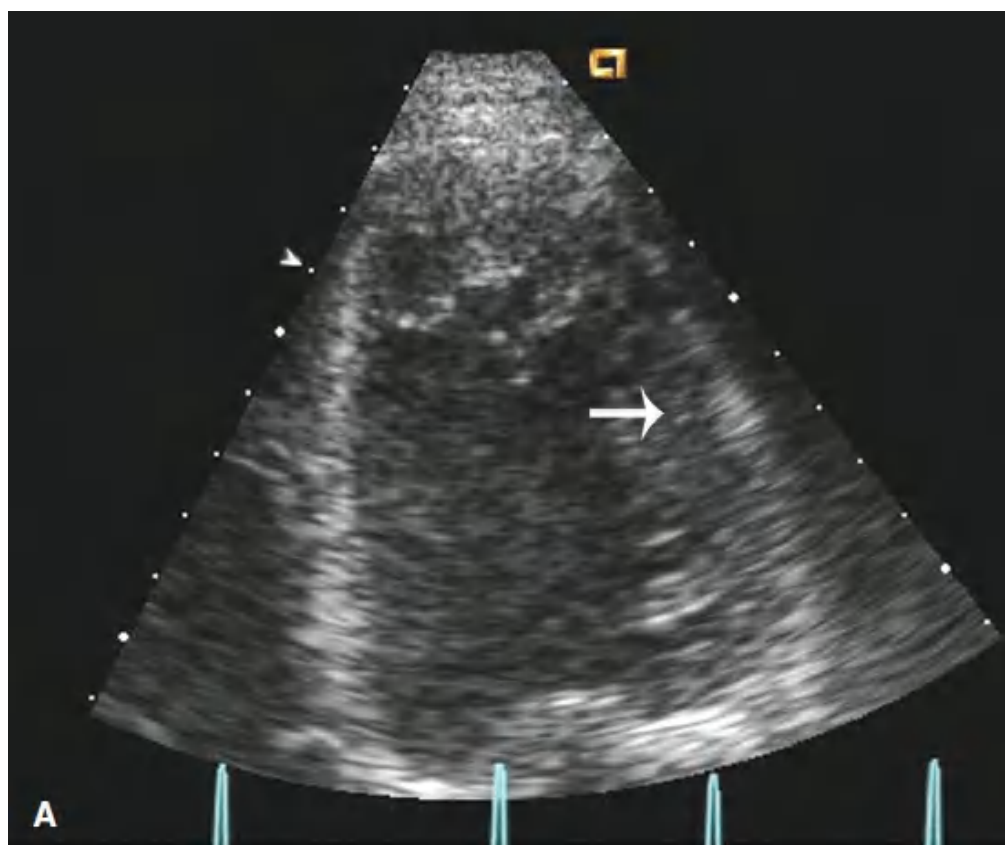
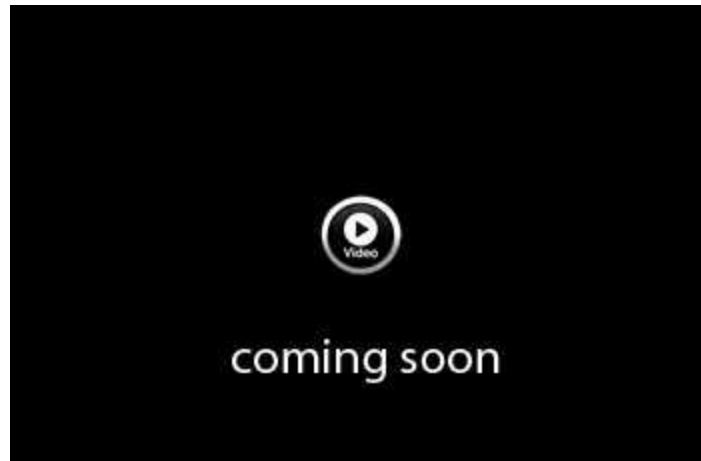


**FIGURE 3.30.** Apical four-chamber view recorded in a patient with ventricular noncompaction. **A:** Without contrast, note the irregular thickening of the apex and lateral wall (*arrow*). After injection of a contrast agent for left ventricular opacification (B), note the contrast in the multiple sinusoids in the apex and lateral wall (*arrows*). 



**Video 3-30**



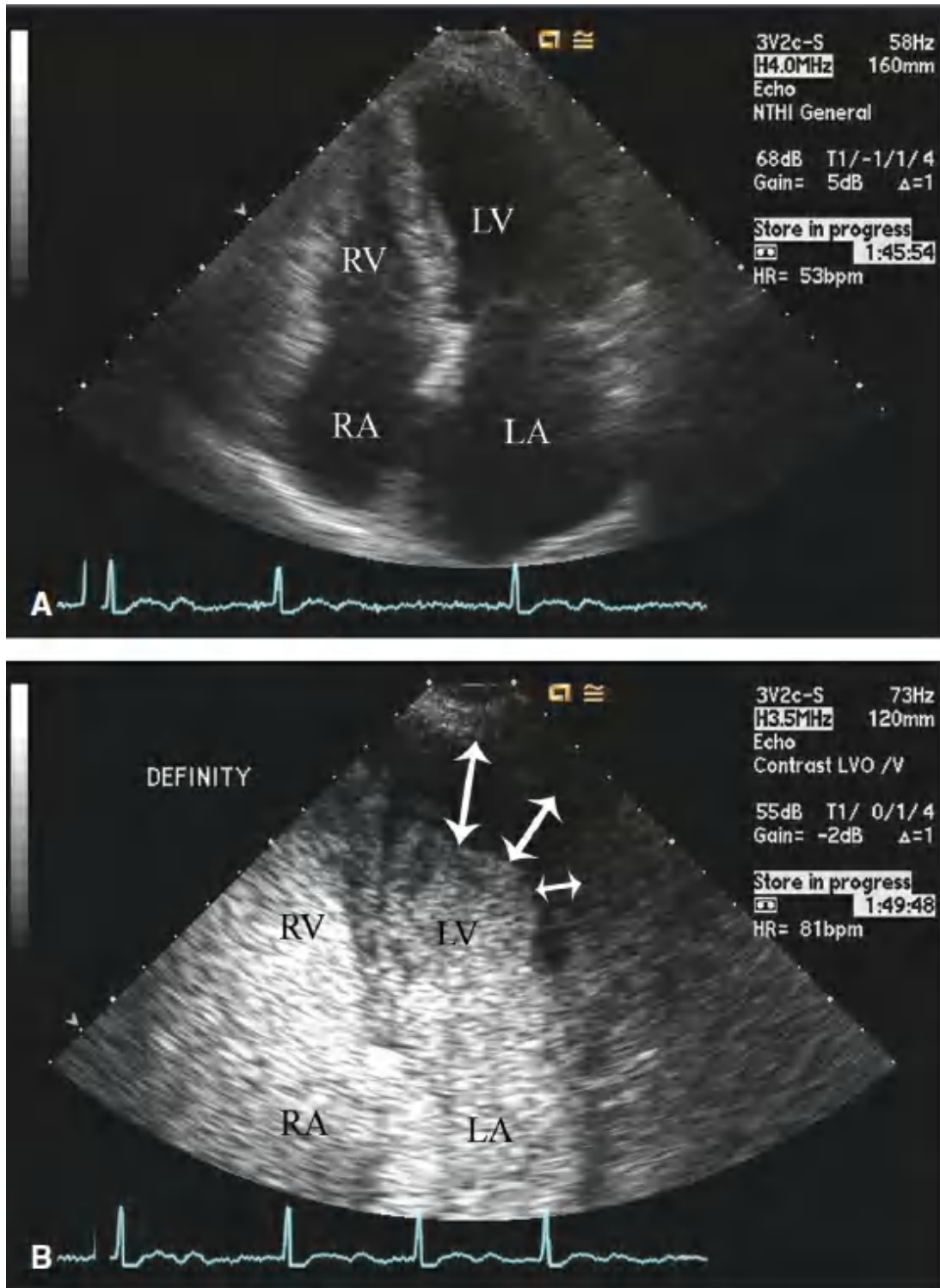
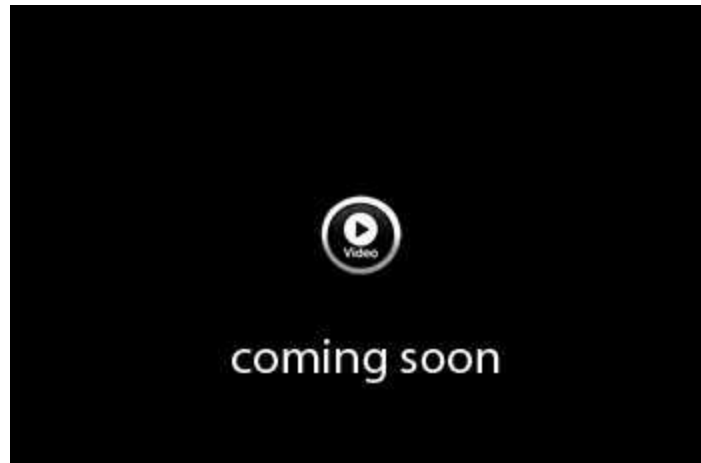
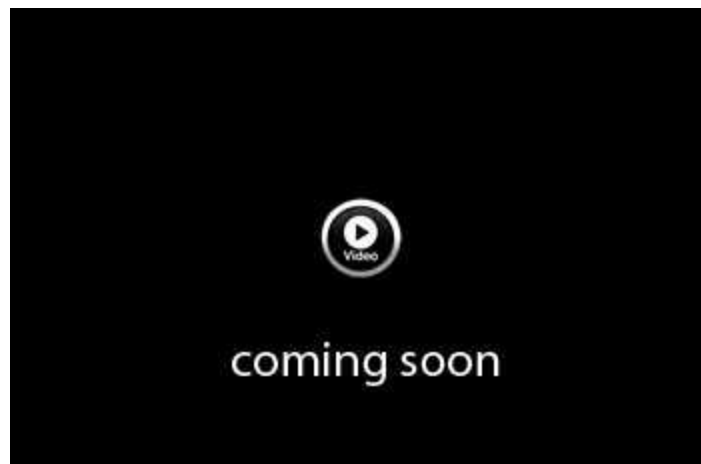


**FIGURE 3.31.** Apical four-chamber view recorded in a patient with an apical variant of hypertrophic cardiomyopathy. **A:** Recorded with standard B-mode imaging, from which pathologic thickening of the apex is not appreciable. After injection of contrast for left ventricle opacification (**B**), the pathologic thickness of the apical left ventricular walls (*double-headed arrows*) can be appreciated.



**Video 3-31a**



**Video 3-31b**

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).

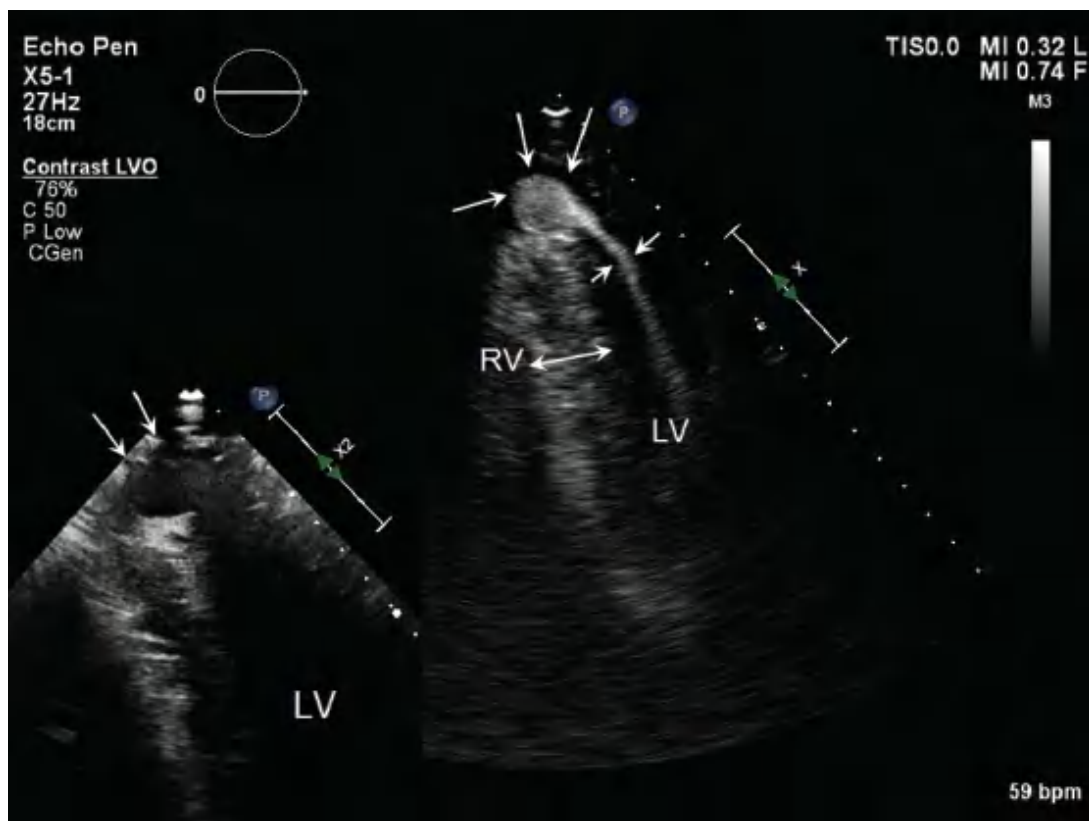
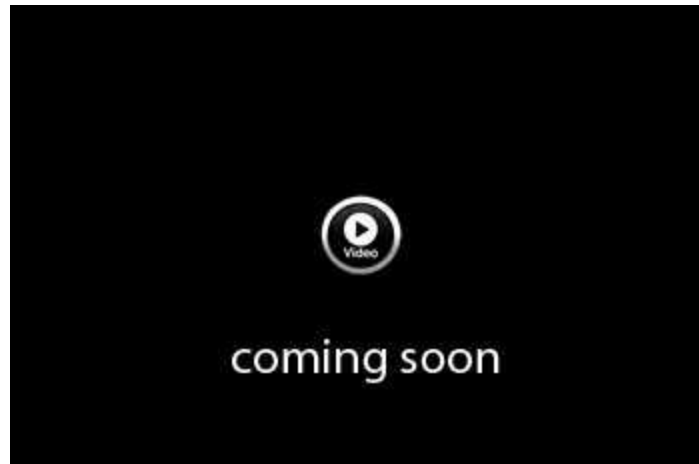
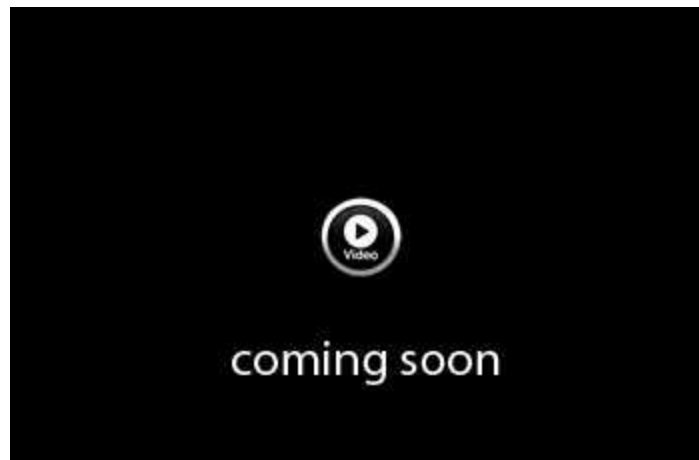


**FIGURE 3.32.** Off-axis apical view recorded in a patient with a nonobstructive apical hypertrophic cardiomyopathy. The central image was recorded after injection of intravenous contrast for left ventricular opacification. Note the marked hypertrophy of the ventricular septum (*double-headed arrow*) 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.

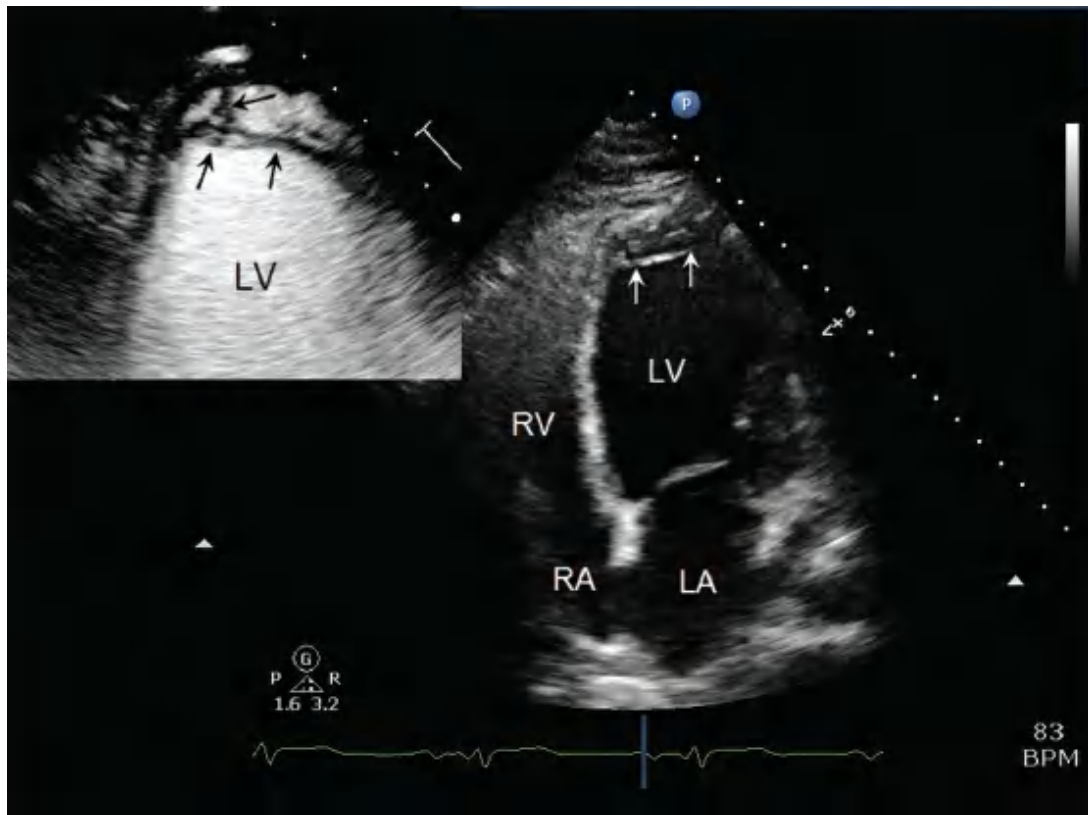
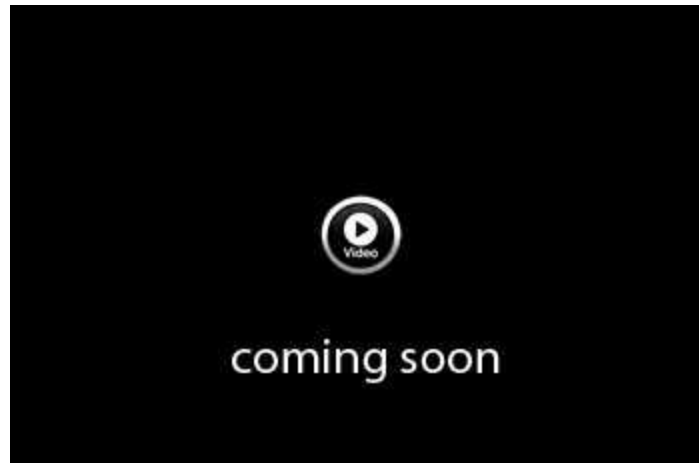
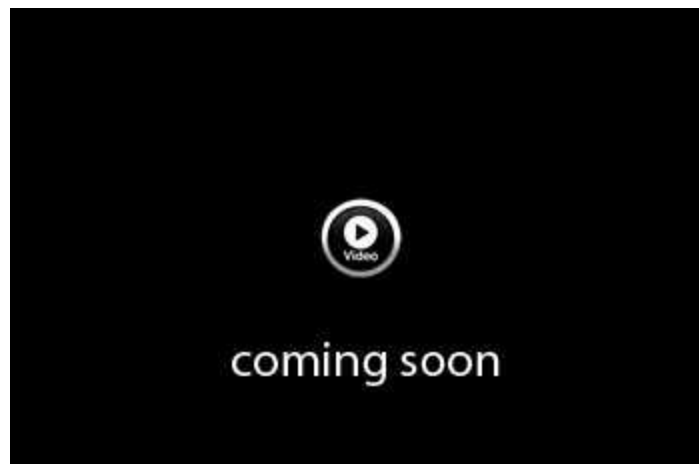


**FIGURE 3.33.** Apical four-chamber view recorded in a patient with an ischemic cardiomyopathy and an anteroapical myocardial infarction. In the central image note the vague complex echo densities at the left ventricular apex potentially representing thrombus (*arrows*). The inset at the upper left 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. 



**Video 3-33a**



**Video 3-33b**

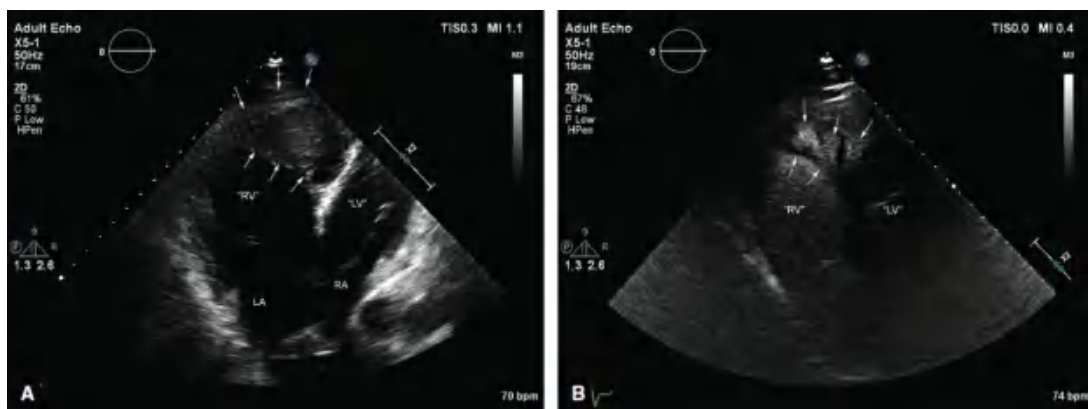
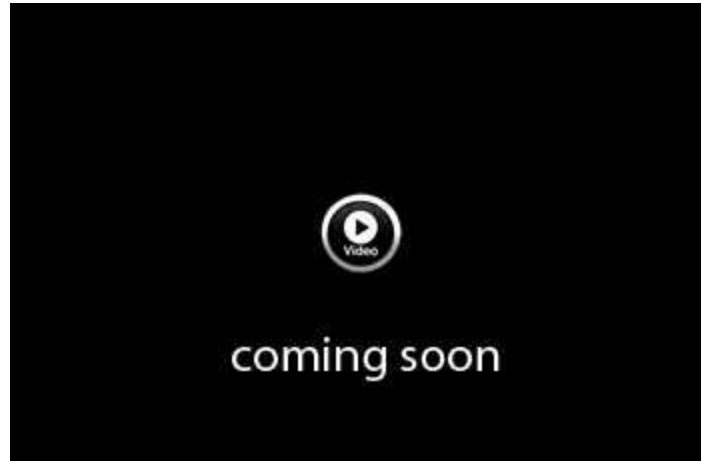
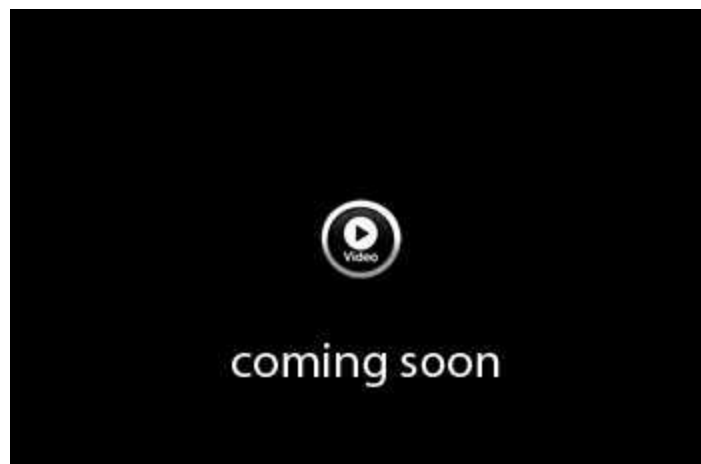


**FIGURE 3.34.** Apical view recorded in a young patient with L-transposition of the great arteries. **A:** Note the dilated anatomical right ventricle which is markedly hypertrophied related to systemic pressure. Note that the distal half of the right

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**

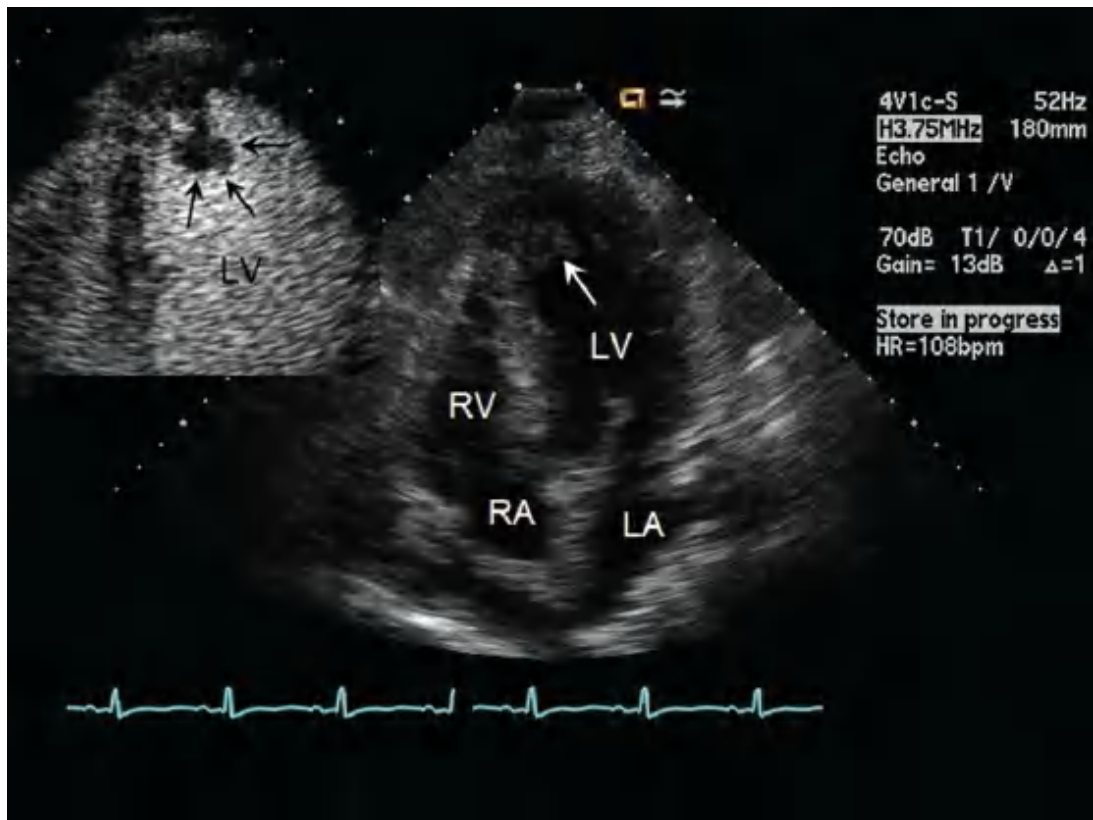
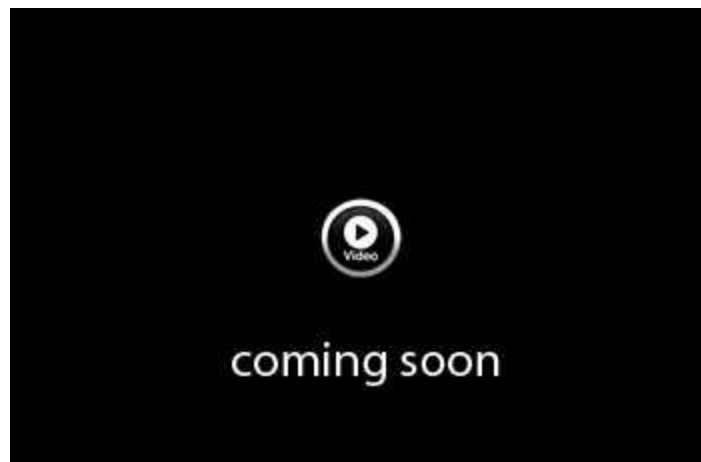
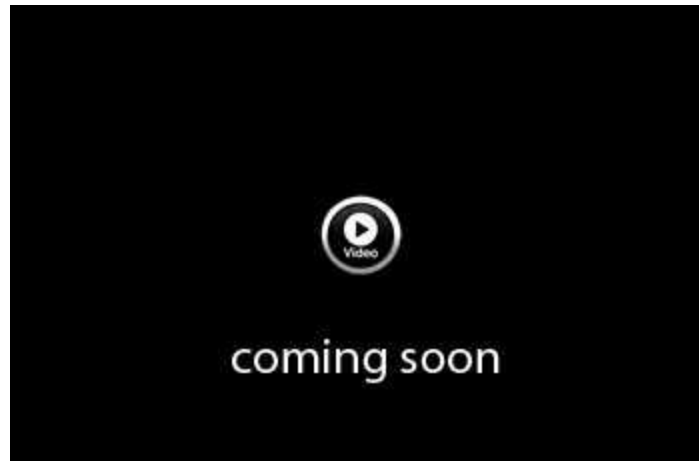


**FIGURE 3.35.** Apical four-chamber view recorded in a patient with metastatic malignancy and a neurologic event. In the central image note the hint of an apical mass (*arrow*). The inset at the upper left was recorded following injection of contrast for ventricular opacification in which a pedunculated mobile mass can clearly be identified in the left ventricular apex (*arrows*). 



**Video 3-35a**





Video 3-35b

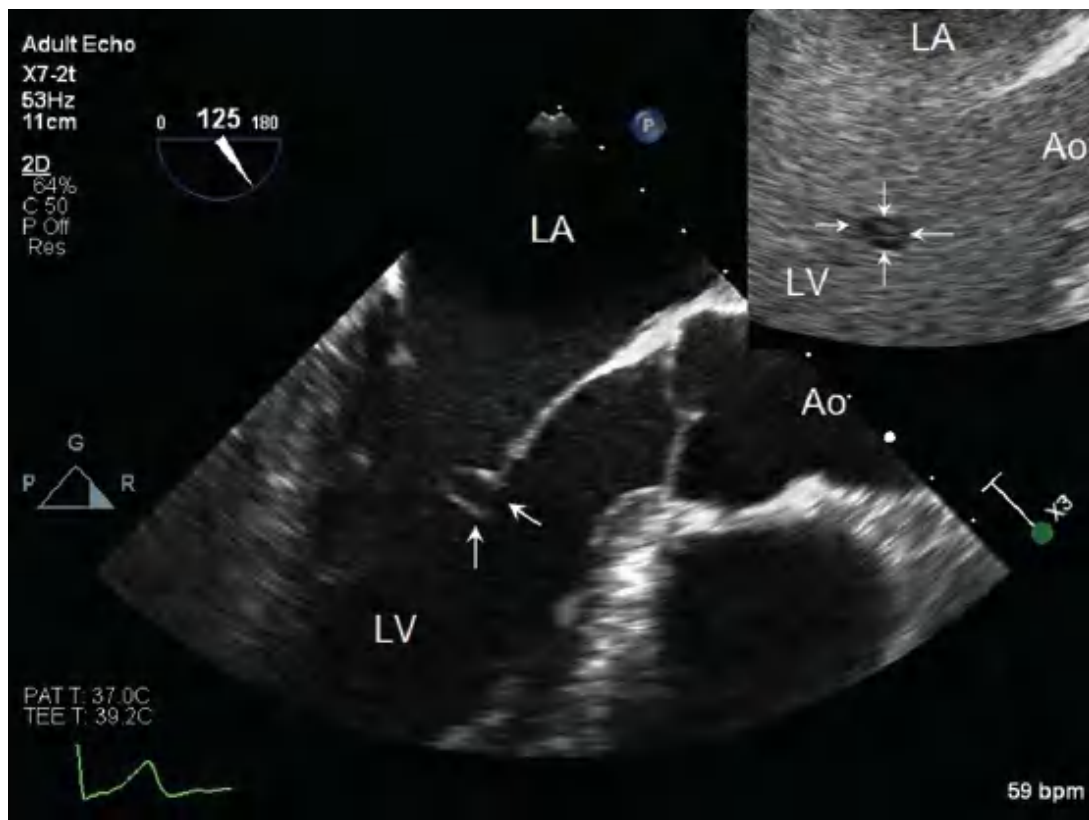
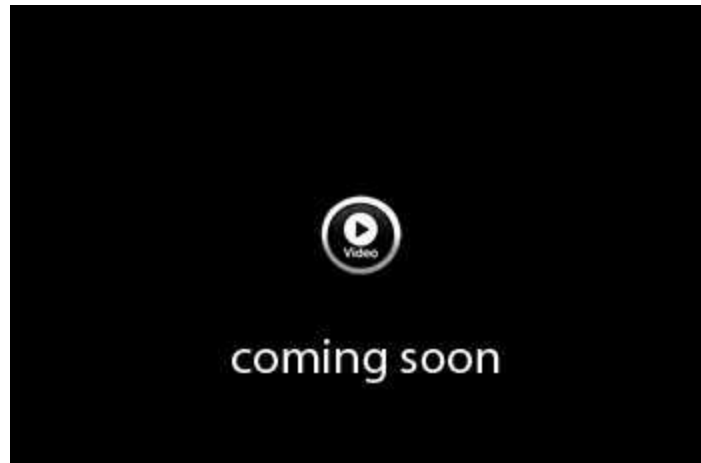
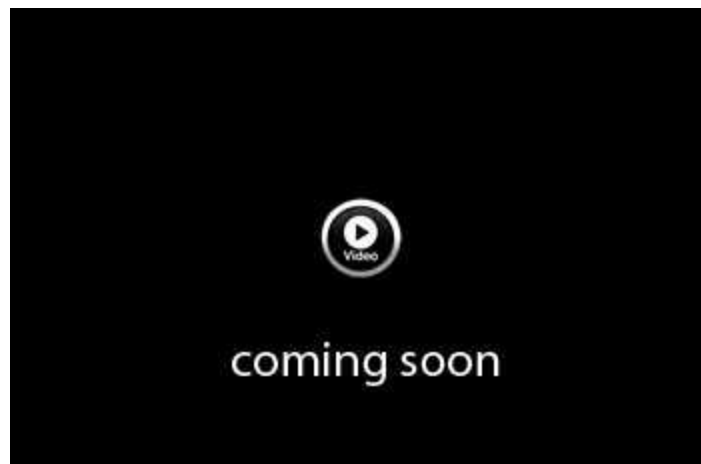


**FIGURE 3.36.** Transesophageal echocardiogram recorded in a young patient prior to an electrophysiologic procedure. Incidental note was made of a small cystic mass on the tip of the anterior mitral valve leaflet (arrows in the central illustration). The inset at the upper right is an expanded view 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). 📺



**Video 3-36a**



**Video 3-36b**

## **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.

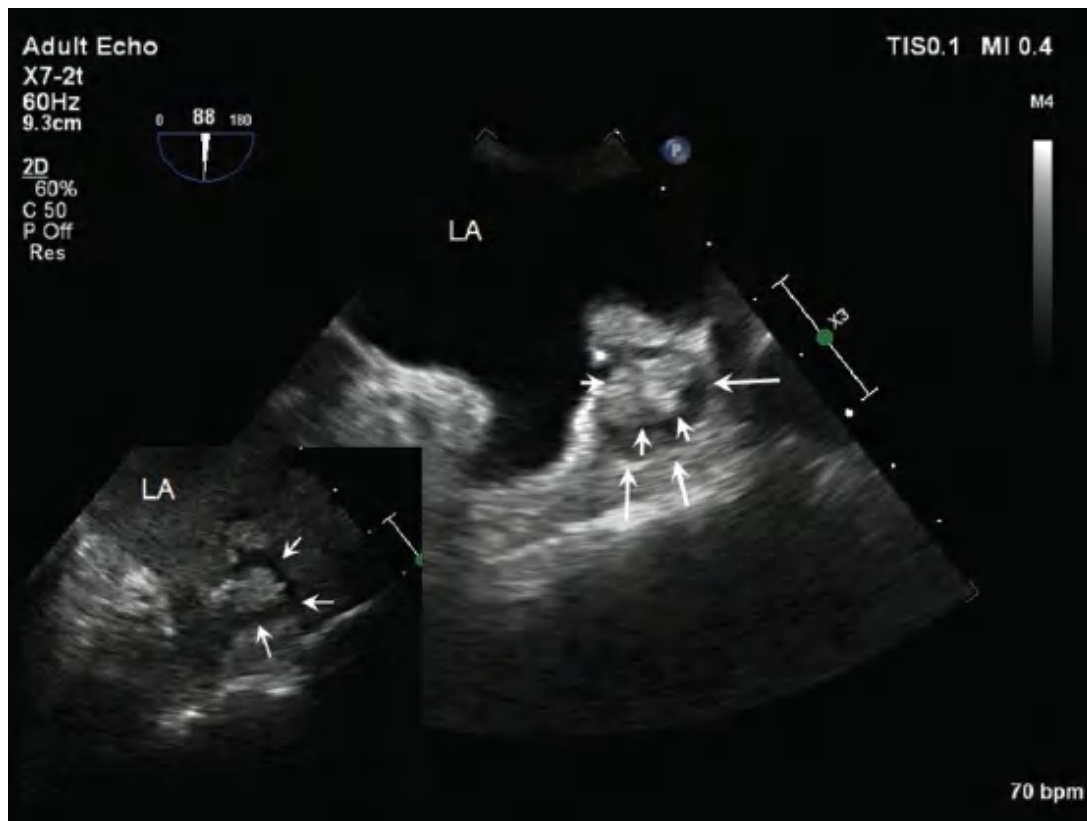
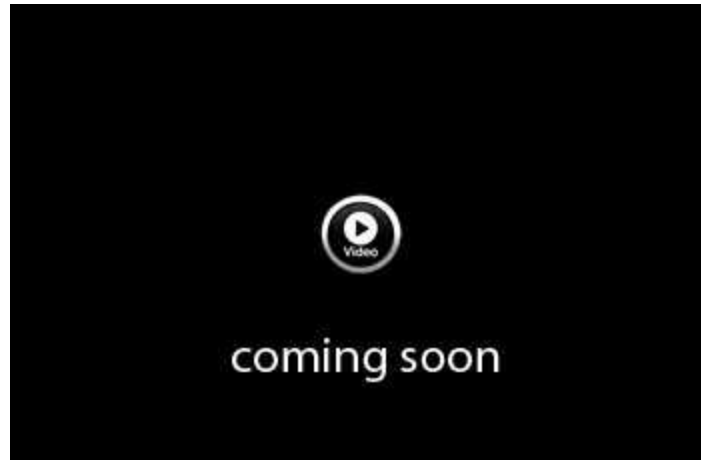
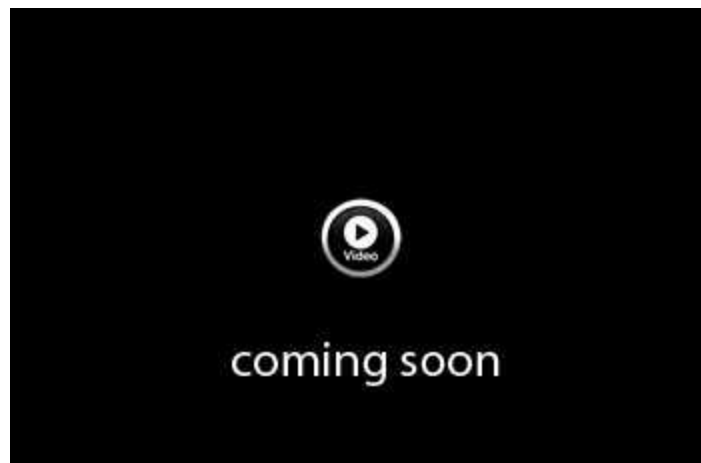


**FIGURE 3.37.** Transesophageal echocardiogram recorded in a patient being evaluated for possible left atrial thrombus prior to catheter ablation for atrial

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**

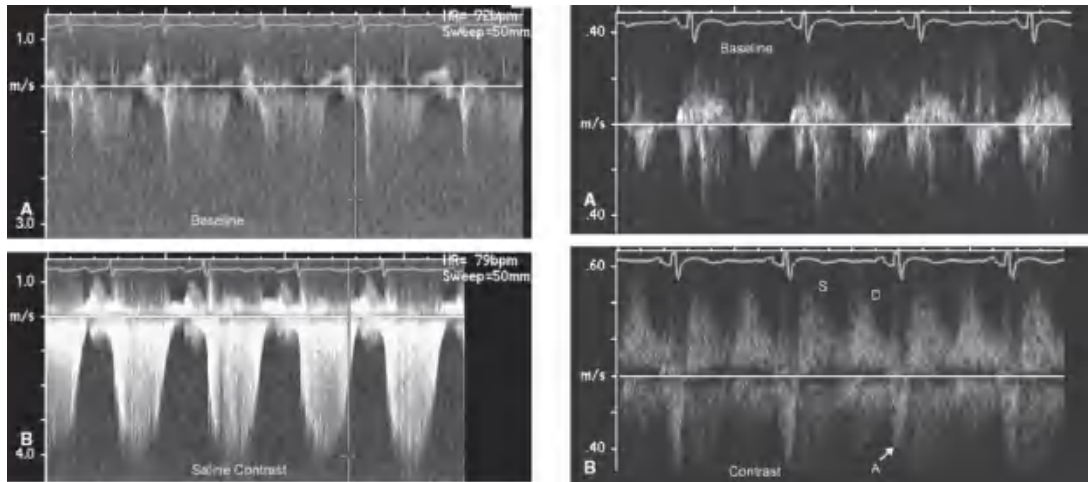


**FIGURE 3.38.** Contrast enhancement of a faint tricuspid regurgitation jet by agitated saline injected into an upper extremity vein. **A:** In the spectral images, note the faint tricuspid regurgitation signal from which it is not possible to ascertain the complete spectral profile or maximal velocity. **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.

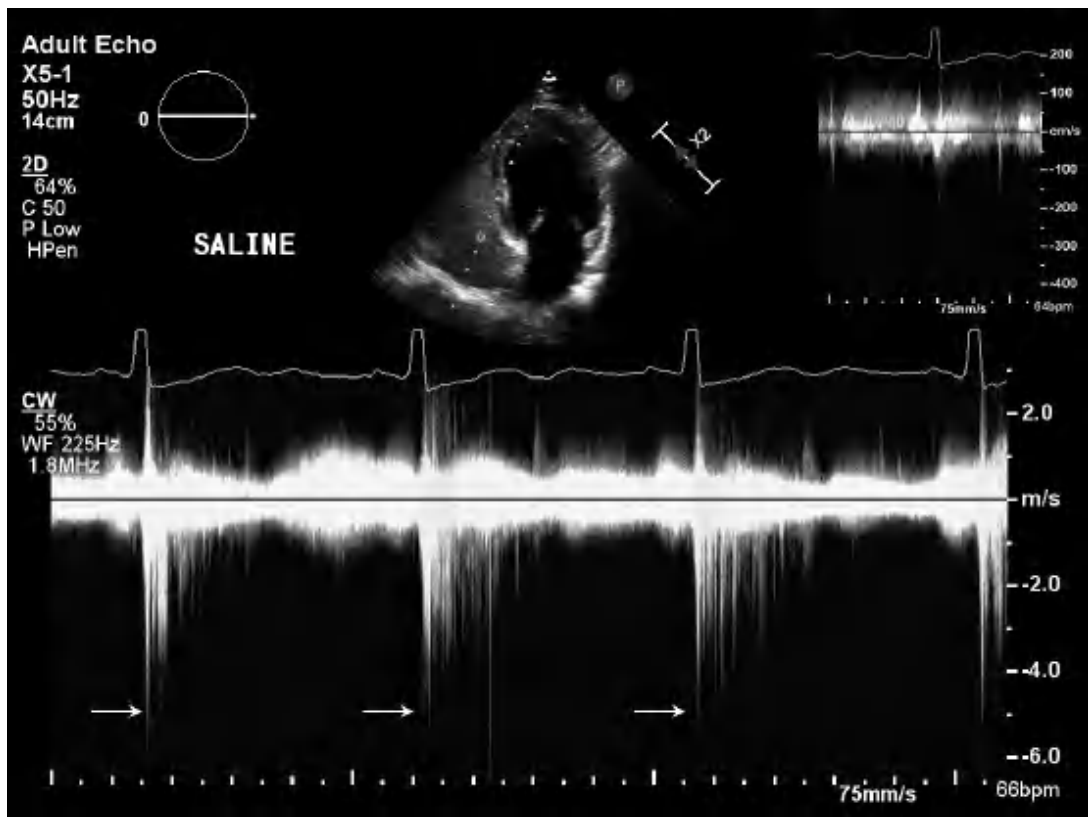


**FIGURE 3.39.** Example of erroneous Doppler signals which are seen following injection of saline contrast. Orientation of the Doppler beam is as noted in the four-chamber view at the top of the figure. Note the numerous high-velocity

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.

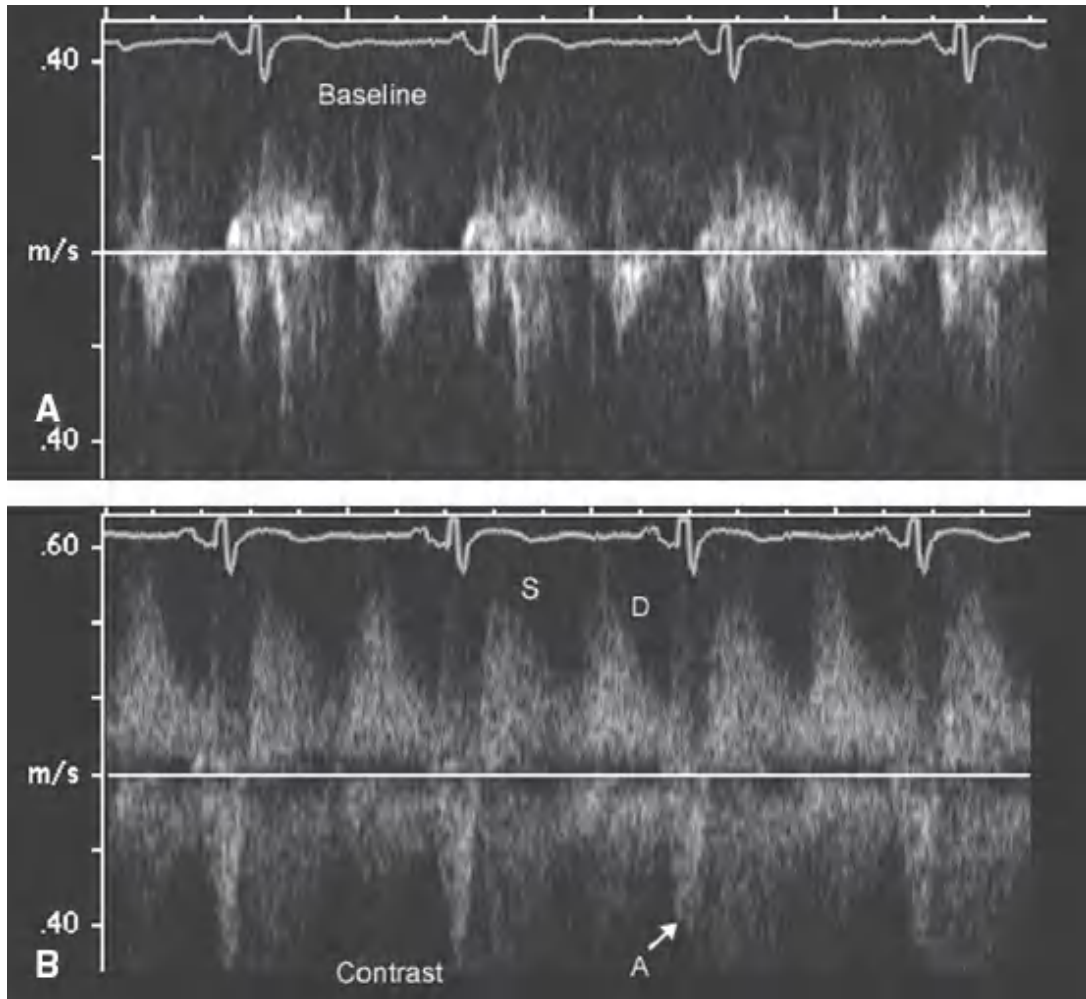


**FIGURE 3.40.** Example of enhancement of pulmonary vein spectral Doppler imaging with intravenous contrast. The spectral signals (**A**) were recorded from an apical view. Note the very poorly defined pulmonary vein inflow signal. **B**: Image recorded in the same patient after injection of a perfluorocarbon-based 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.



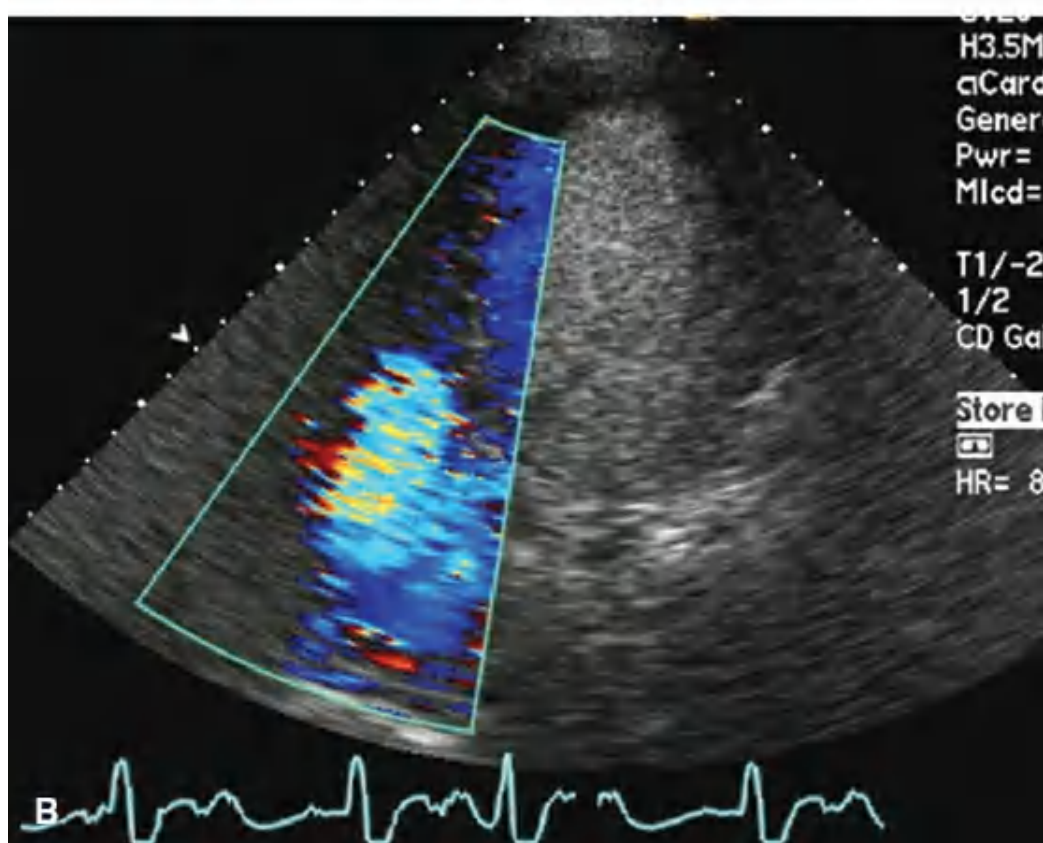
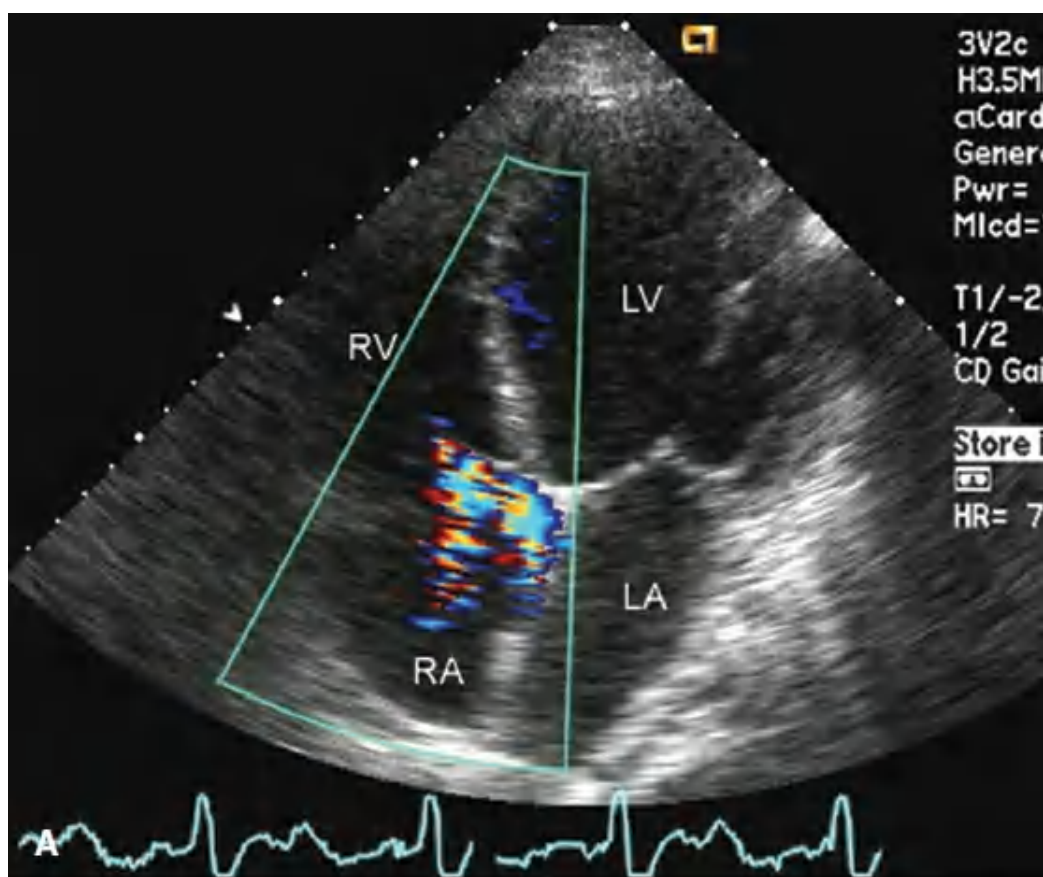
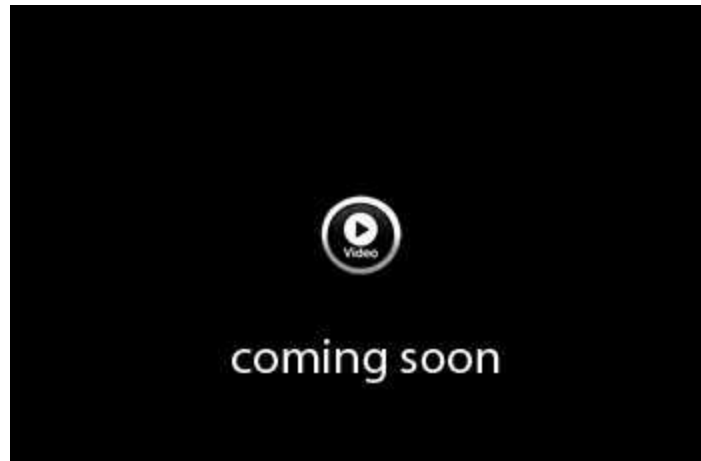
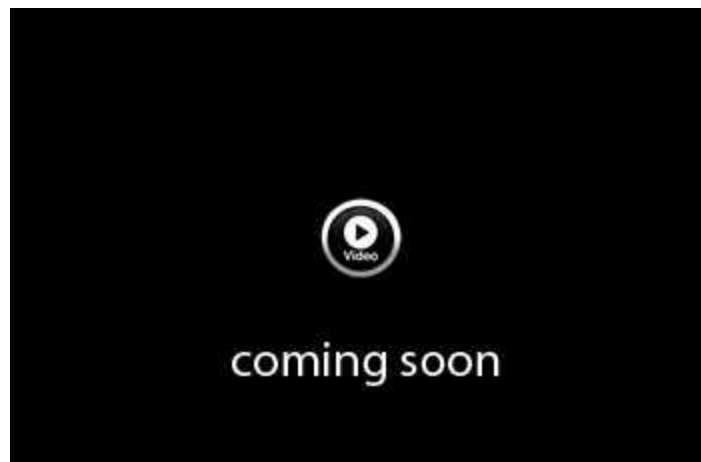




**FIGURE 3.41.** Apical four-chamber view recorded in a patient with mild tricuspid regurgitation before (**A**) and after (**B**) injection of a perfluorocarbon-based contrast agent. **A:** Note the relatively disorganized tricuspid regurgitation jet consistent with mild regurgitation. **B:** Note the dramatic increase in the size and intensity of the color flow signal jet when intracavitary contrast is present. 



**Video 3-41a**



**Video 3-41b**

**Table 3.2**

## CONTRAST ARTIFACTS

Agent/ultrasound related

Attenuation

Shadowing

Apical destruction

Physiologic  
Competitive flow  
SVC-IVC  
Marginated flow  
Incomplete blood pool mixing  
Eustachian valve

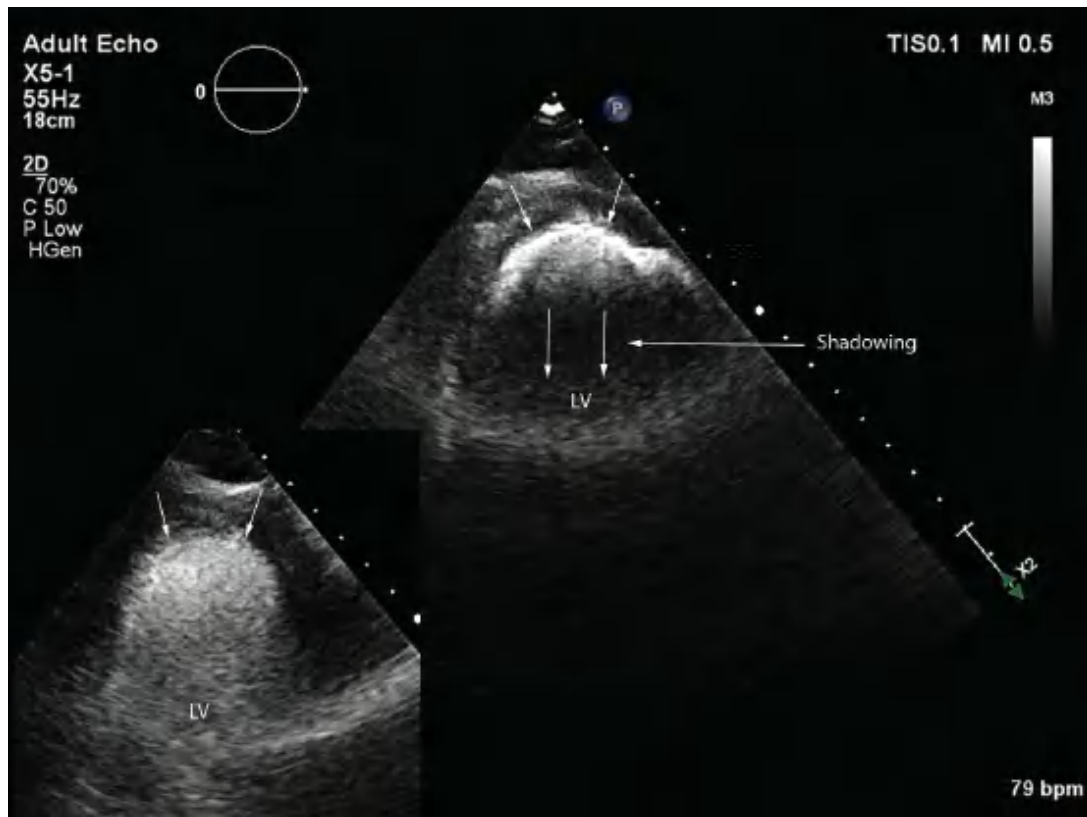
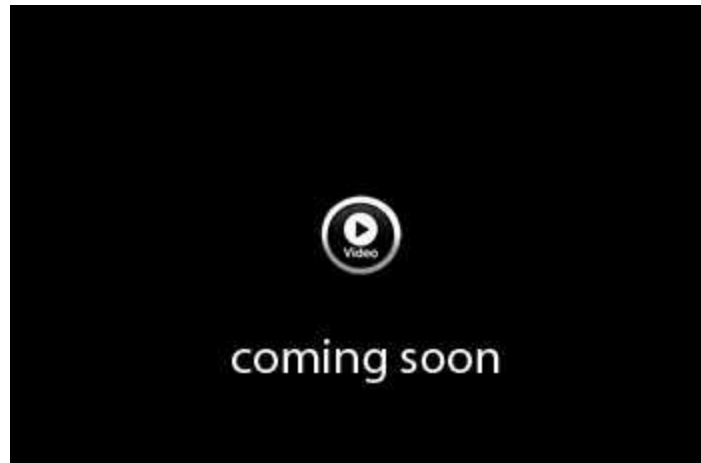
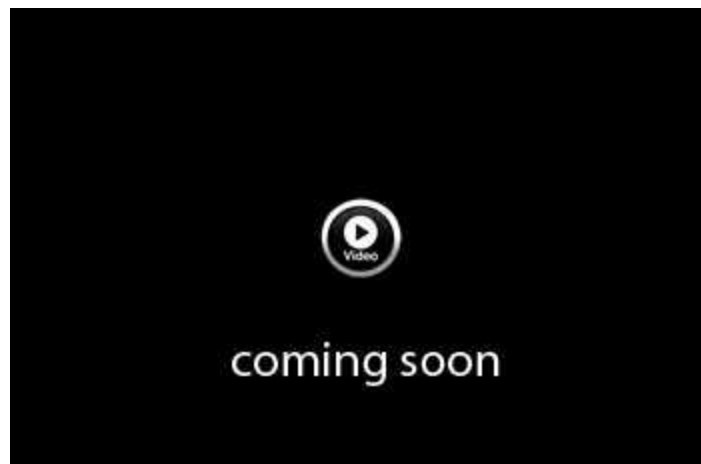


**FIGURE 3.42.** Example of an attenuation effect following injection of intravenous contrast. The central image is an apical four-chamber view recorded shortly after appearance of contrast in the left ventricle. Note the dense contrast effect at the apex of the left ventricle (*downward-pointing arrows*) 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. 



**Video 3-42a**



**Video 3-42b**

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.

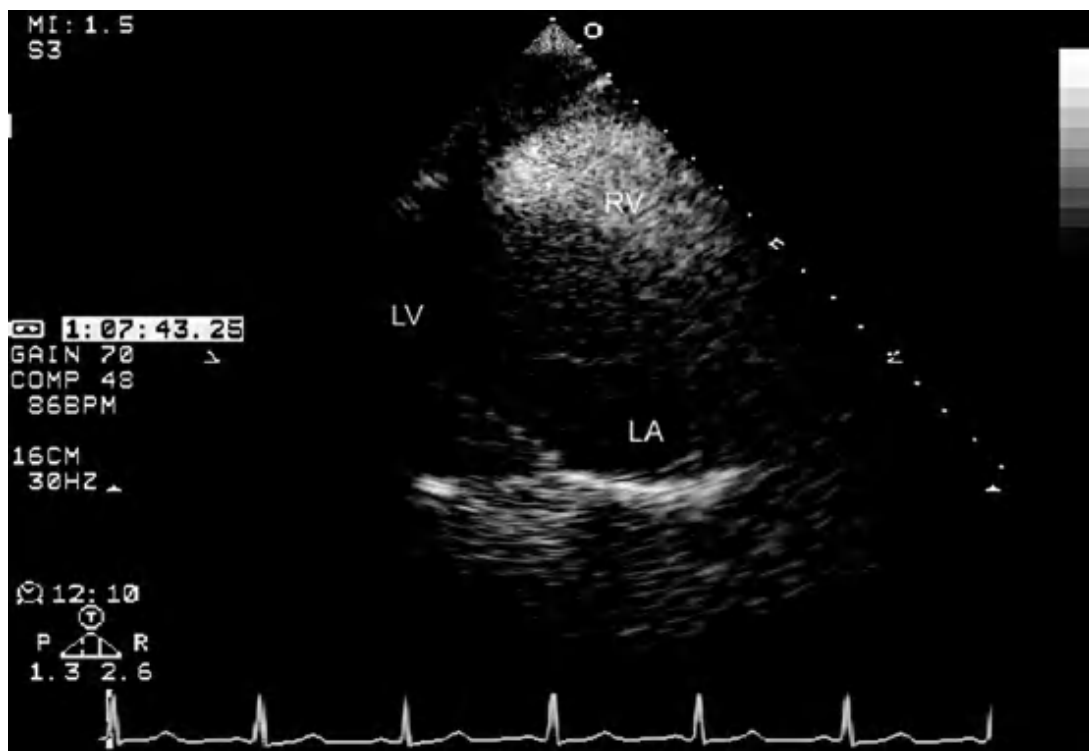


**FIGURE 3.43.** Parasternal long-axis view recorded immediately after injection of ultrasound contrast. Note the significant attenuation of ultrasound signal behind the dense bolus of contrast in the right ventricular outflow tract, which precludes visualization of any posterior structures. 



**Video 3-43**

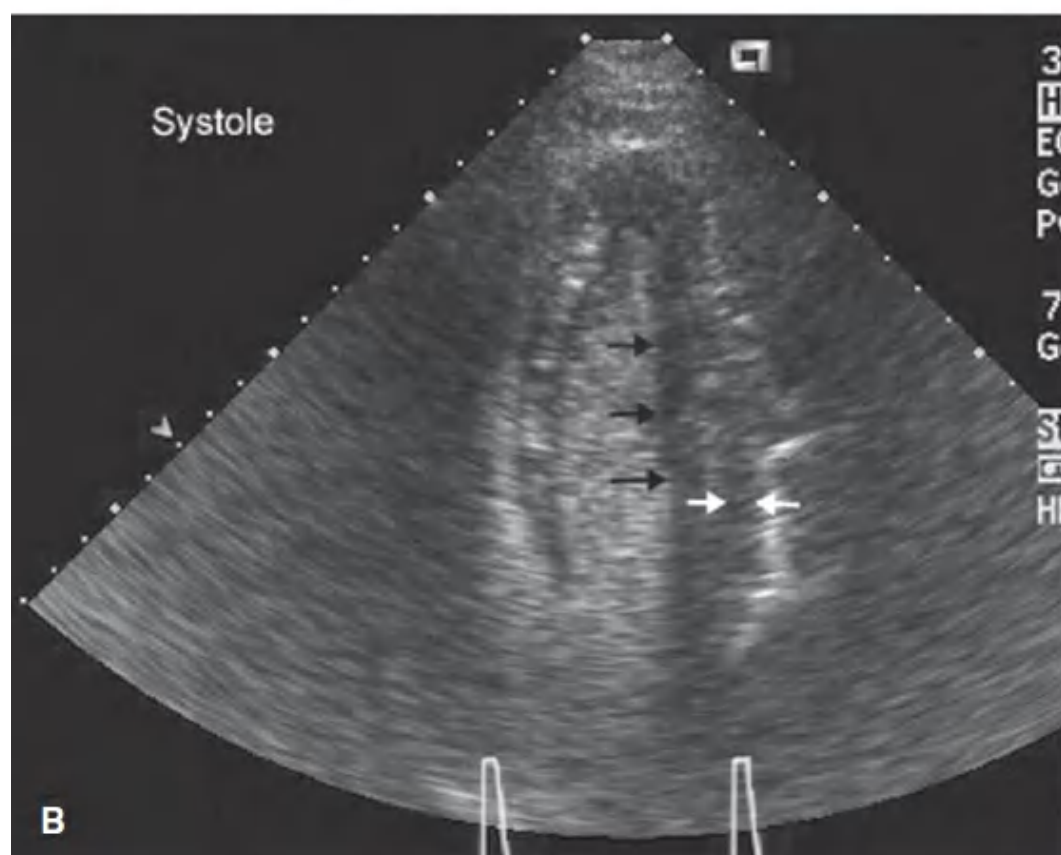
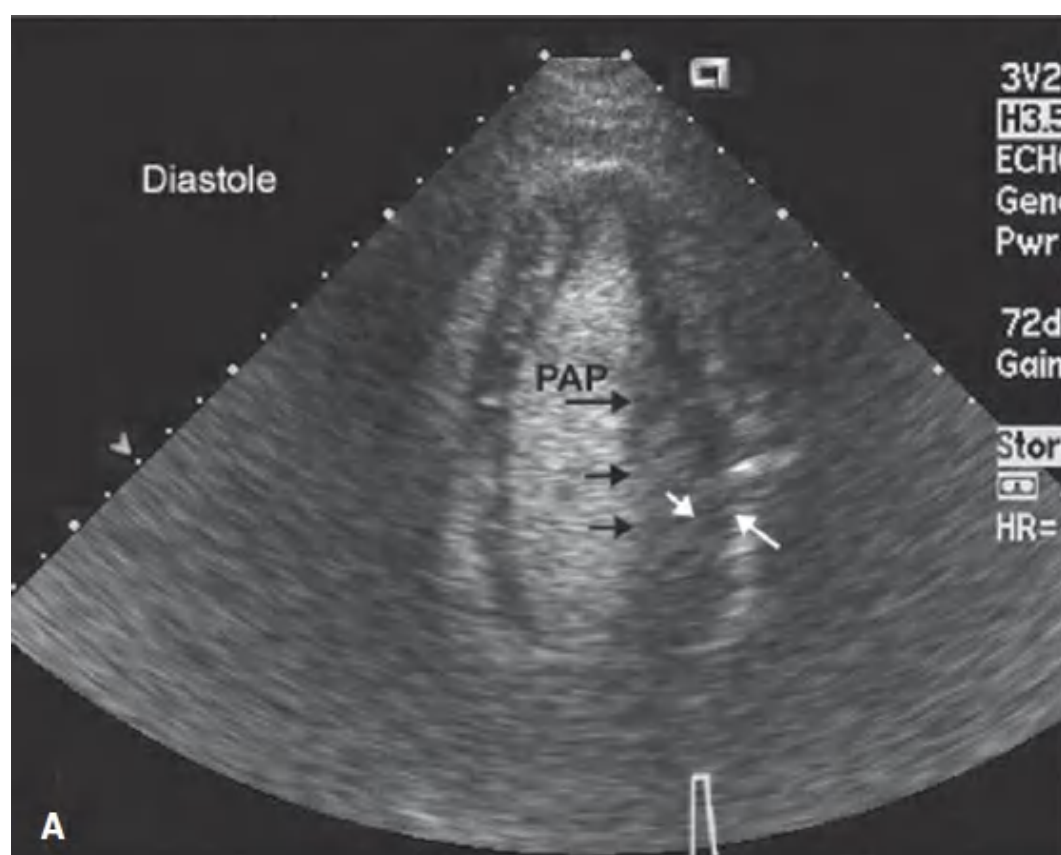
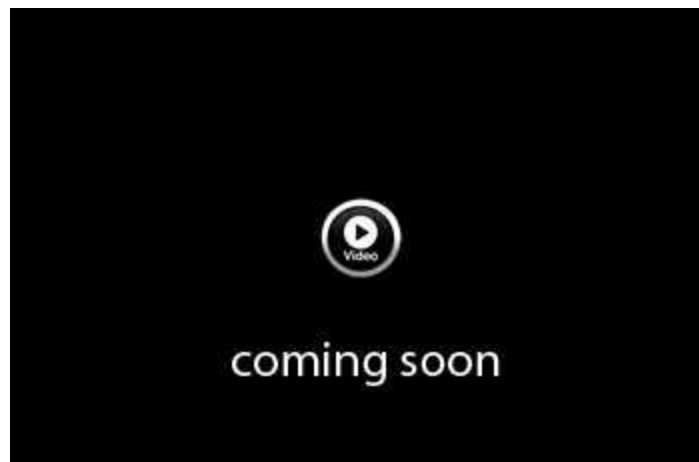


**FIGURE 3.44.** Apical four-chamber view demonstrates a papillary muscle shadow. **A:** Image was recorded in diastole. Note the location of the papillary muscle (*black arrows*) and the faint shadow behind it. Also note the true location and thickness of the lateral wall (*white arrows*). **B:** Image 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**

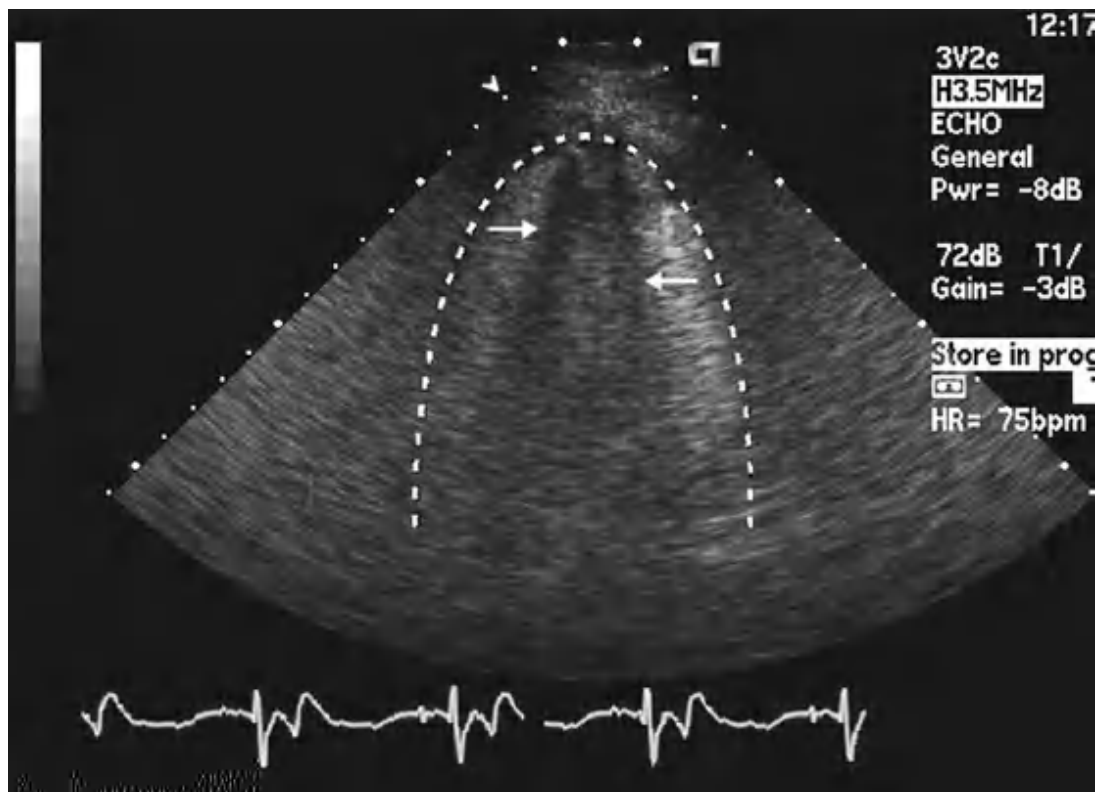
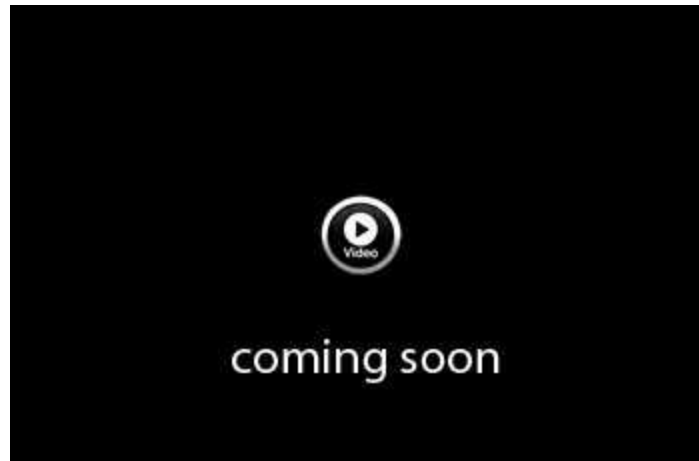


**FIGURE 3.45.** Apical four-chamber view recorded after intravenous injection of a perfluorocarbon-based contrast agent in a patient with an apical aneurysm and focal calcification in the apex. Note the two distinct shadows (*arrows*) arising from the apex in the otherwise smooth homogeneous filling of the left ventricular cavity. The *dotted lines* represent the true cavity boundary. 



**Video 3-45**

## **MYOCARDIAL PERFUSION CONTRAST ECHOCARDIOGRAPHY**

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.

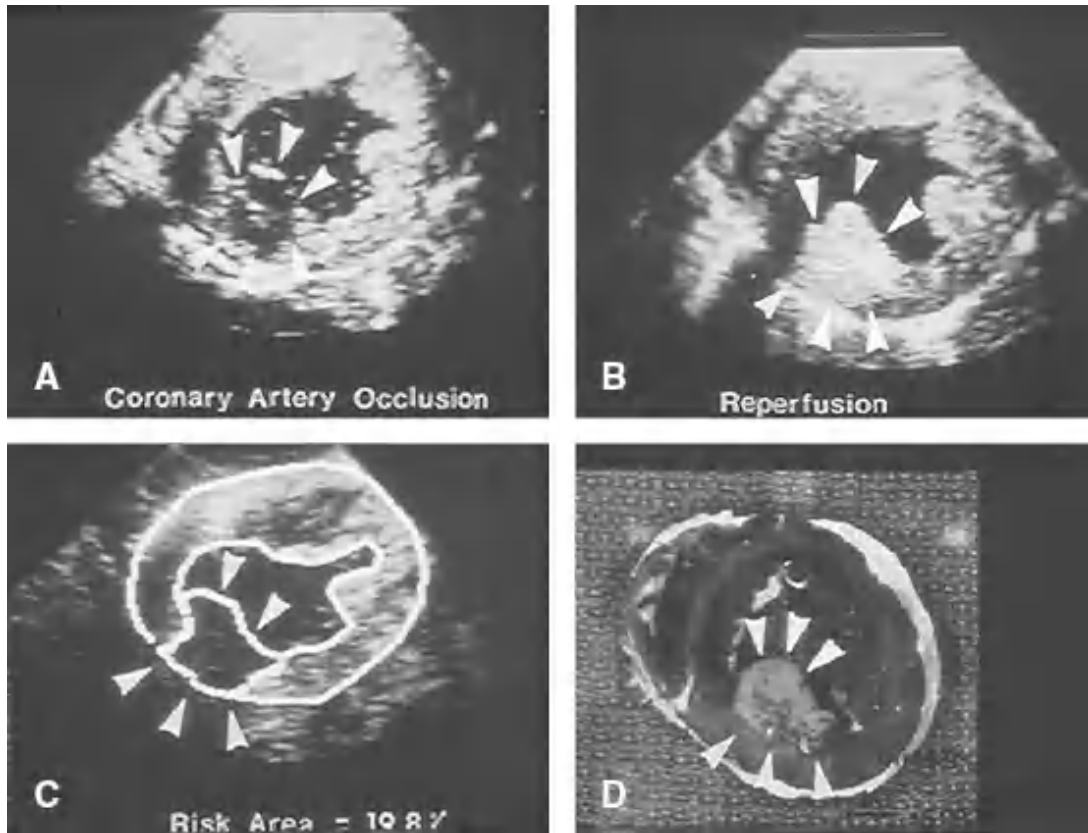


**FIGURE 3.46.** Contrast echocardiogram demonstrates myocardial contrast effect in an animal model of acute myocardial infarction. **A:** A short-axis image immediately after occlusion of a coronary artery. Note the opacification of the majority of the myocardium and the absence of contrast effect in 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.

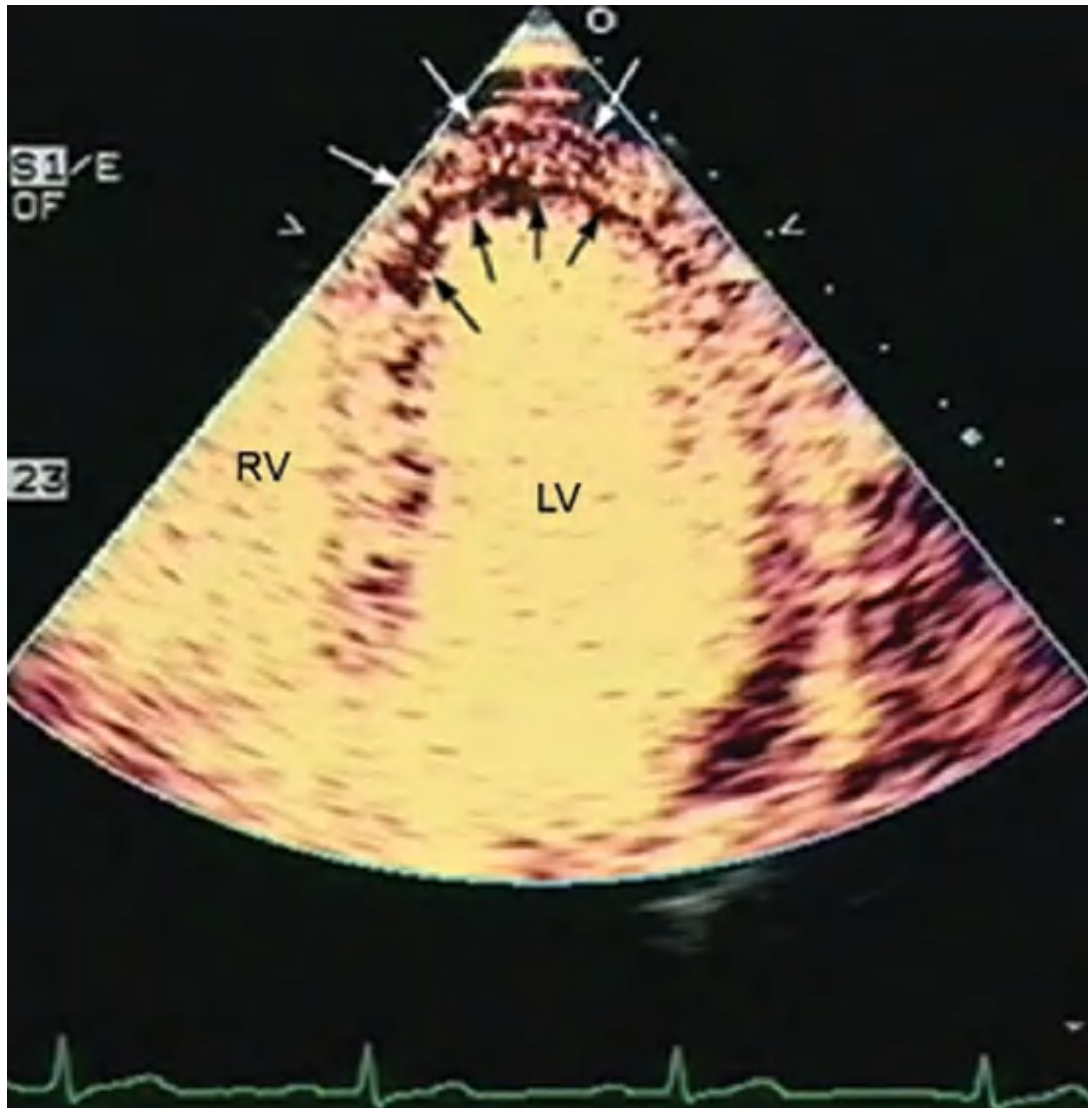


**FIGURE 3.47.** Myocardial contrast echocardiogram performed using a continuous infusion in continuous low mechanical index imaging. This frame was recorded approximately 20 cardiac cycles after a burst phase and reveals the absence of contrast effect in the apical myocardium (*arrows*) and a robust 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.

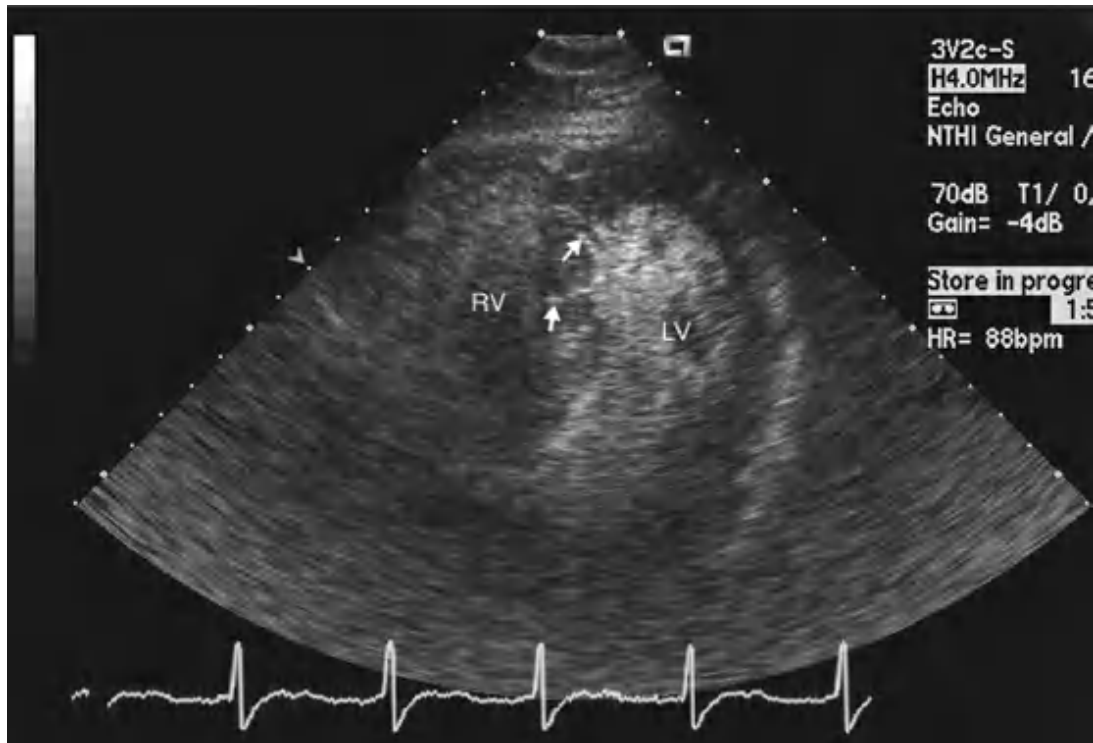


**FIGURE 3.48.** Apical two-dimensional echocardiogram recorded after injection of a perfluorocarbon-based contrast agent. Note the opacification of intramural vessels within the ventricular septum (*small arrows*).

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](#)).

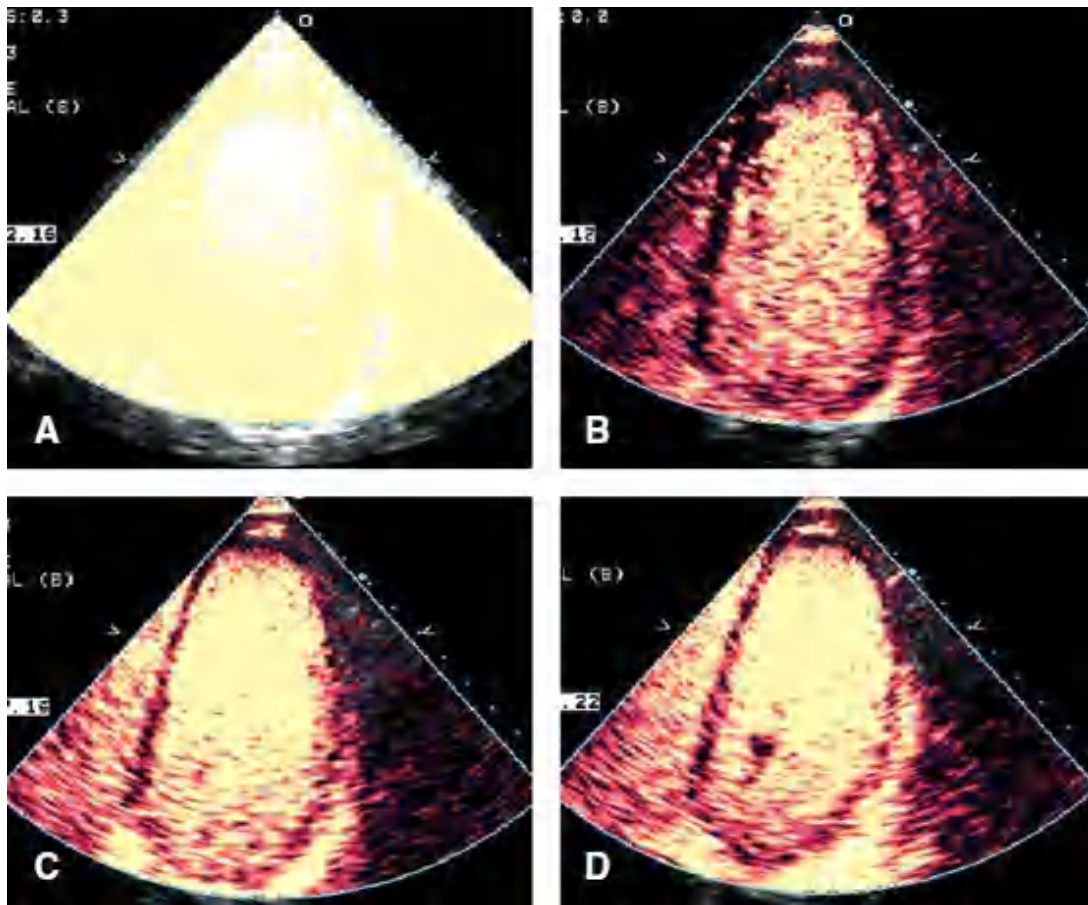
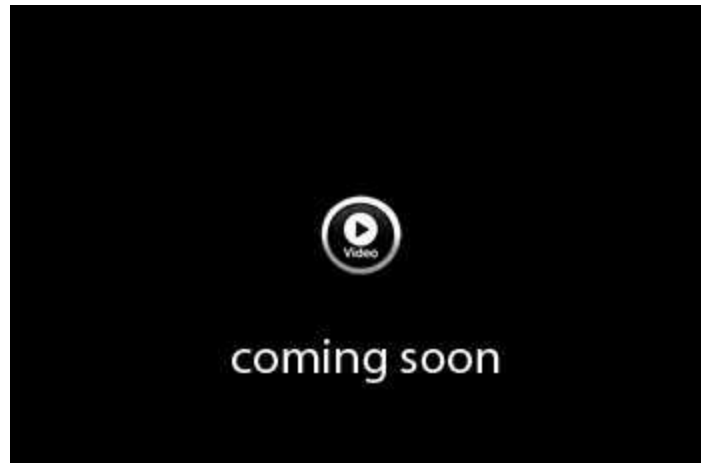


**FIGURE 3.49.** Apical four-chamber view recorded after intravenous injection of a perfluorocarbon-based contrast agent for the purpose of myocardial perfusion echocardiography. **A:** Image was recorded at the time of a high mechanical index “burst.” **B:** Image was recorded immediately after the 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. 





**Video 3-49**

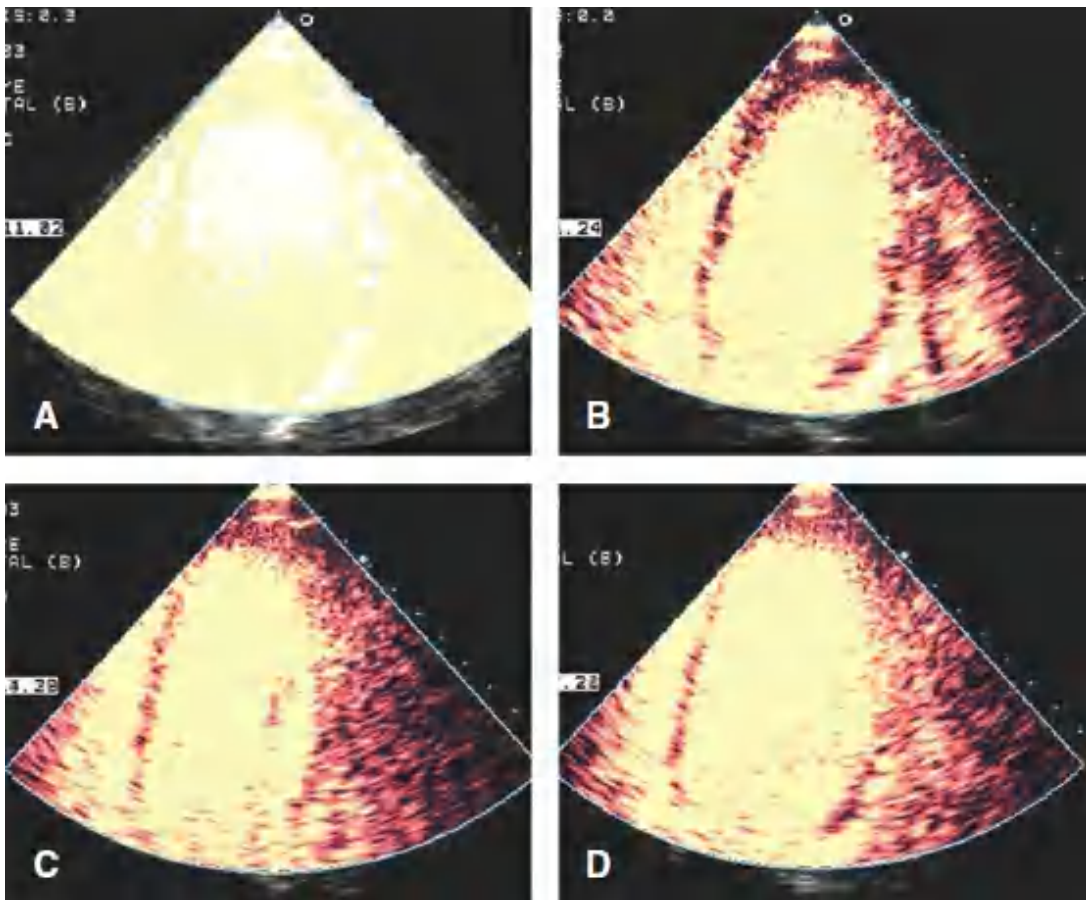
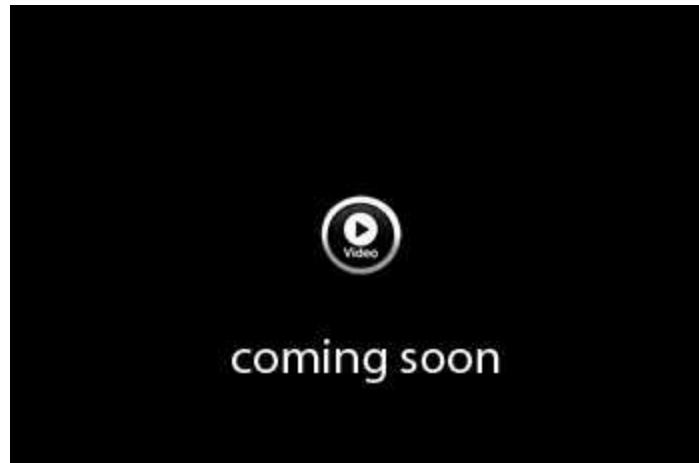


**FIGURE 3.50.** Apical four-chamber view recorded in the same patient depicted in [Figure 3.39](#) at the time of hyperemia due to dipyridamole infusion. The format and timing are identical to those for [Figure 4.44](#). In the presence of a hyperemic state, note the increase in the contrast effect in the ventricular myocardium and the more rapid development of significant contrast effect in the ventricular myocardium. 📺



### Video 3-50

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  $\alpha$ ,  $\beta$ , 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.

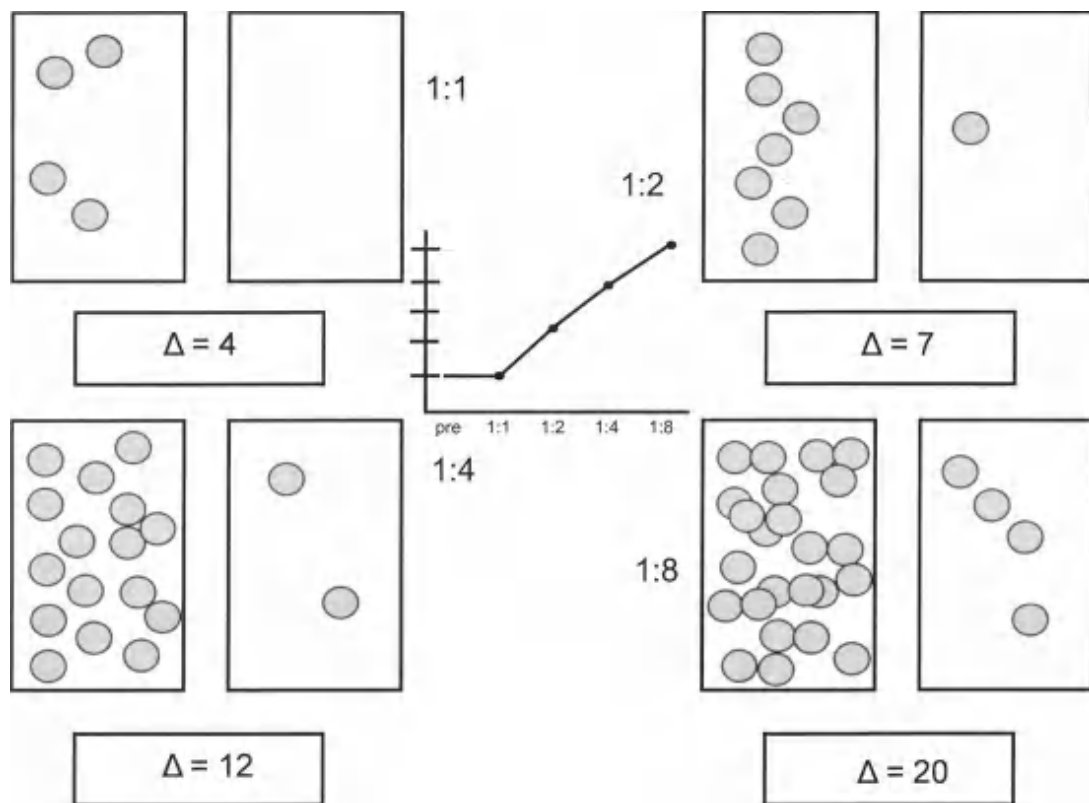


**FIGURE 3.51.** Graphic demonstration of the intermittent imaging technique for creating a time intensity curve. Four pairs of images are presented. For each, the schematic on the left represents the amount of contrast before imaging and on the right the amount of contrast after contrast imaging. In 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.

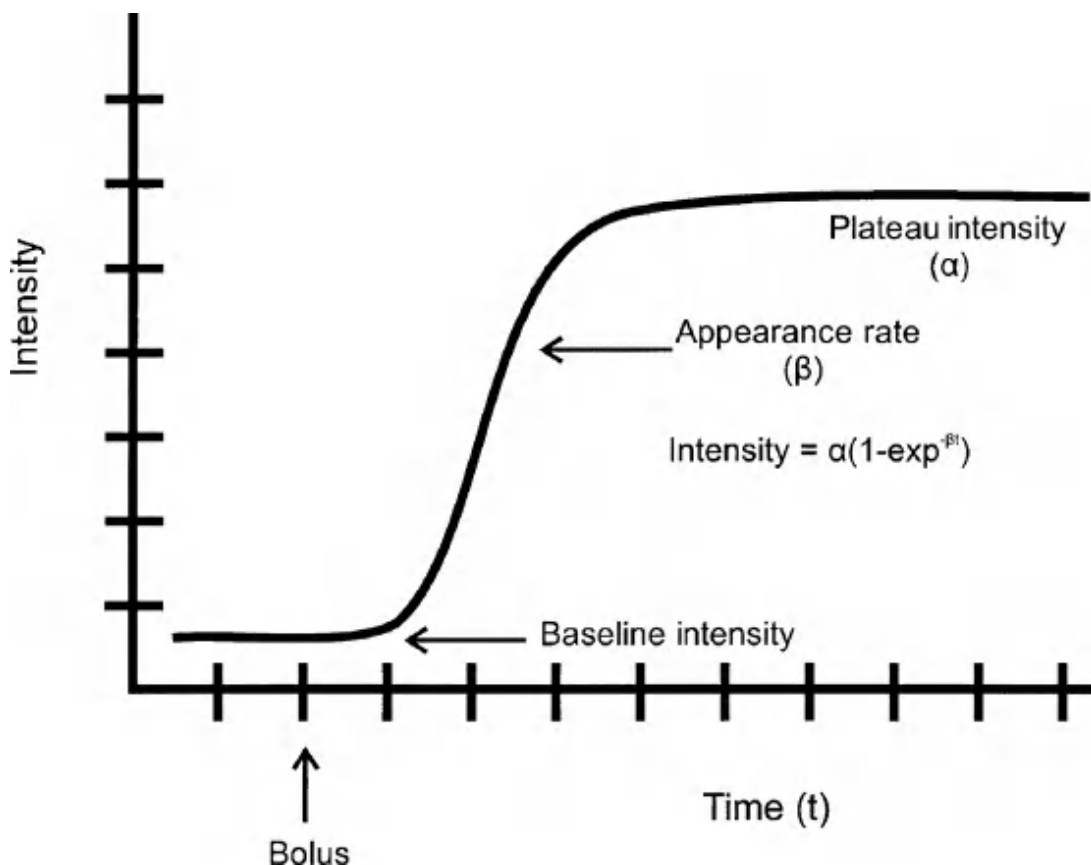


**FIGURE 3.52.** Stylized time appearance curve of contrast within the ventricular myocardium depicting the different parameters of a contrast appearance curve. See text for details.

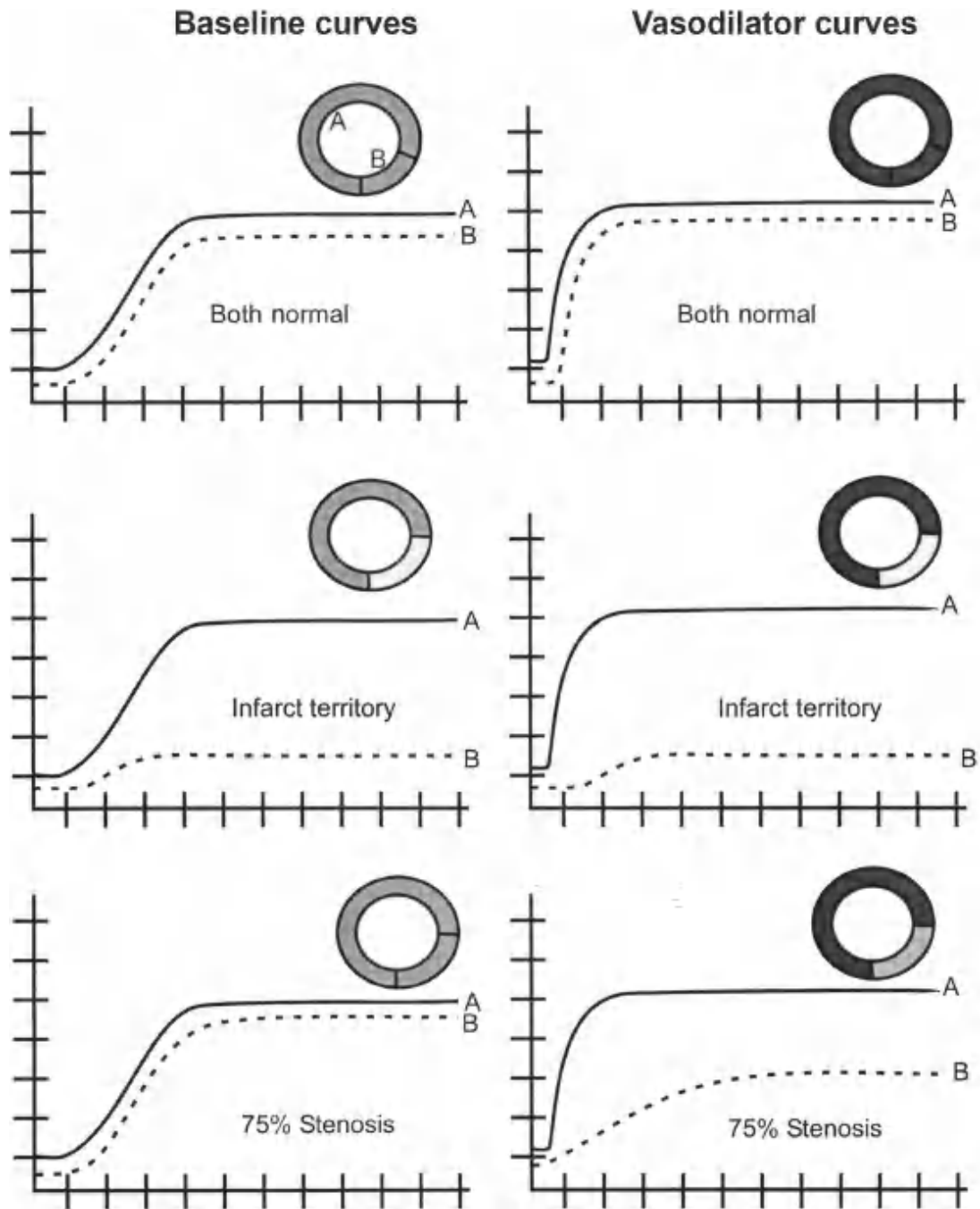


**FIGURE 3.53.** Stylized time appearance curves depict normal coronary flow and different disease states. The curves on the left are all depictions of baseline appearance curves and those on the right are the anticipated appearance curves at the time of vasodilator stress. Two separate coronary territories 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 ( $\alpha$ ) equivalent to that seen at baseline, but the rate of increase of contrast effect ( $\beta$ ) 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 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.

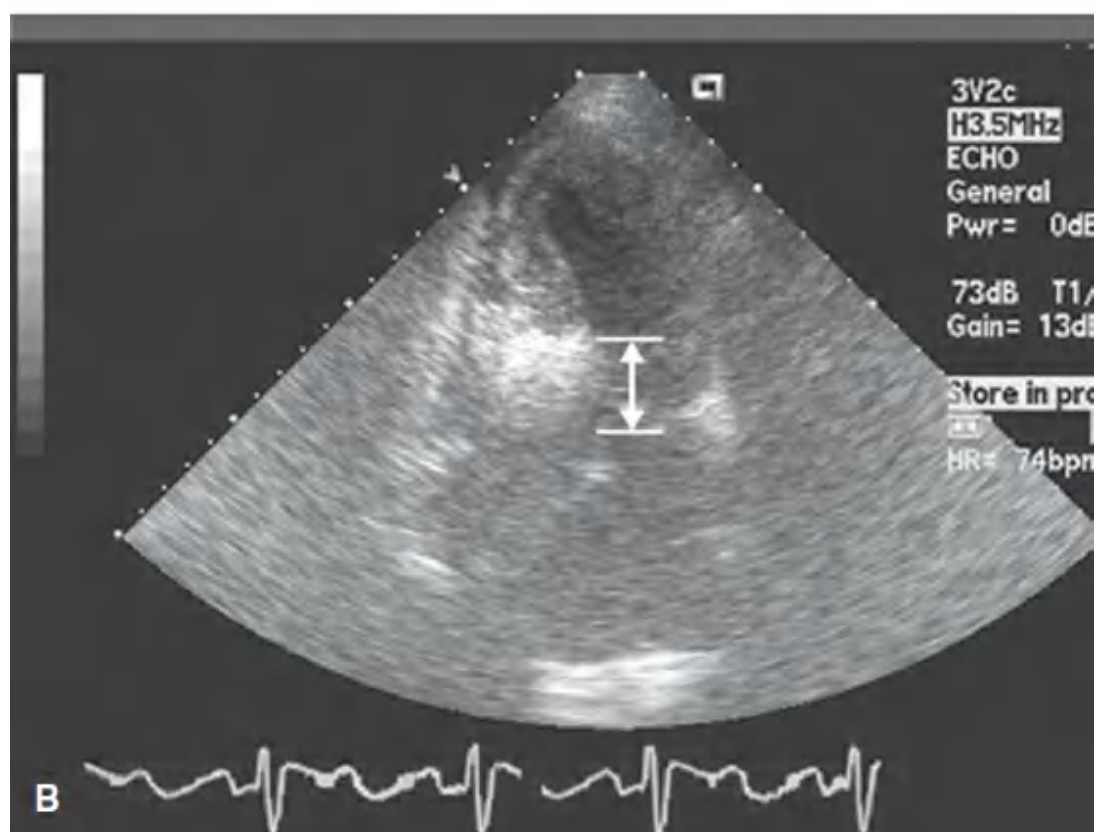
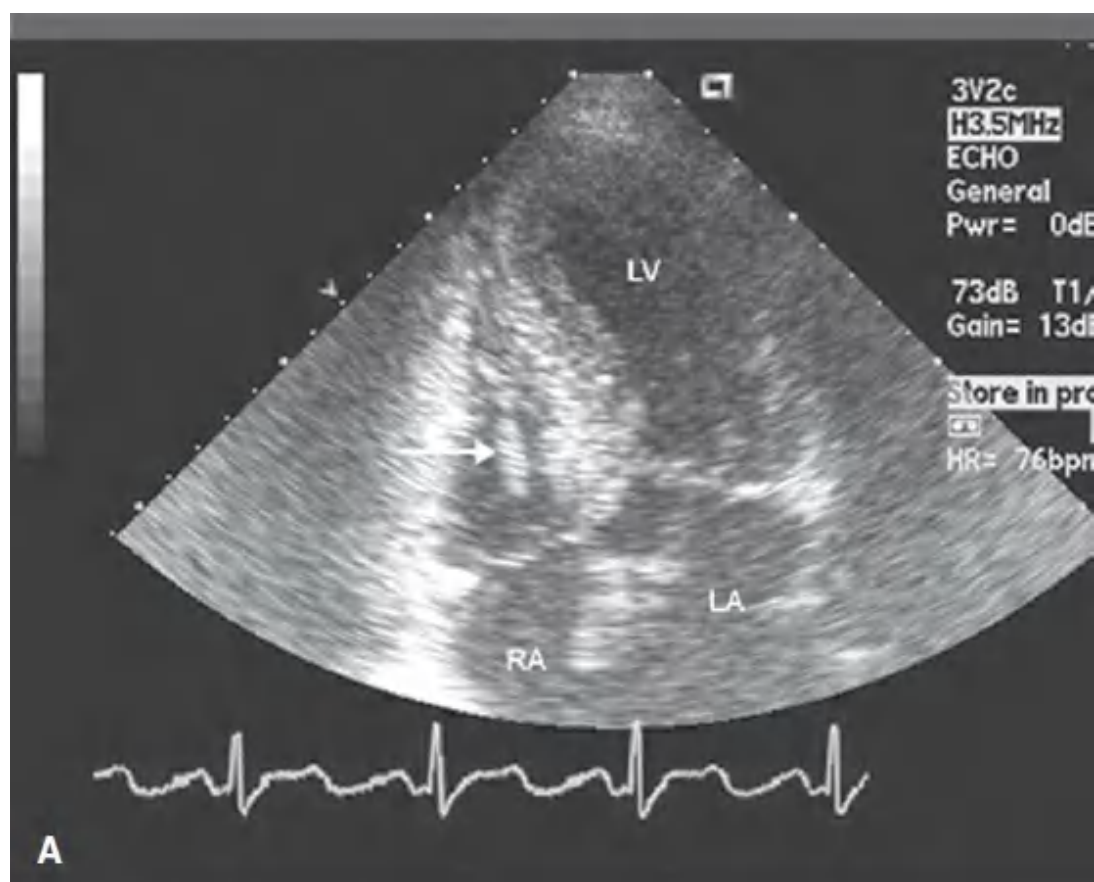
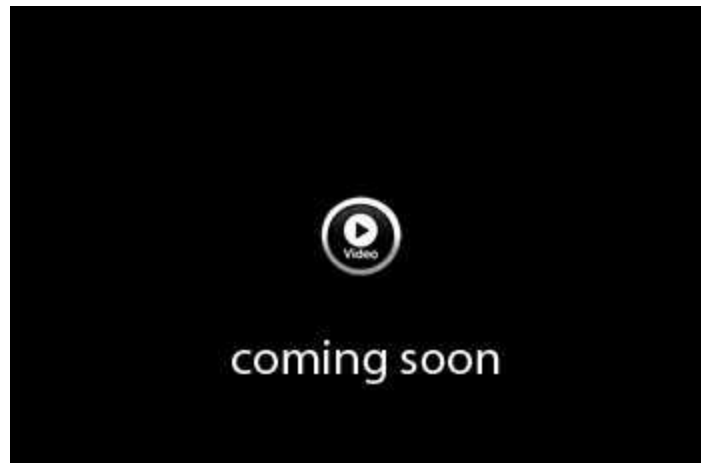
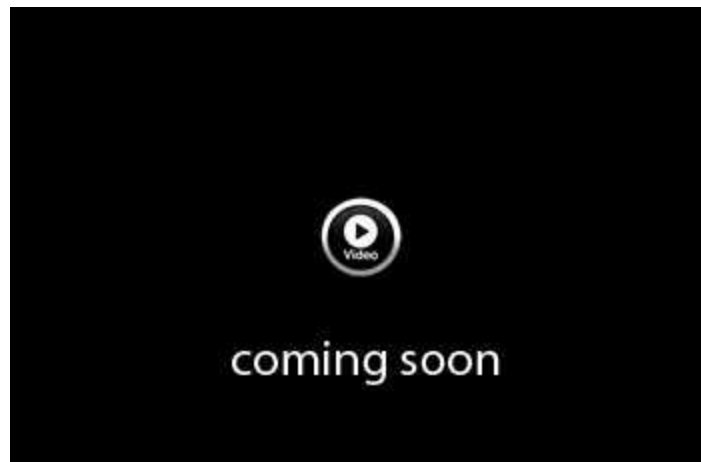


**FIGURE 3.54.** Apical four-chamber view recorded in a patient with a hypertrophic cardiomyopathy undergoing alcohol septal reduction therapy. **A:** Image recorded under basal conditions. There is a pacemaker catheter in the right ventricle (*arrow*) and systolic anterior motion of the mitral valve. 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. 📺



**Video 3-54a**



**Video 3-54b**

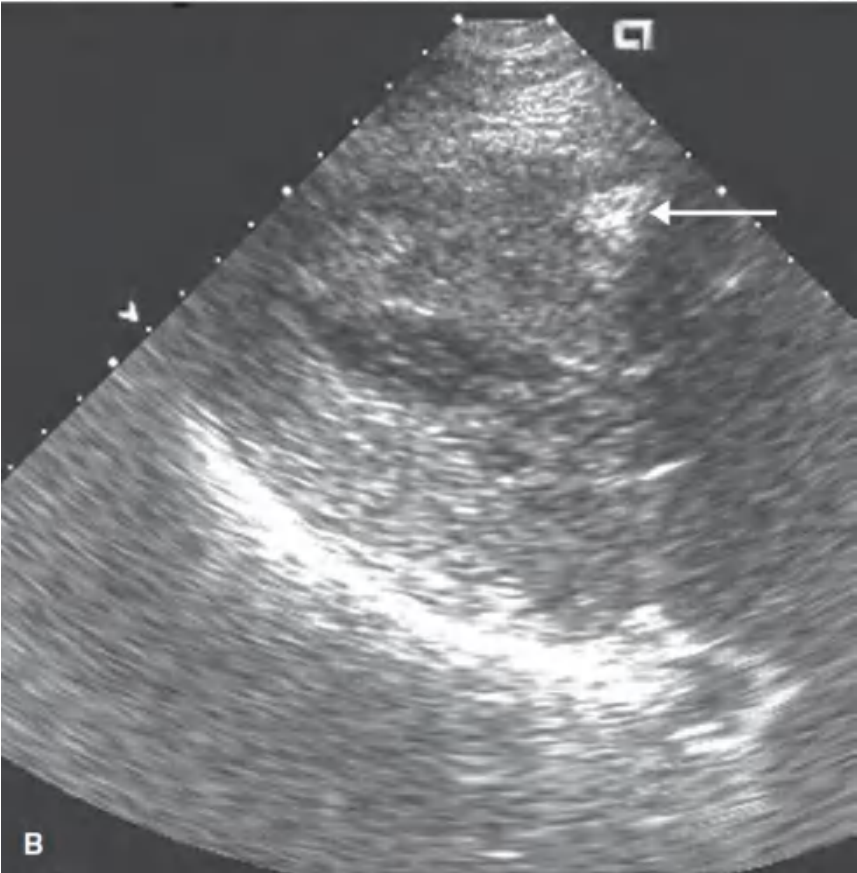
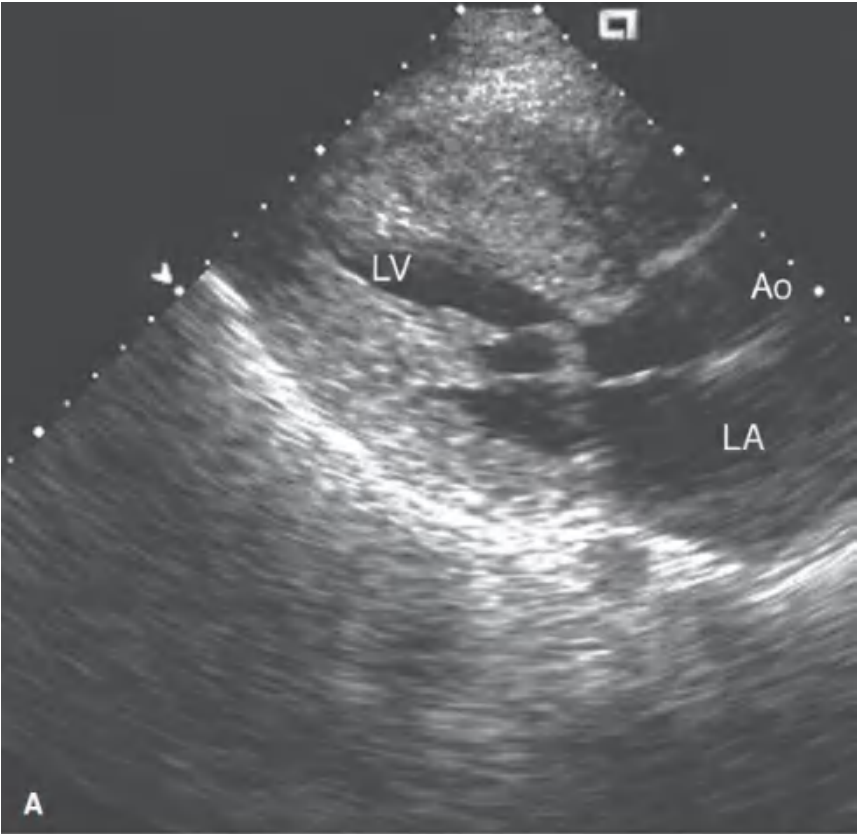
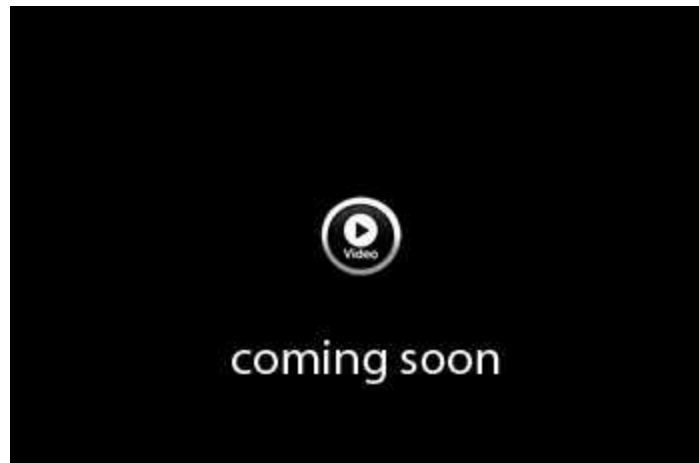




**FIGURE 3.55.** Parasternal long-axis view recorded in a patient with hypertrophic cardiomyopathy being considered for alcohol septal reduction therapy. **A:** Image was recorded at baseline. Note the hypertrophy of the ventricular septum and the systolic anterior motion of the mitral valve. **B:** 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**

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### **MYOCARDIAL PERFUSION CONTRAST ECHOCARDIOGRAPHY**

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## Chapter 4

# The Comprehensive Echocardiographic Examination

Echocardiography is a highly versatile technique that can be applied in variety of 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

**Anatomic imaging domains**

- Single-line interrogation
  - M-mode echocardiography
- Multiple-line interrogation
  - Two-dimensional echocardiography
- Multiple-dimensional imaging
  - Three-dimensional imaging
    - Reconstructed
    - Real-time three-dimensional imaging

**Doppler domains**

- Pulsed Doppler methods
  - Single-interrogation volume
  - Multiple-interrogation volume
  - Saturated-interrogation volume area
    - Color flow imaging
  - M-mode color interrogation

**Continuous wave Doppler**

**Analysis domains**

- Frequency shift
- Power spectrum
- Variance
- Correlation methods
- Tissue velocity imaging
- Strain rate imaging

**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:

- . 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.



**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 4.2**

**TRANSTHORACIC ECHOCARDIOGRAPHIC VIEWS**

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

|                     |                                   |                                  |
|---------------------|-----------------------------------|----------------------------------|
| Two chamber         | Narrow angle of the valves, septa | MR, AR                           |
| Long axis           | 3D color of the valves, septa     | MR, AR                           |
| “Five” chamber      | Zoom on any region of interest    | LV outflow, AR                   |
| <b>Subcostal</b>    |                                   |                                  |
| 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     |                                   |                                  |
| <b>Suprasternal</b> |                                   |                                  |
| 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.

## 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.

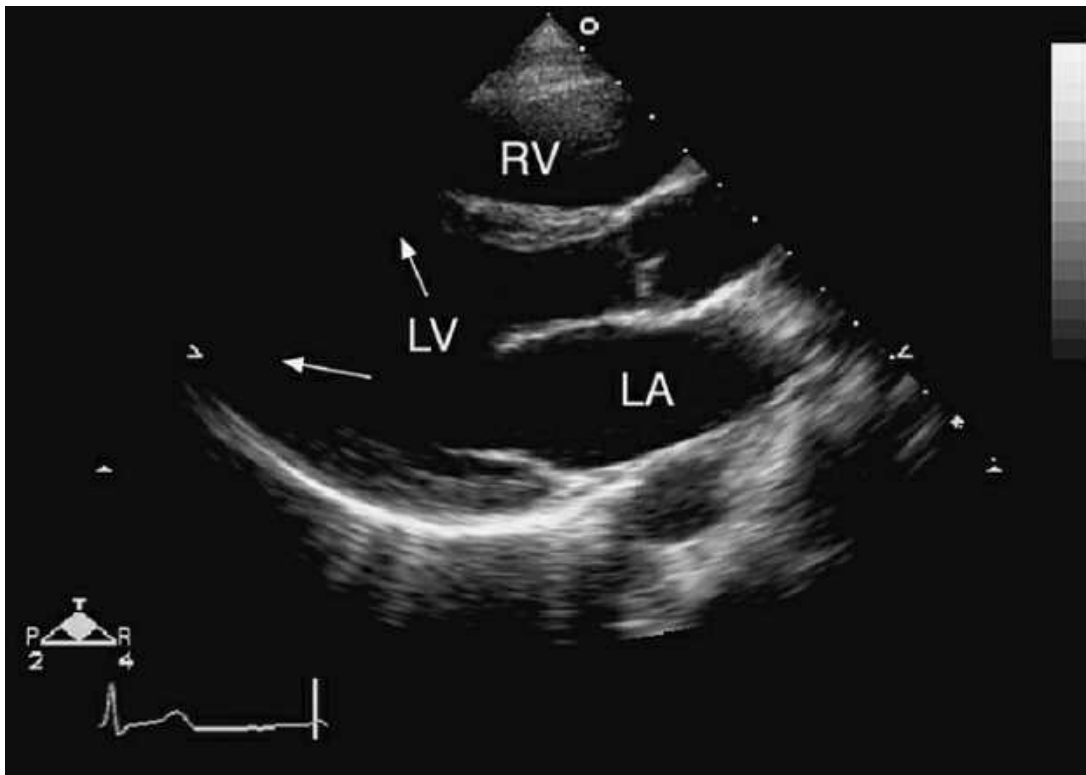
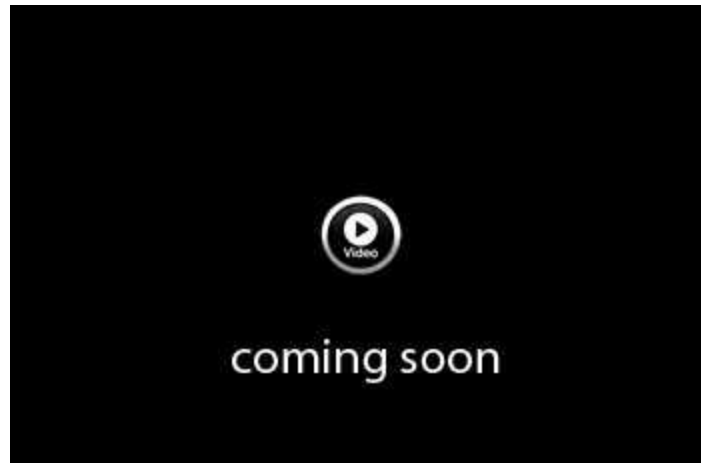


**FIGURE 4.2.** An example of rib shadowing (*arrows*). The presence of the rib relative to the transducer footprint obscures the distal septum and posterior wall of the left ventricle. 📺



### Video 4-2



**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.

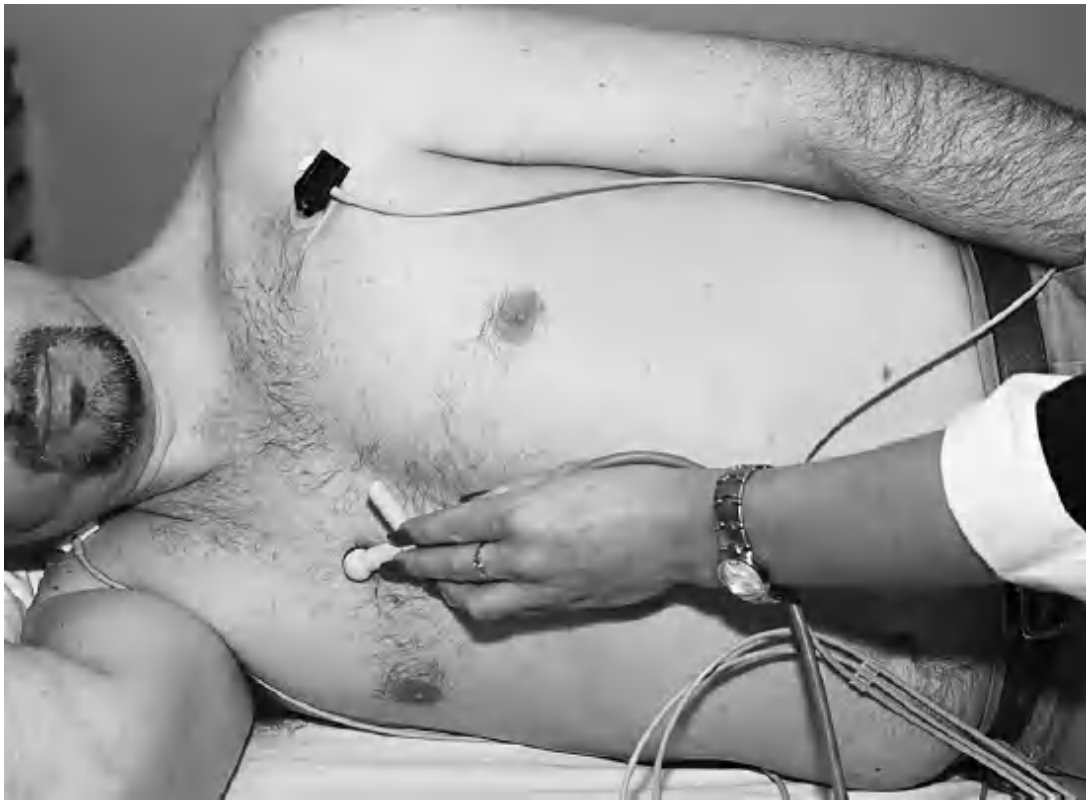


**FIGURE 4.4.** The right lateral decubitus position is shown, and a Pedoff transducer is applied to record ascending aortic flow.

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.

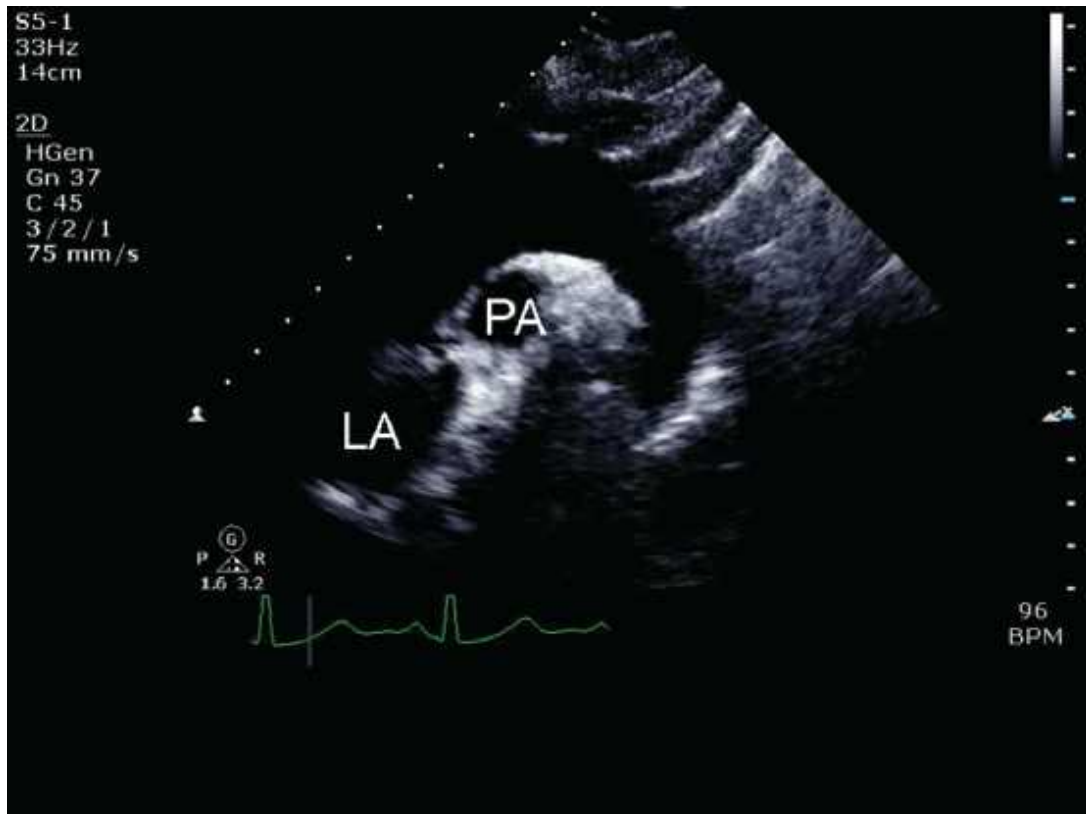
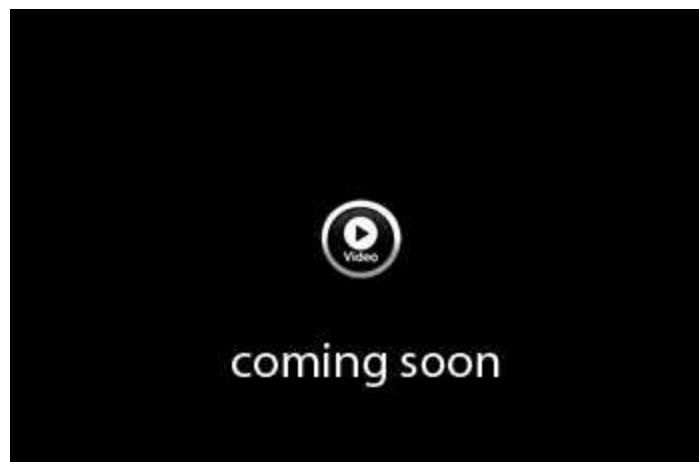


**FIGURE 4.7.** The suprasternal notch provides a unique opportunity to record the aortic arch and the origin of the great vessels. 



## Video 4-7

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.

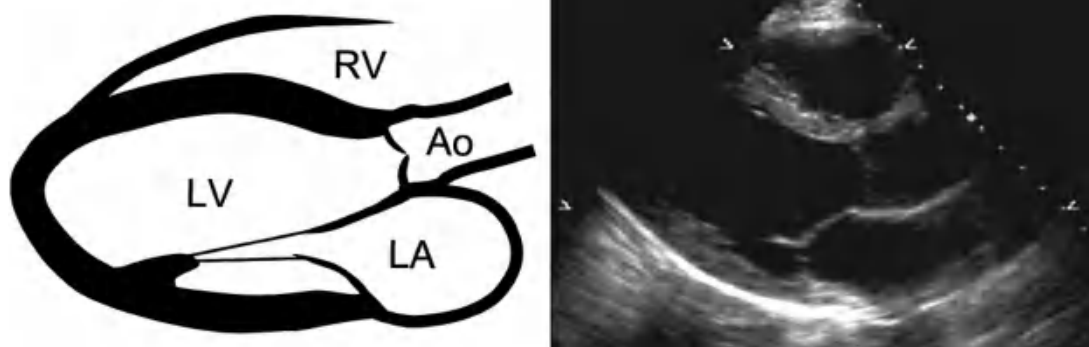
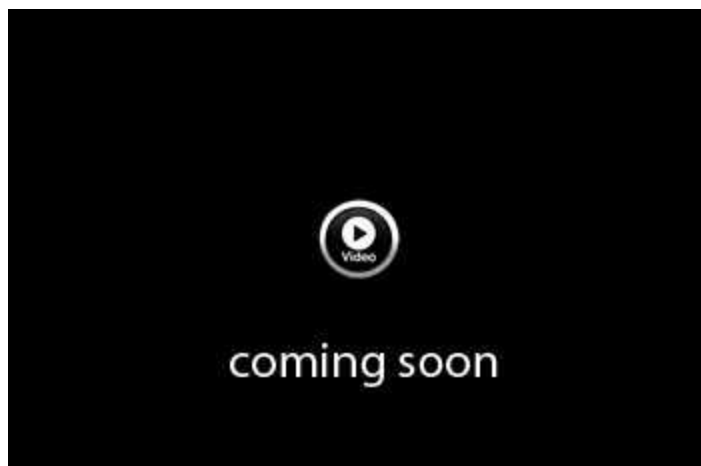


**FIGURE 4.8.** The parasternal long-axis view. 



**Video 4-8**

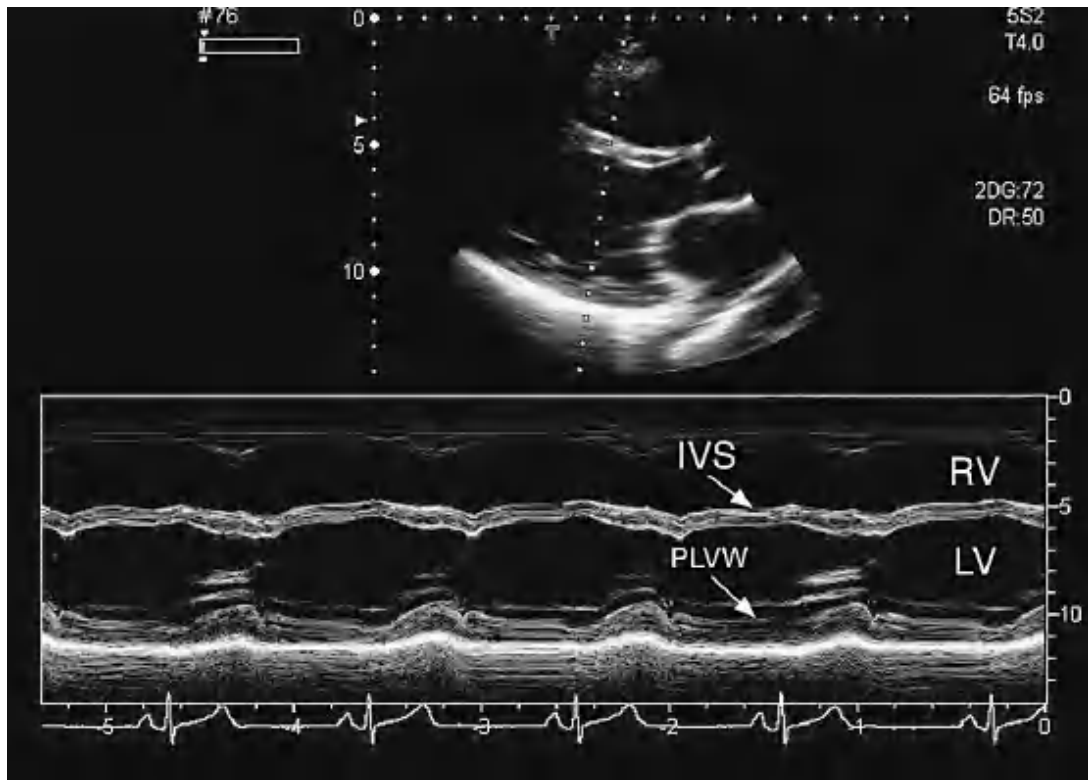


**FIGURE 4.9.** From the two-dimensional image, an M-mode display at the midventricular level is derived. PLVW, posterior left ventricular wall.

## **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.

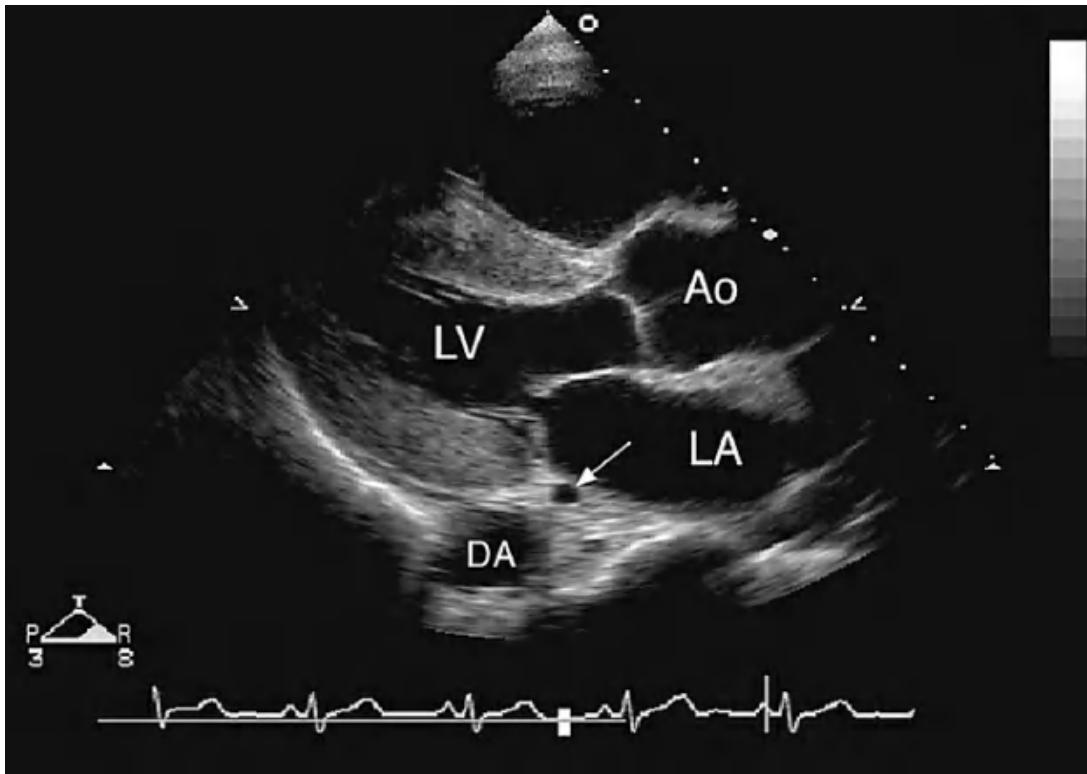
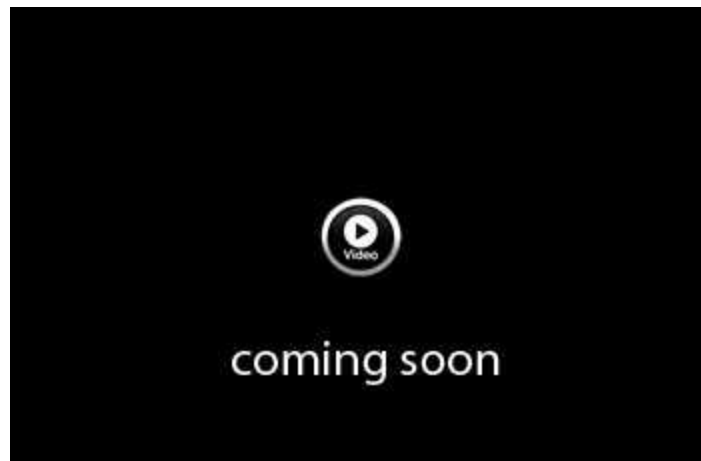


**FIGURE 4.10.** This parasternal long-axis view illustrates the relationship between the coronary sinus (*arrow*) and the descending aorta (DA). 📺



**Video 4-10**

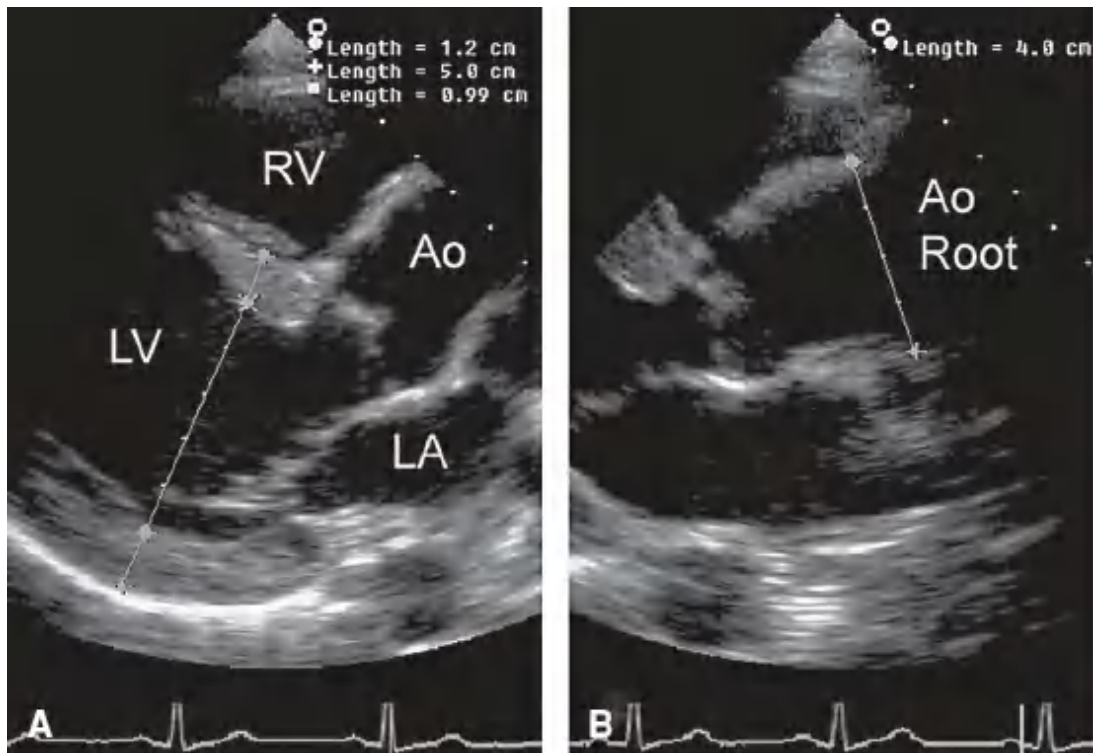


**FIGURE 4.11.** **A:** The parasternal long-axis view is adjusted so that the scan plane is parallel to the long axis of the left ventricle. In this plane, the proximal aorta appears normal. **B:** The plane is rotated slightly counterclockwise to better align with the long axis of the ascending aorta. 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.

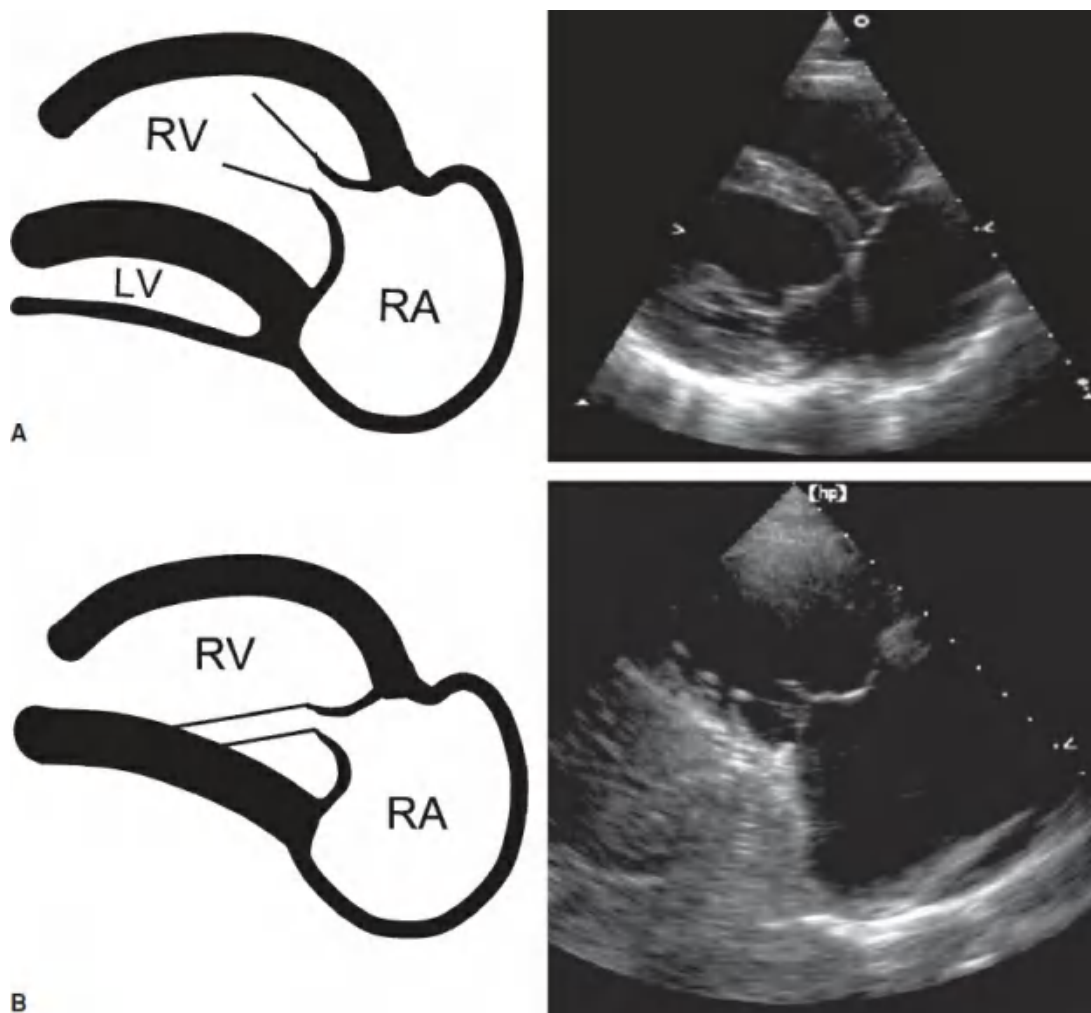
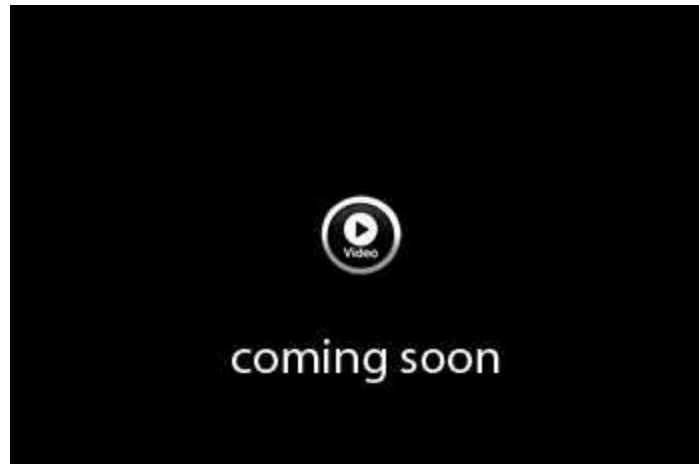


**FIGURE 4.12.** Two examples of the right ventricular inflow view are shown. **A:** A



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. 



### Video 4-12

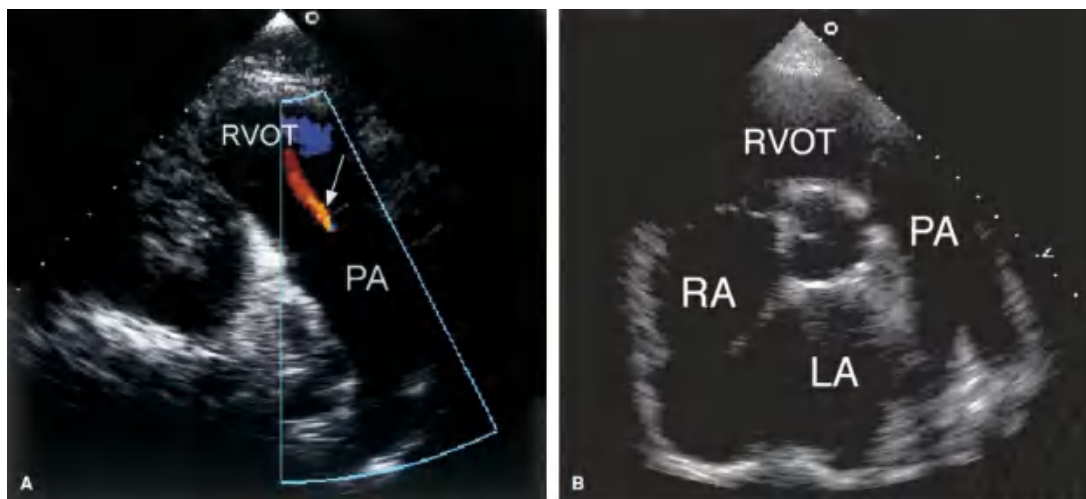


**FIGURE 4.13.** **A:** The right ventricular outflow view records the right ventricular outflow tract and the main PA. Trivial pulmonary valve regurgitation (*arrow*) is illustrated. **B:** The bifurcation of the main pulmonary artery is seen from the basal short-axis view.

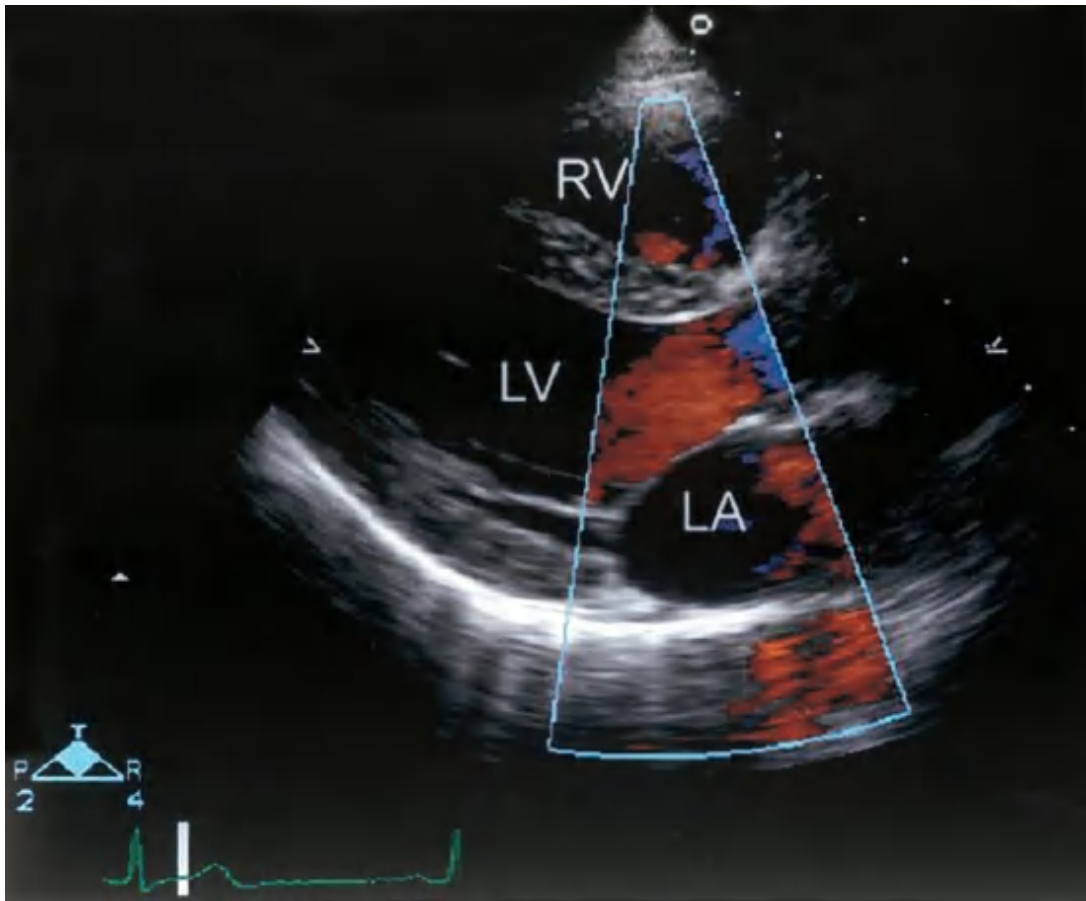
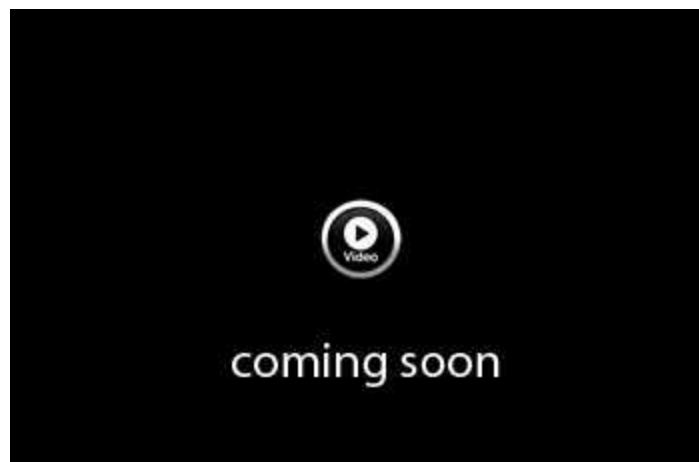


**FIGURE 4.14.** The parasternal long-axis view with color flow imaging. 📺



### Video 4-14

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.

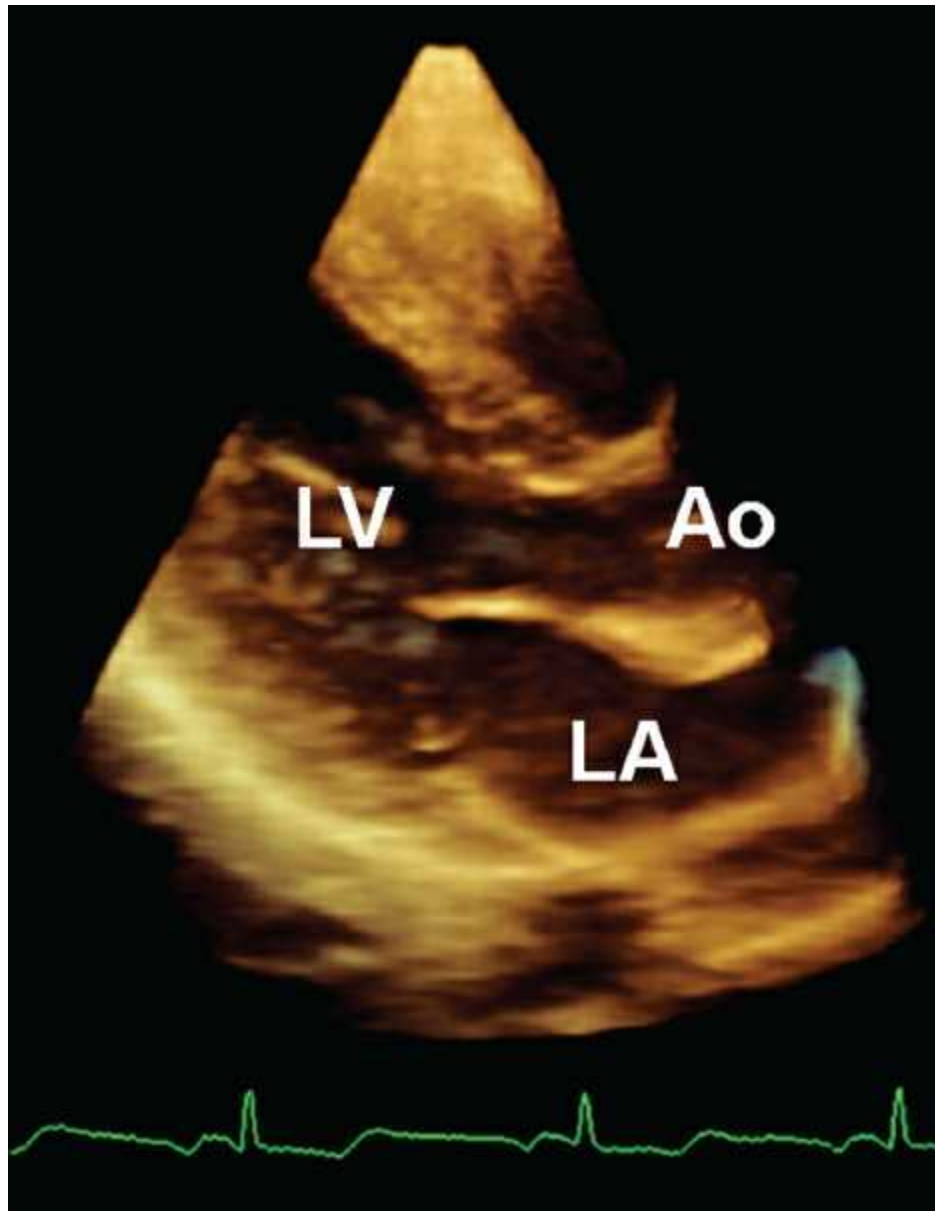
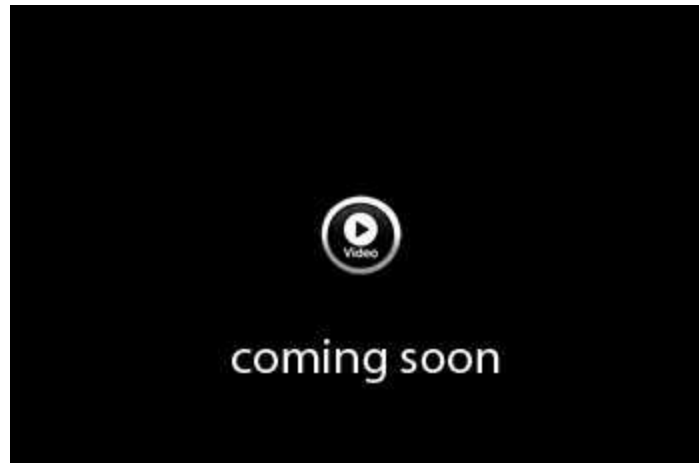


**FIGURE 4.15.** A three-dimensional image from a normal subject, recorded from the parasternal window. The image is oriented in the long-axis plane and illustrates how the thickness of slice can be used to record three-dimensional depth. 



**Video 4-15**

## **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).

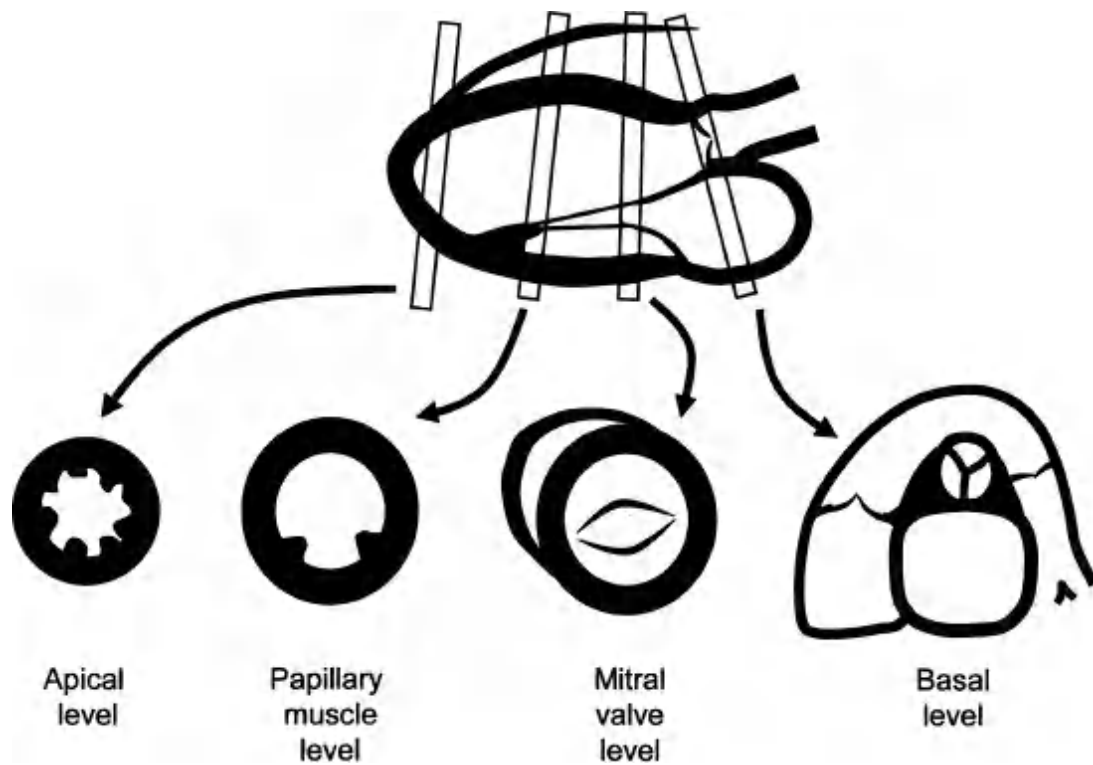
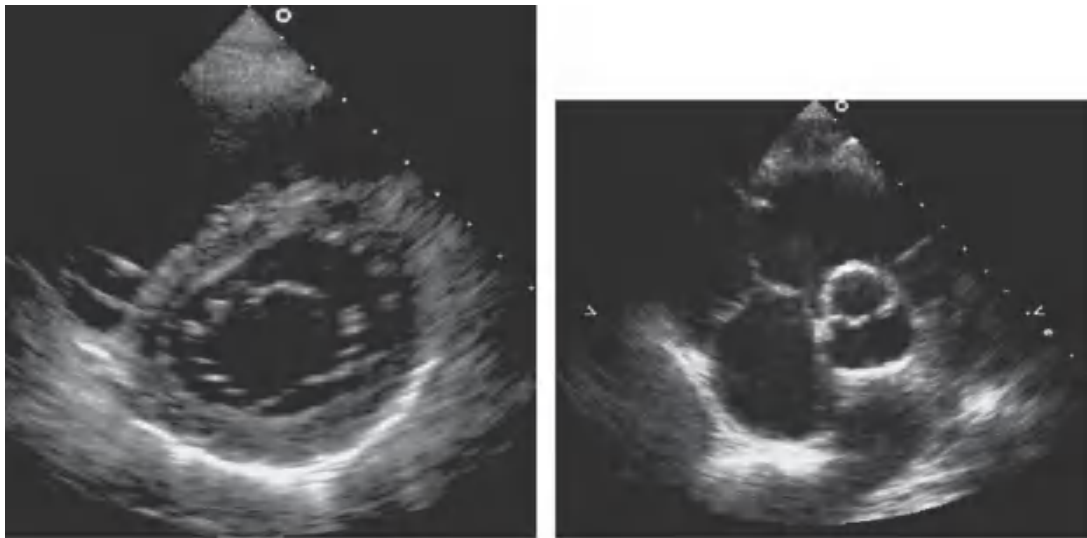
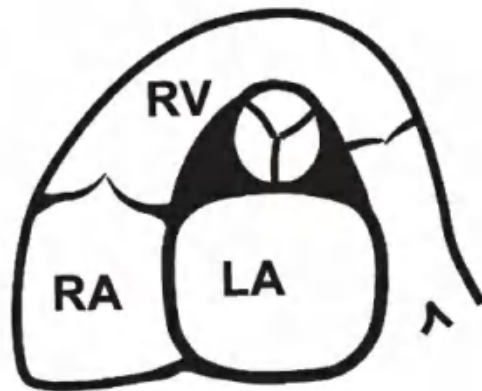


**FIGURE 4.16.** This schematic demonstrates the various short-axis planes that can be derived from the parasternal long-axis view. Note that the planes are not exactly parallel but provide views of anatomy from apex to base.

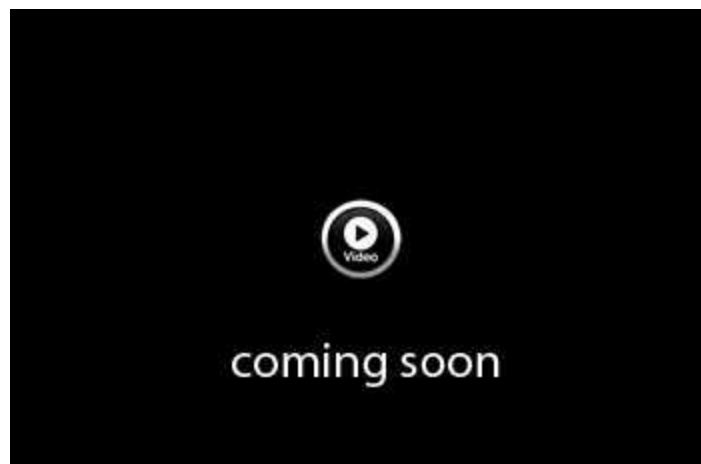


A



B

**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.



**Video 4-17**



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.

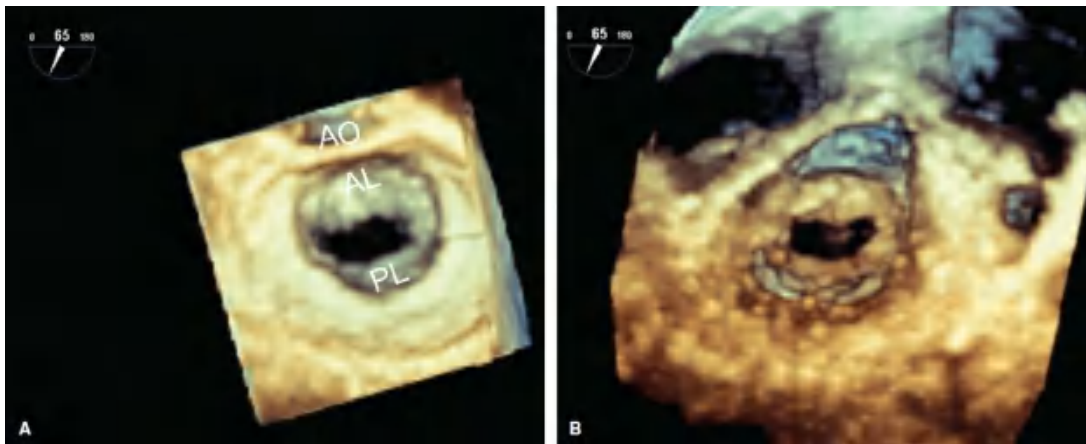
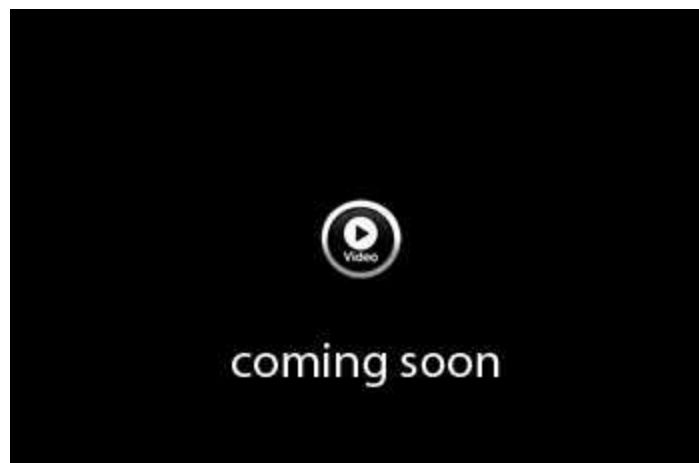
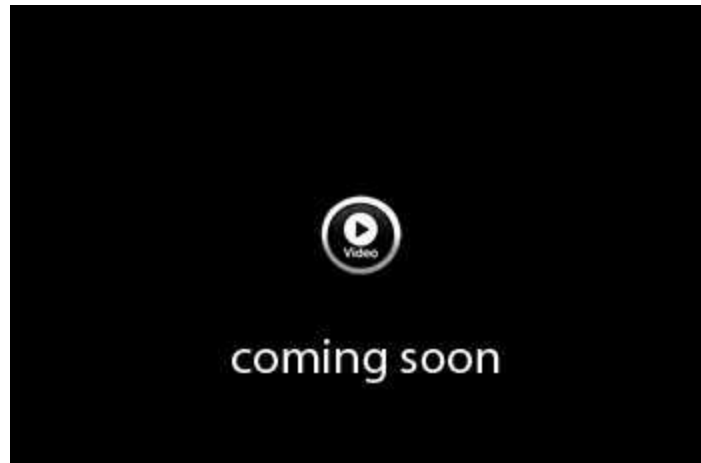


**FIGURE 4.18.** Three-dimensional views of a stenotic mitral valve are shown. In (A), the valve is shown from the perspective of the left atrium. In (B), from the perspective of the left ventricle. AL, anterior mitral leaflet; PL, posterior mitral leaflet. 



**Video 4-18a**



**Video 4-18b**

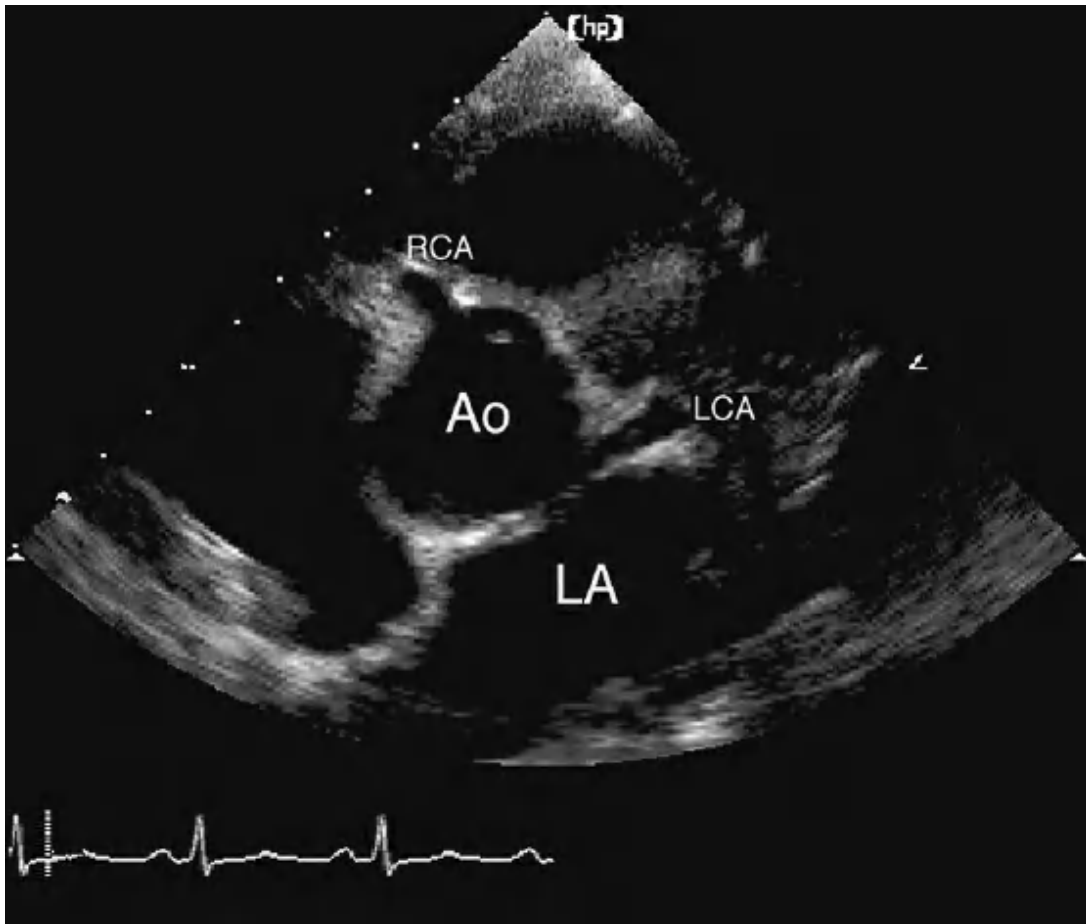


**FIGURE 4.19.** From the basal short-axis view just above the aortic valve, the origins of the left coronary artery (LCA) and right coronary artery (RCA) can be recorded.

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.

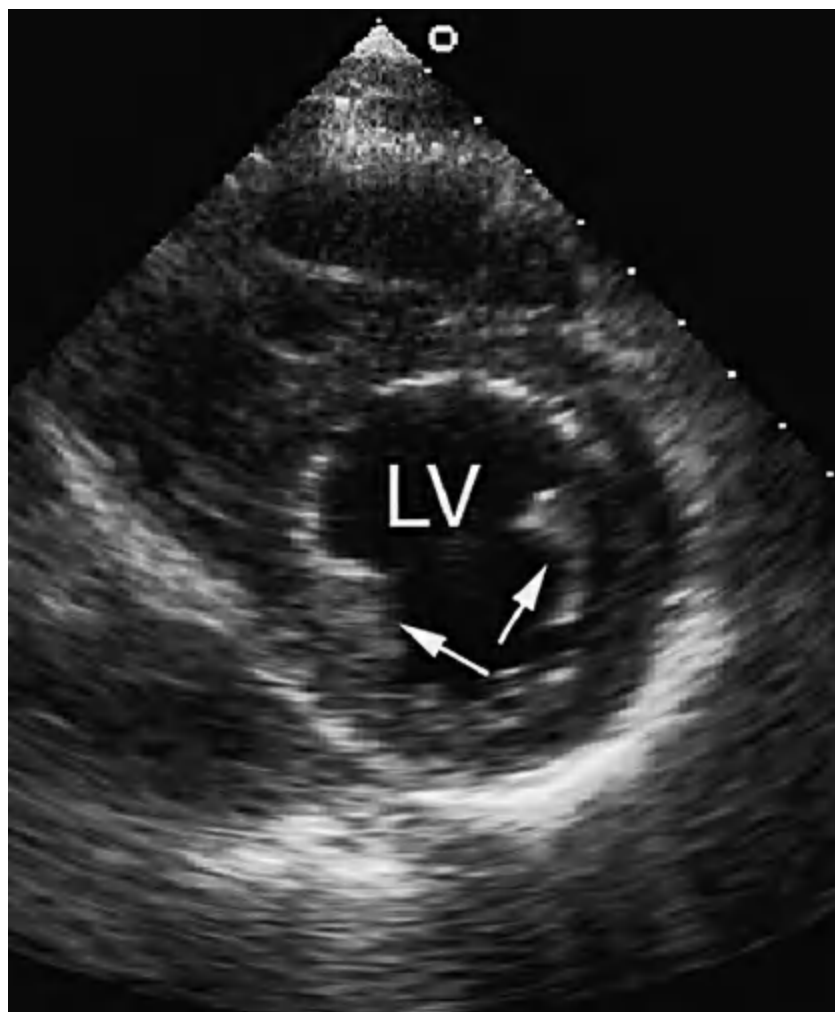
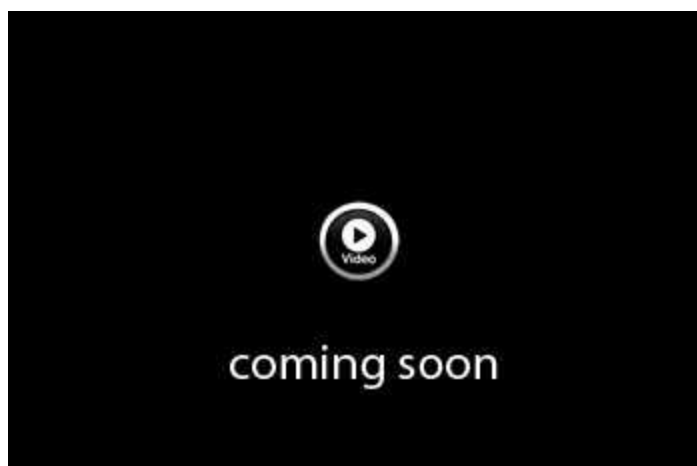


**FIGURE 4.20.** A short-axis plane at the level of the papillary muscles (*arrows*).



**Video 4-20**

## APICAL VIEWS

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.

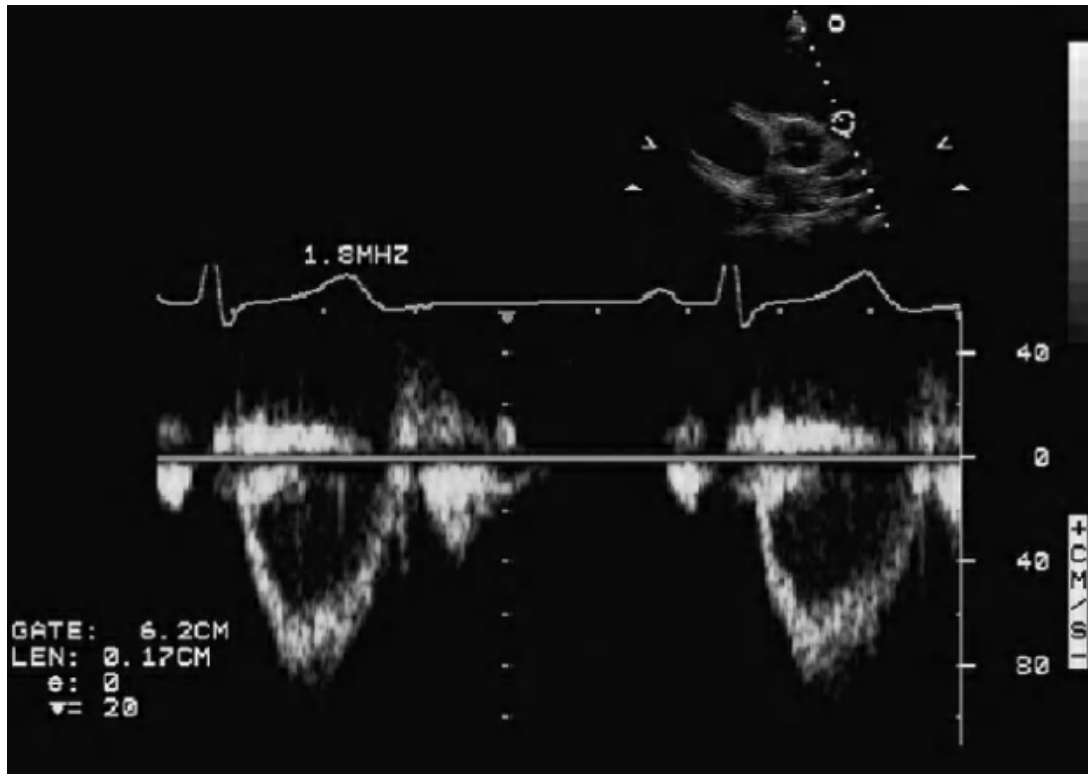
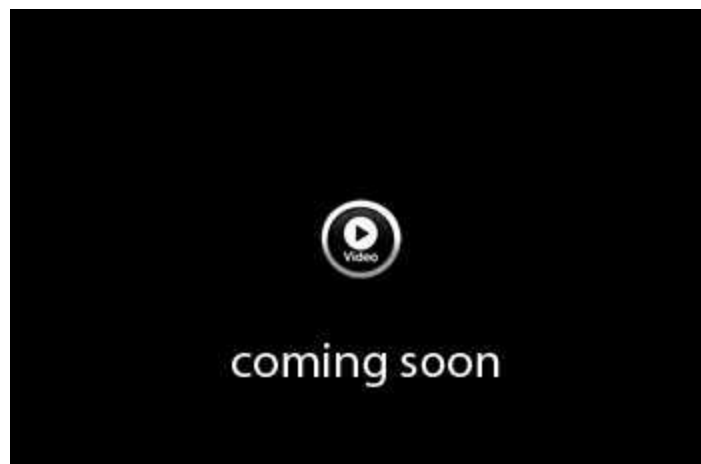


**FIGURE 4.21.** The basal short-axis view is ideal to record flow through the pulmonary valve using pulsed Doppler imaging.



**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**

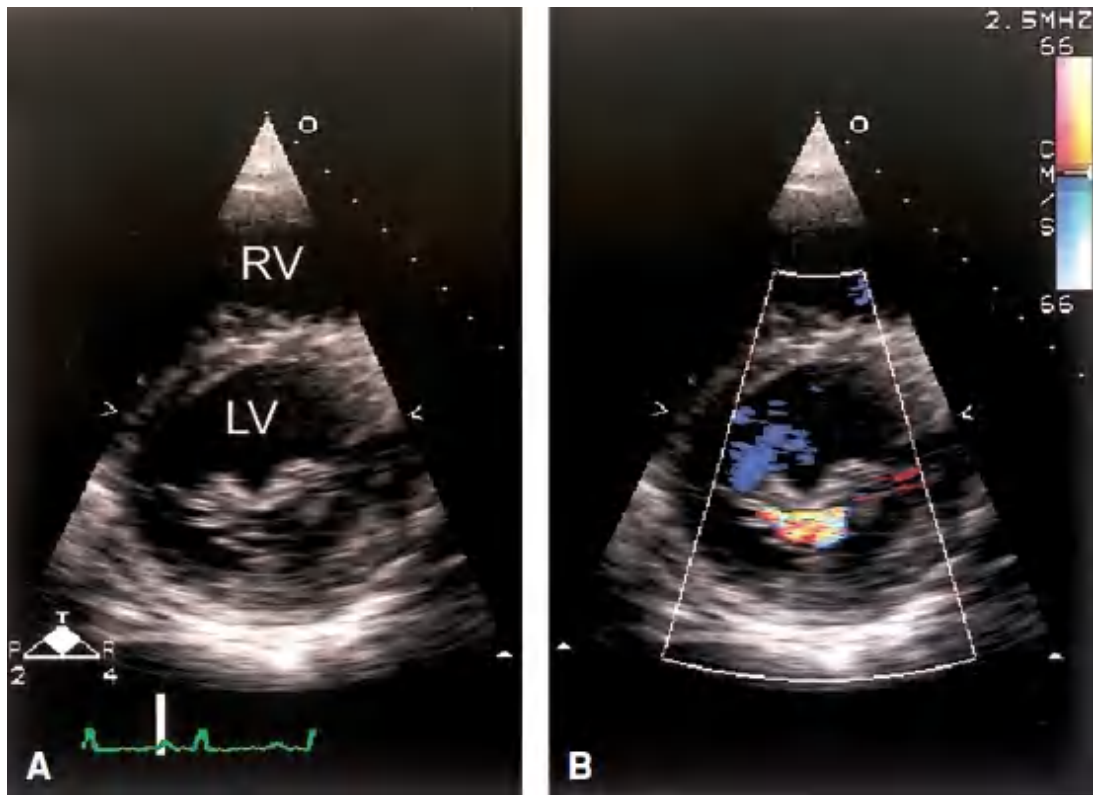
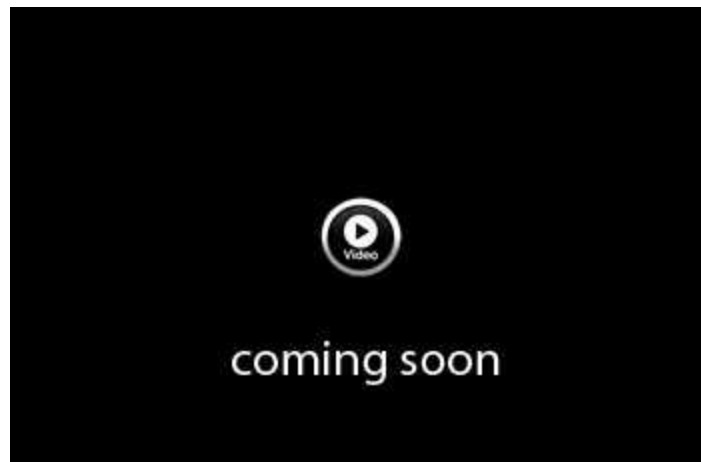


**FIGURE 4.23.** At the level of the tips of the mitral leaflets, the short-axis view permits the mitral regurgitation jet to be recorded. **A:** Two-dimensional imaging shows thickened mitral leaflets. **B:** Color flow imaging shows the extent of the regurgitant jet at the same level. 📺



**Video 4-23**



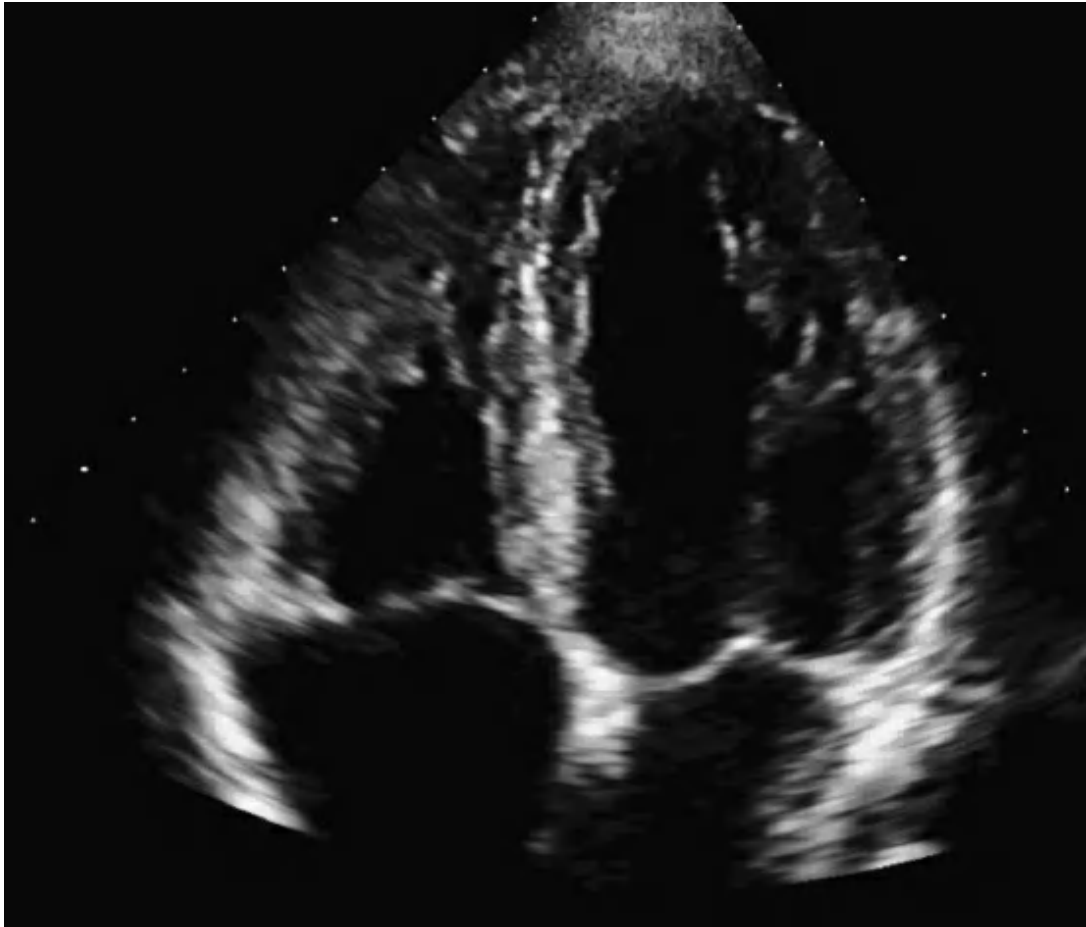
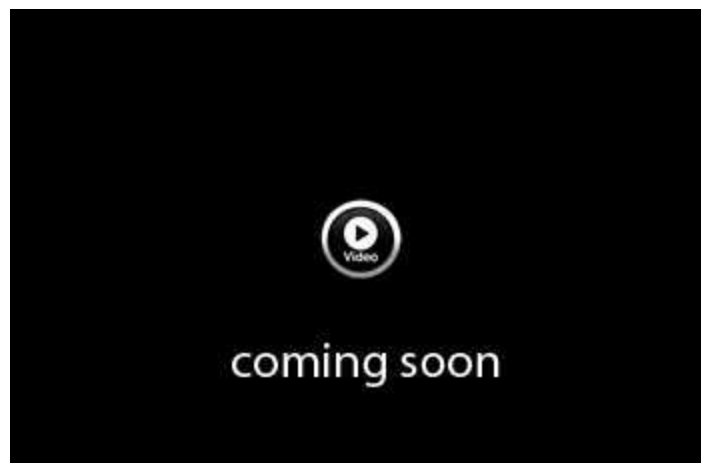


**FIGURE 4.24.** The apical four-chamber view. 📺



**Video 4-24**

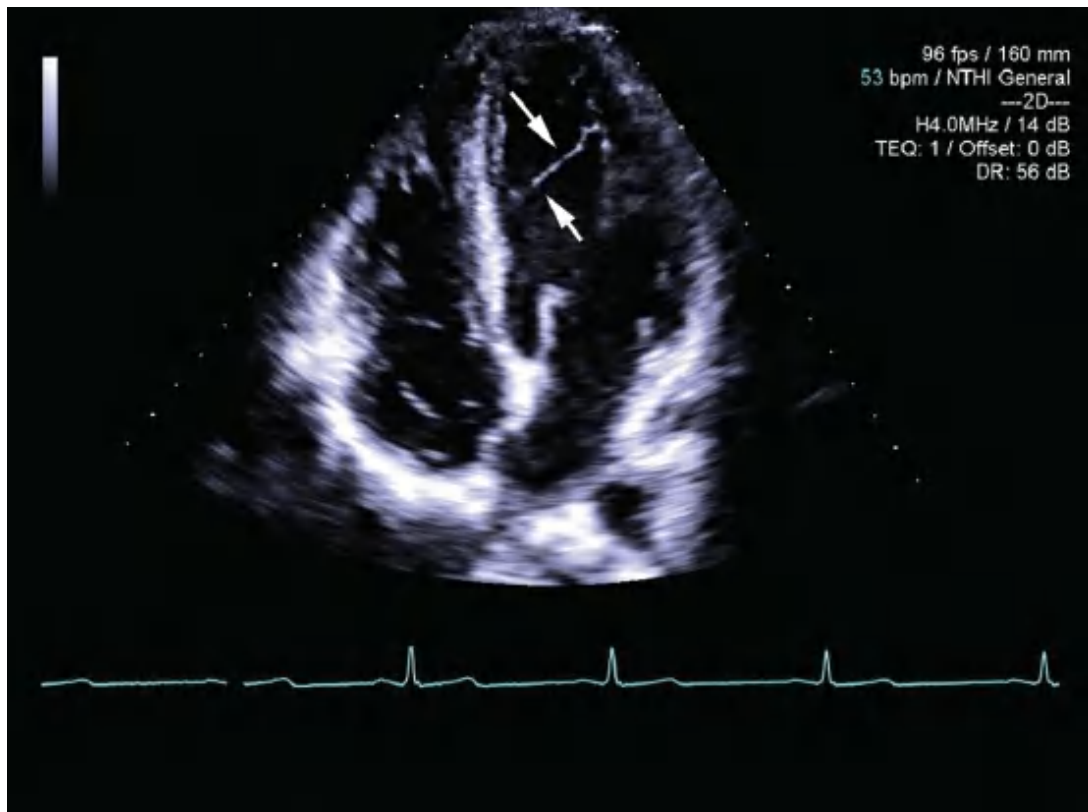
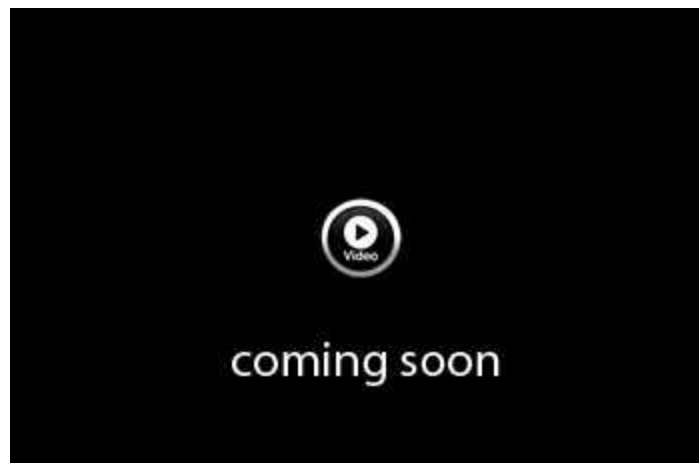


**FIGURE 4.25.** An example of a false tendon (*arrows*) in the left ventricular cavity.



### 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.

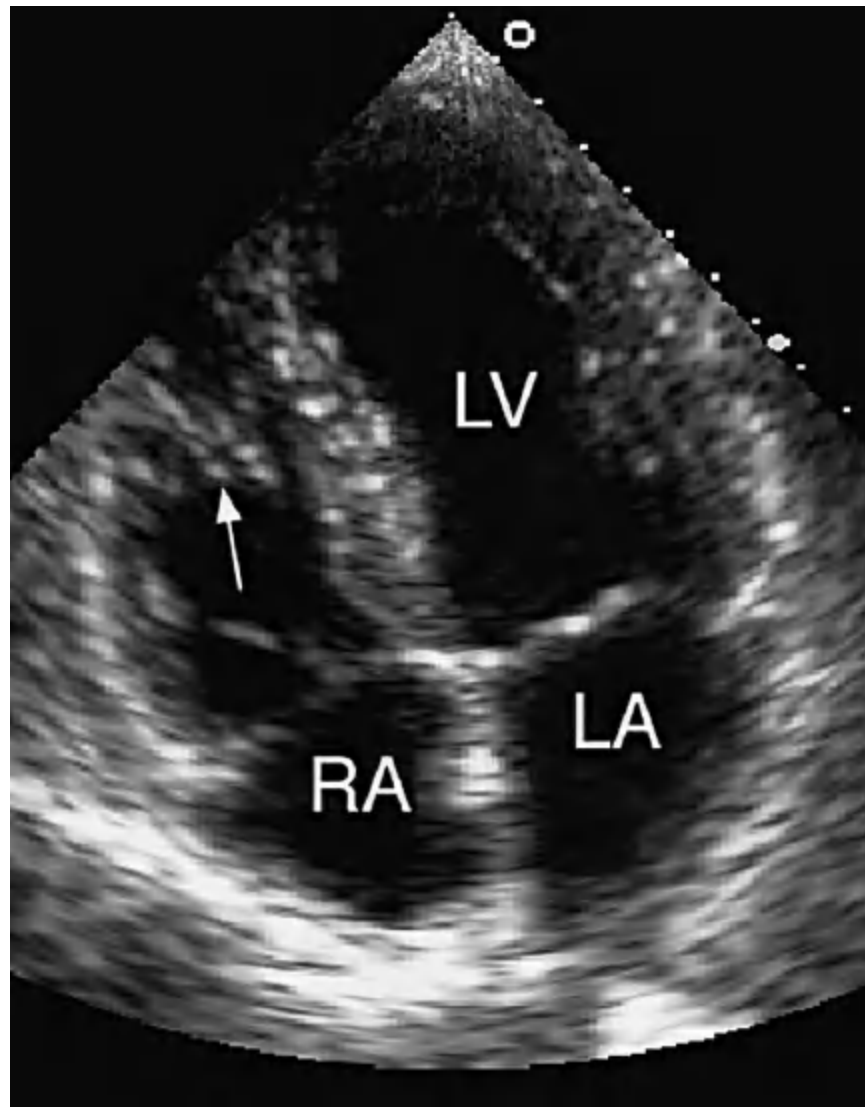


**FIGURE 4.26.** An apical four-chamber view demonstrates a moderator band (*arrow*) in the right ventricular apex.

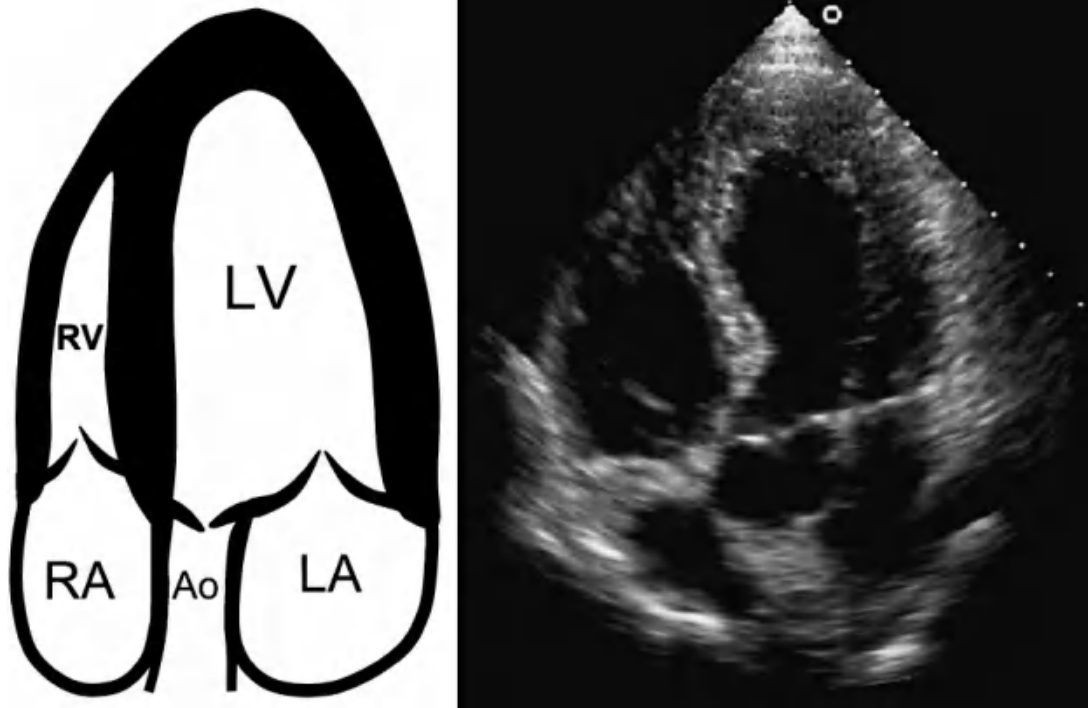
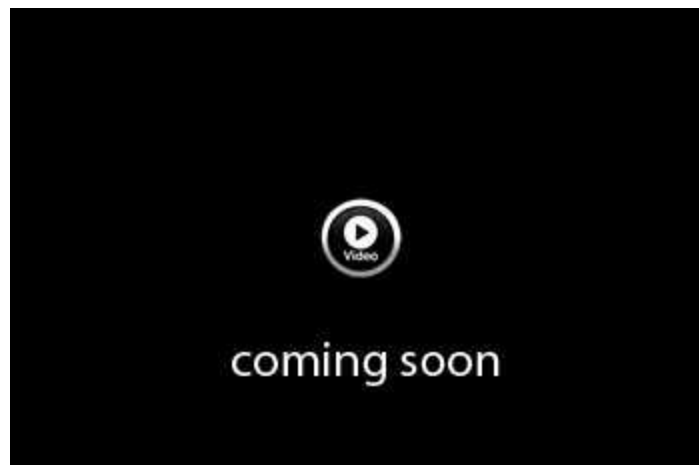


**FIGURE 4.27.** Starting from the four-chamber view, the transducer can be tilted to a shallower angle to produce a plane that includes the left ventricular outflow tract and proximal aorta. 📺



### Video 4-27

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.

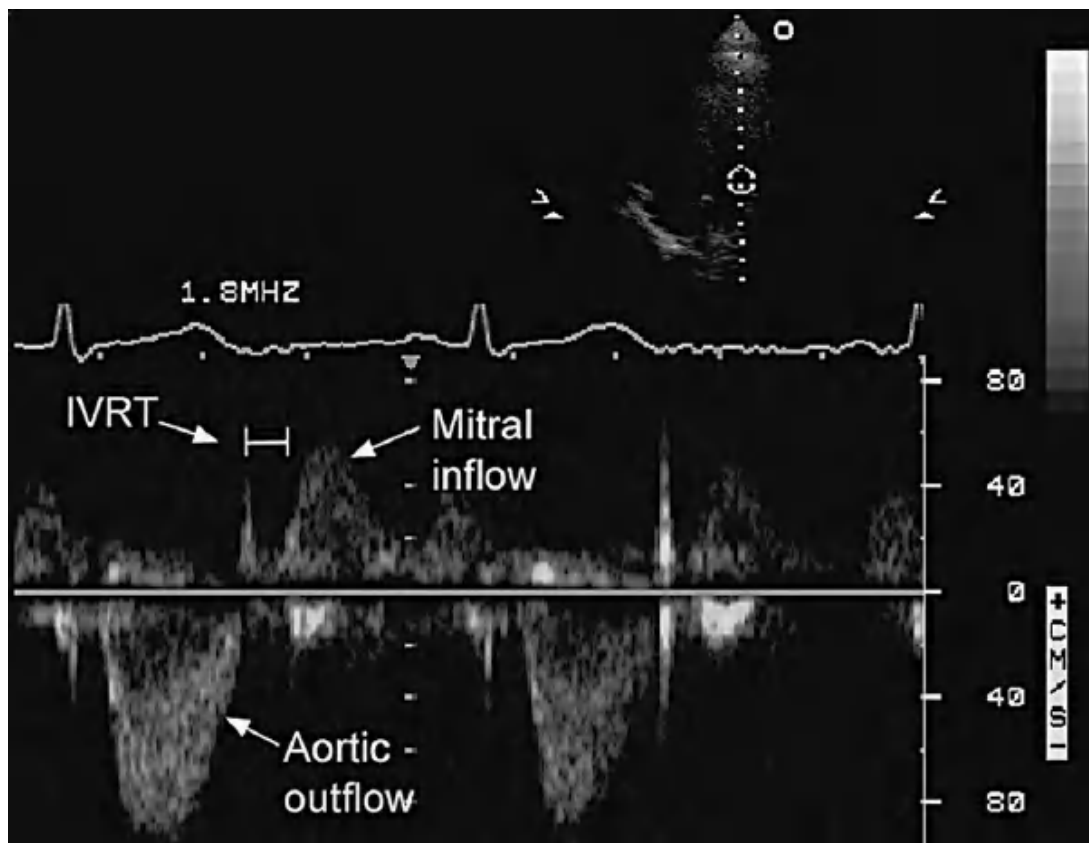


**FIGURE 4.28.** From the apical five-chamber view, simultaneous recording of aortic outflow and mitral inflow can be performed. This permits isovolumic relaxation time (IVRT) to be measured.

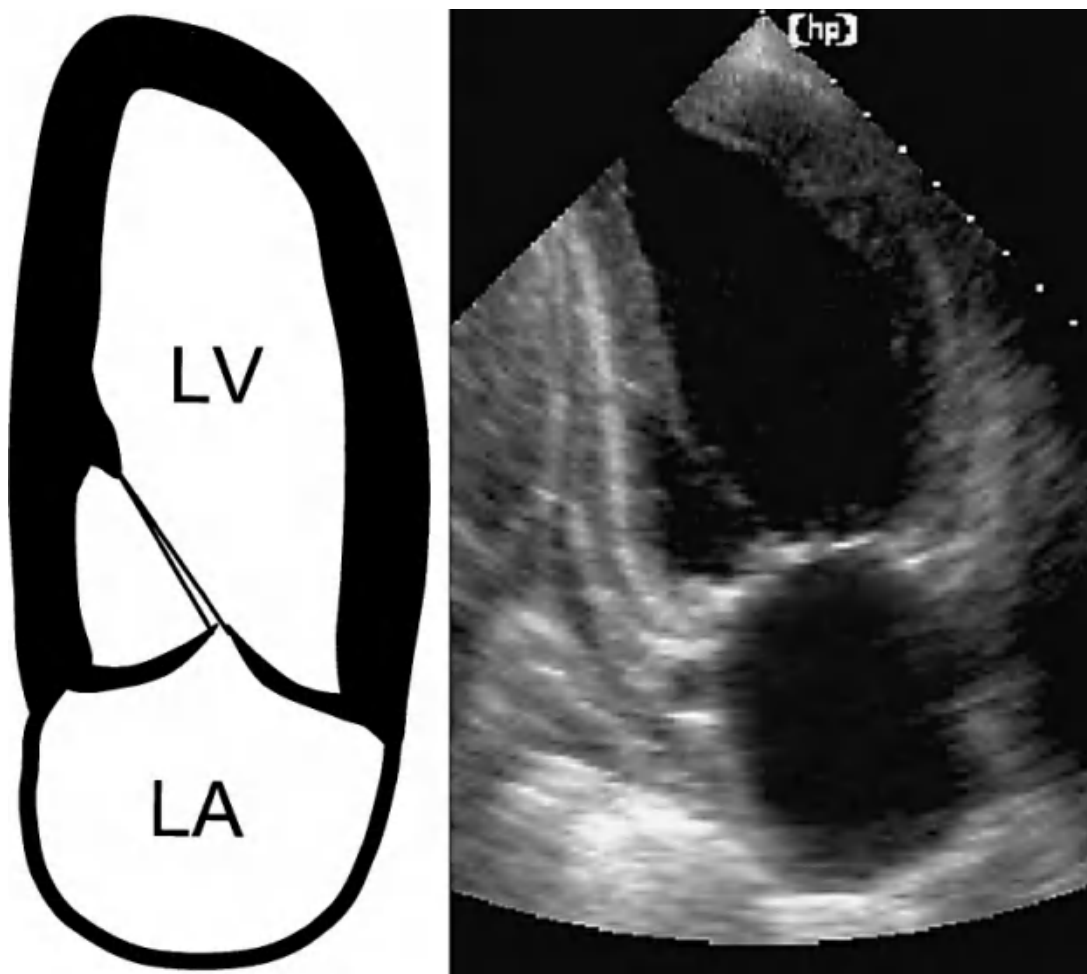
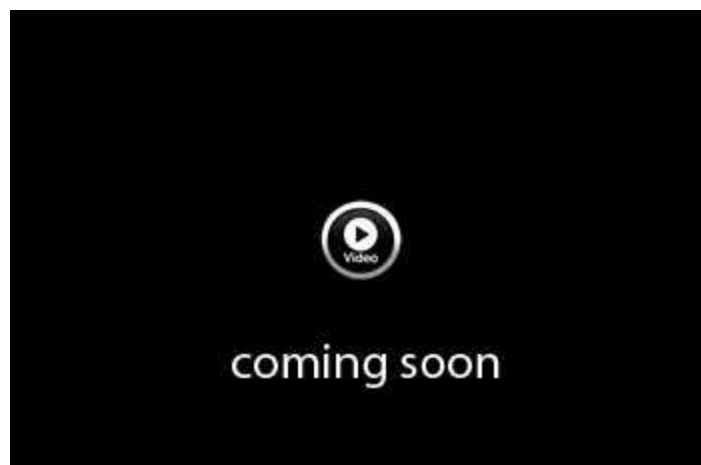


FIGURE 4.29. An apical two-chamber view. 



Video 4-29

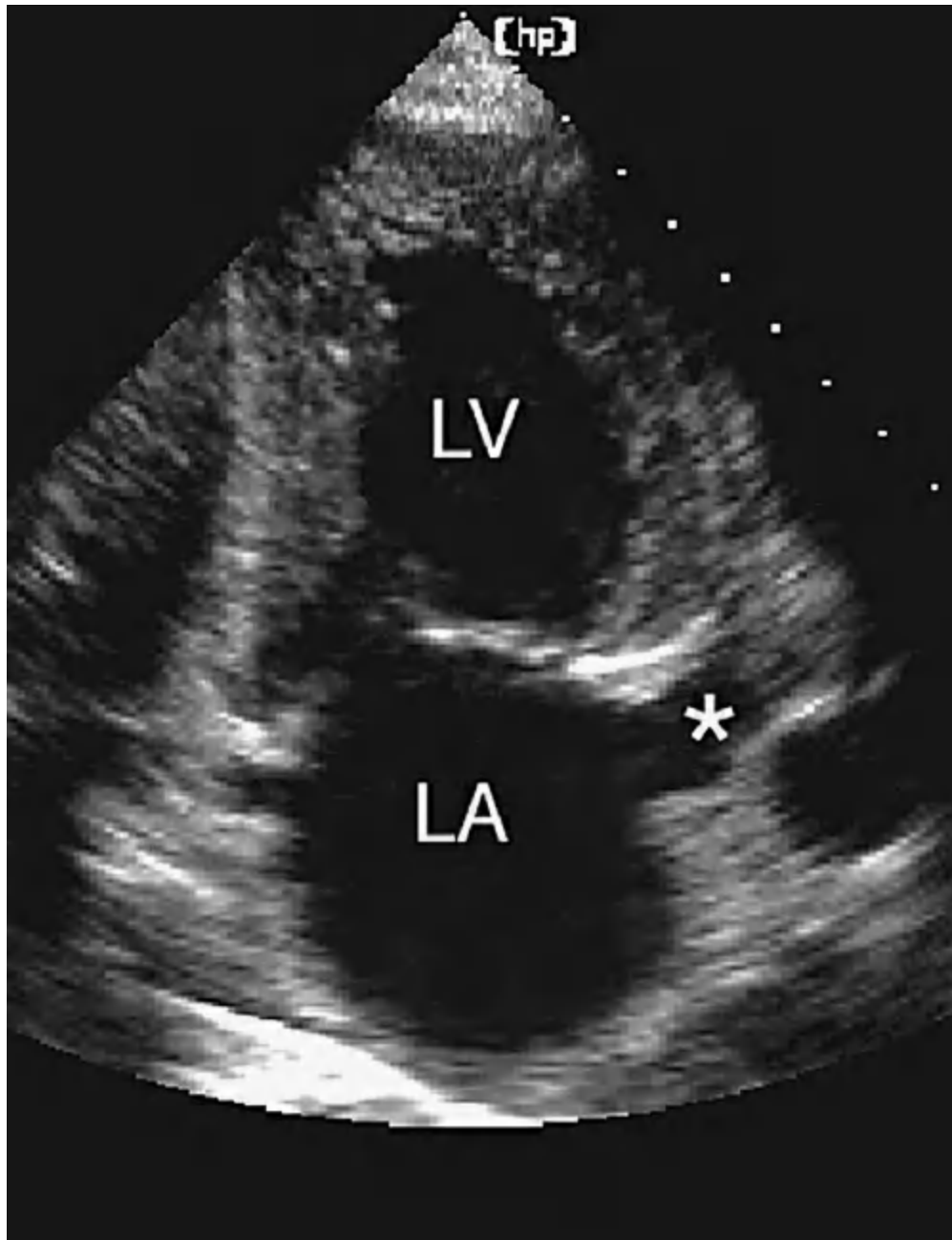
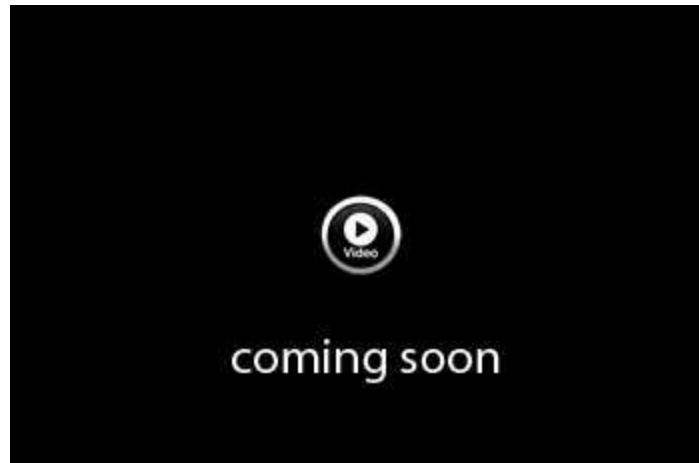


**FIGURE 4.30.** The two-chamber view sometimes allows the left atrial appendage (\*) to be visualized. 📺





### **Video 4-30**

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](#).

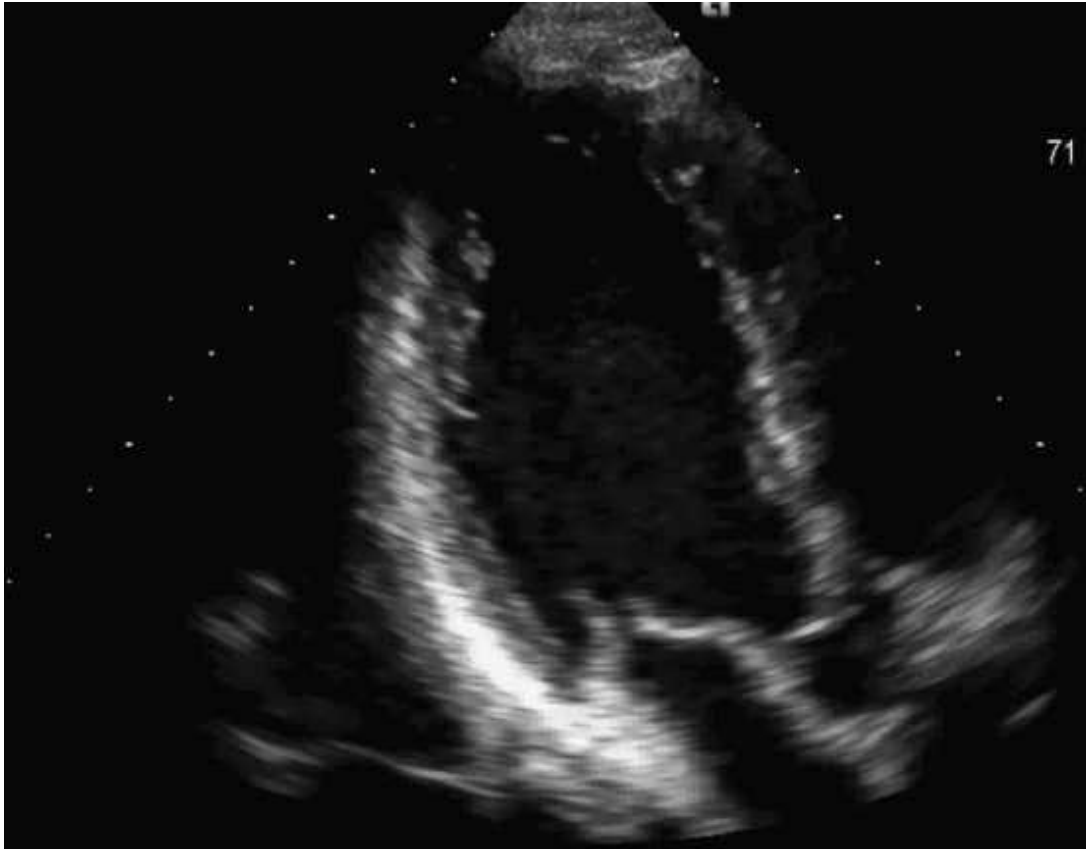
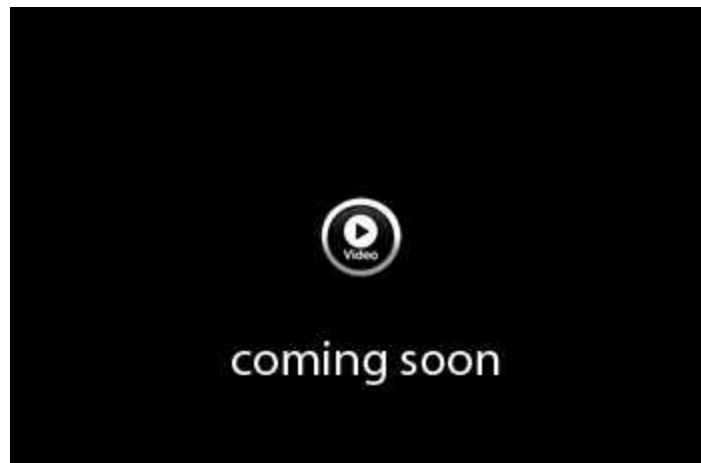


**FIGURE 4.31.** The apical long-axis view is similar to the parasternal long-axis view but is recorded from a lower interspace. 📺



**Video 4-31**

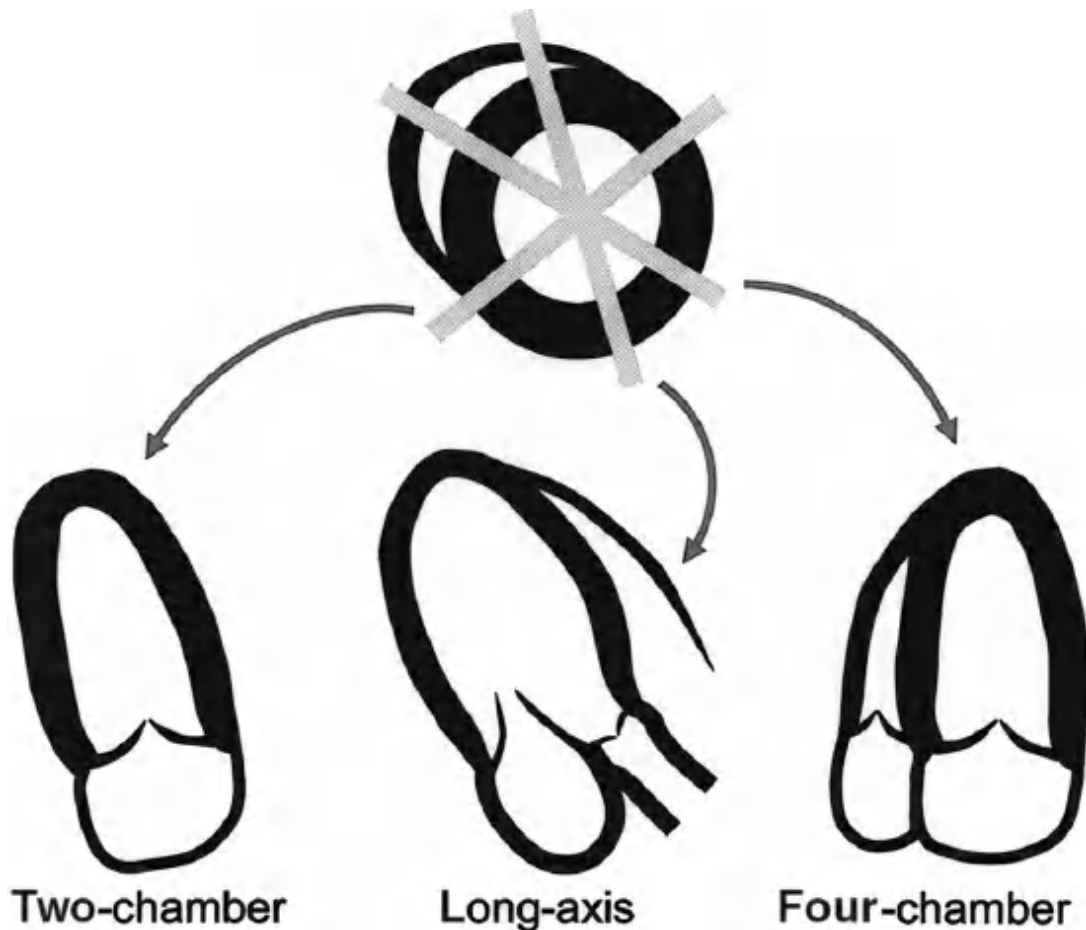


**FIGURE 4.32.** The relationship among the various apical long-axis views and the parasternal short axis. See text for details.

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

recorded. Finally, from the apical views, color Doppler imaging should be routinely performed to assess for regurgitation of the mitral, aortic, or tricuspid valve.

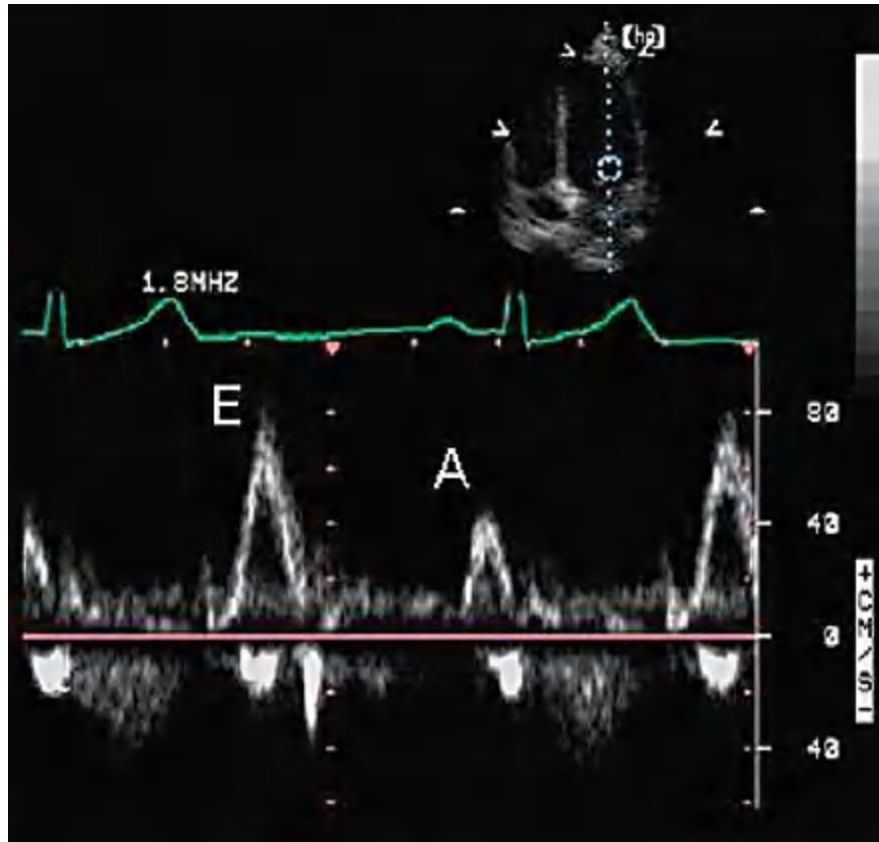


**FIGURE 4.33.** This composite image illustrates how volume and ejection fraction are derived from the three-dimensional image. By tracking the endocardial border over the course of the cardiac cycle, calculation of instantaneous volume of the left ventricle is performed. This represents a semi-automated approach to quantify end-systolic (ESV) and end-diastolic (EDV) volumes from which ejection fraction (EF) is derived.

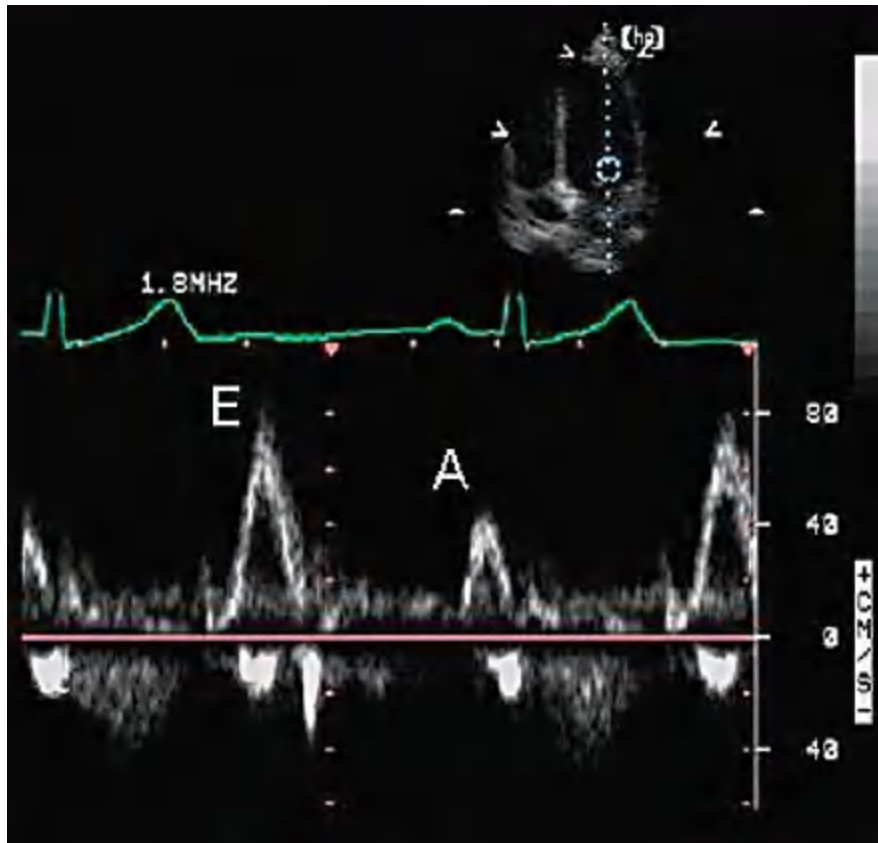


**FIGURE 4.34.** The apical four-chamber view is ideal to record mitral inflow using pulsed Doppler imaging. In this normal example, the E- and A-waves are demonstrated.

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](#)).

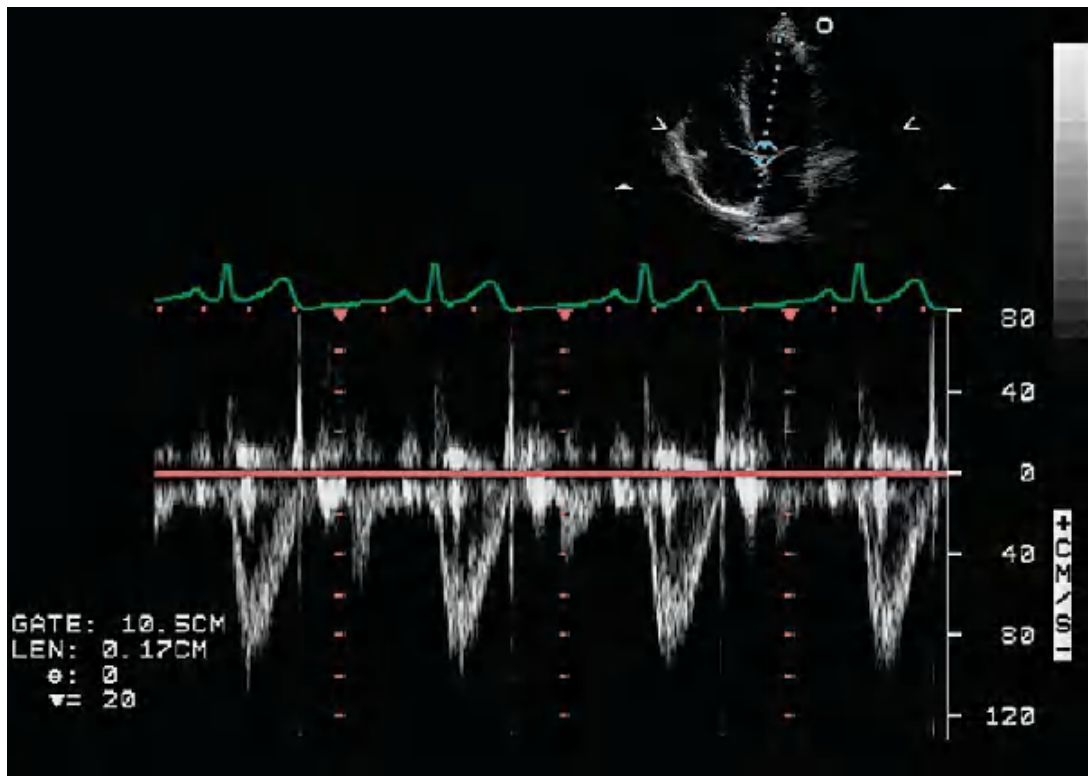


**FIGURE 4.35.** The apical five-chamber view allows recording of aortic outflow using the pulsed Doppler technique.

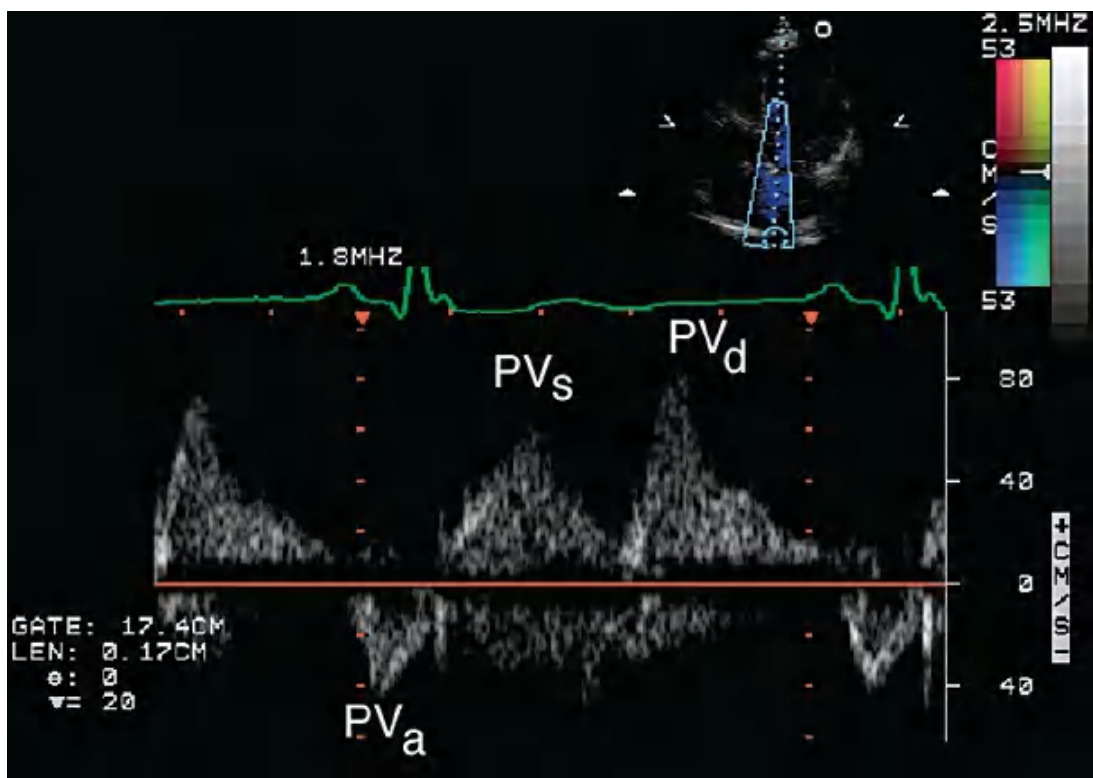


**FIGURE 4.36.** From the apical four-chamber view, pulsed Doppler imaging can

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).

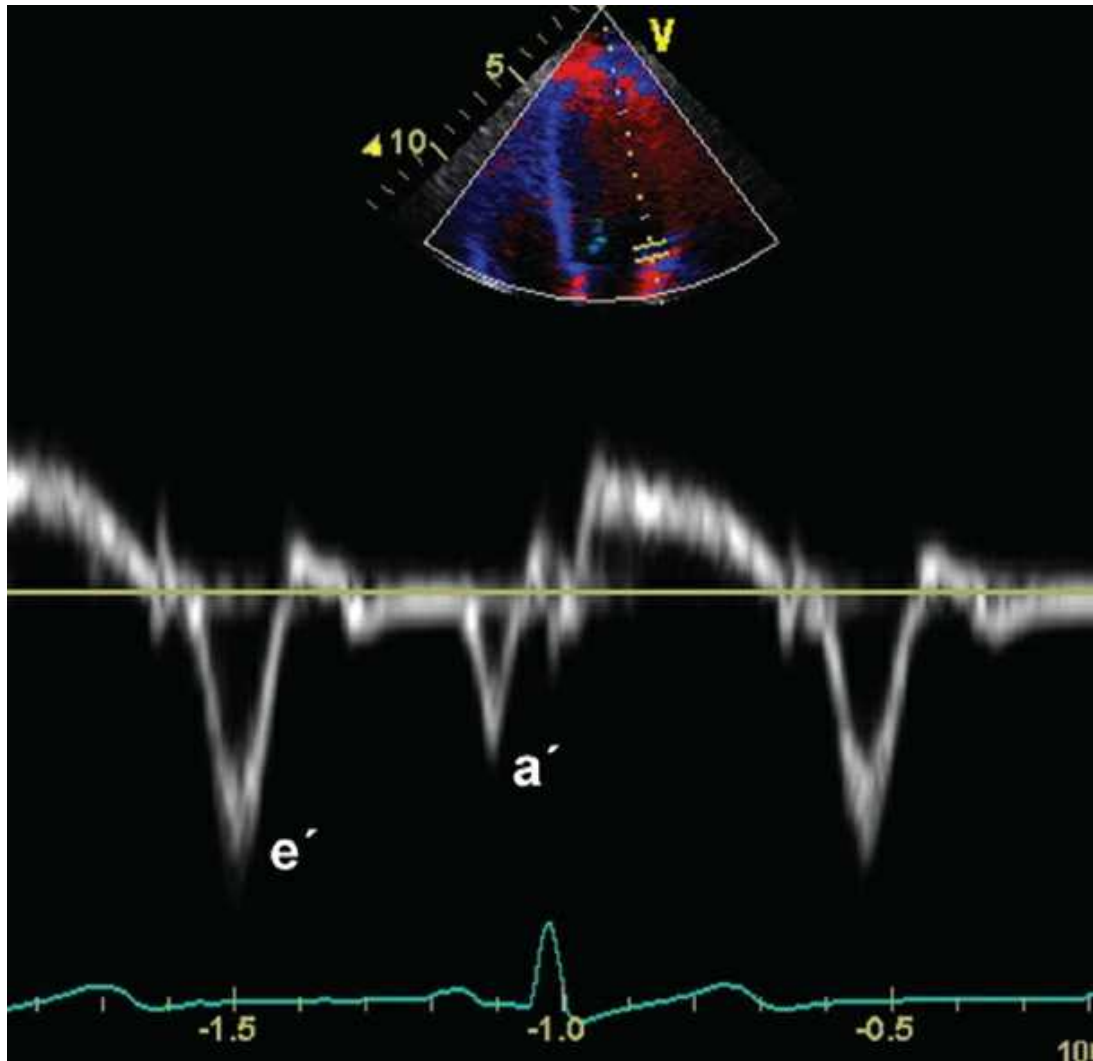


**FIGURE 4.37.** Tissue Doppler lateral imaging of the lateral mitral annulus demonstrates velocity away from the transducer in systole and two waves toward the transducer (e' and a') in diastole.



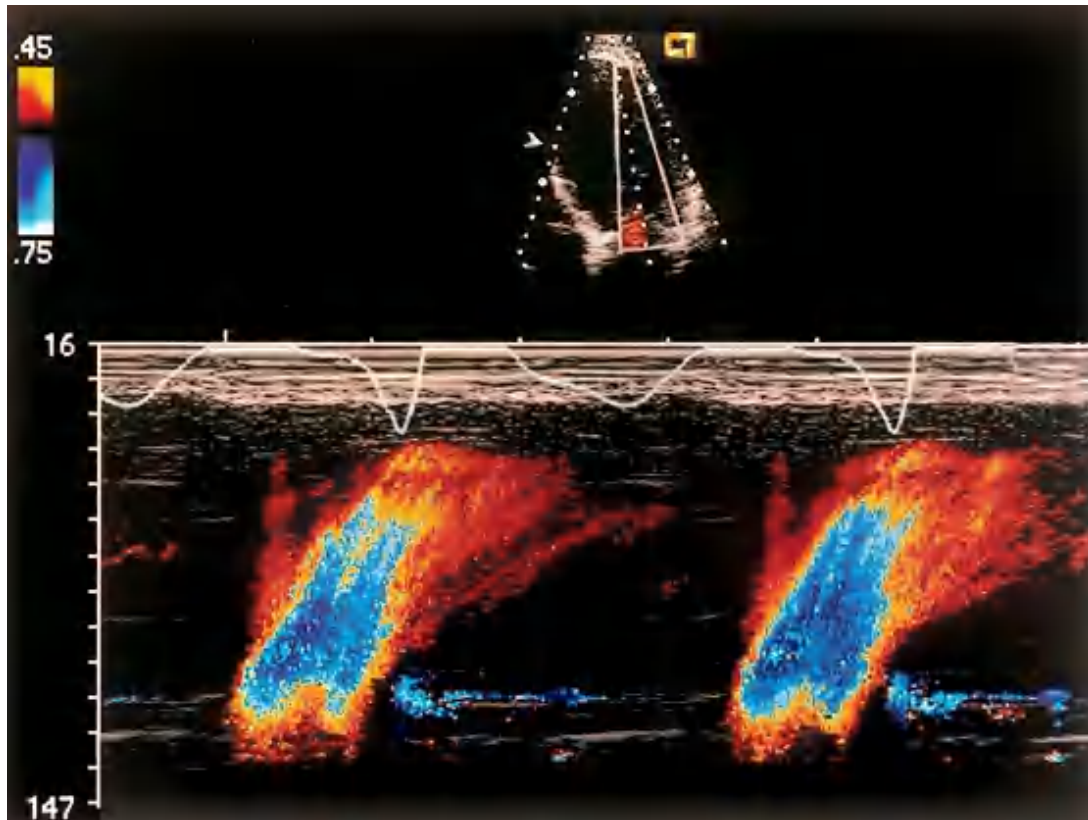


**FIGURE 4.38.** Color M-mode recording of mitral inflow during diastole, recorded from the apical window.

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.

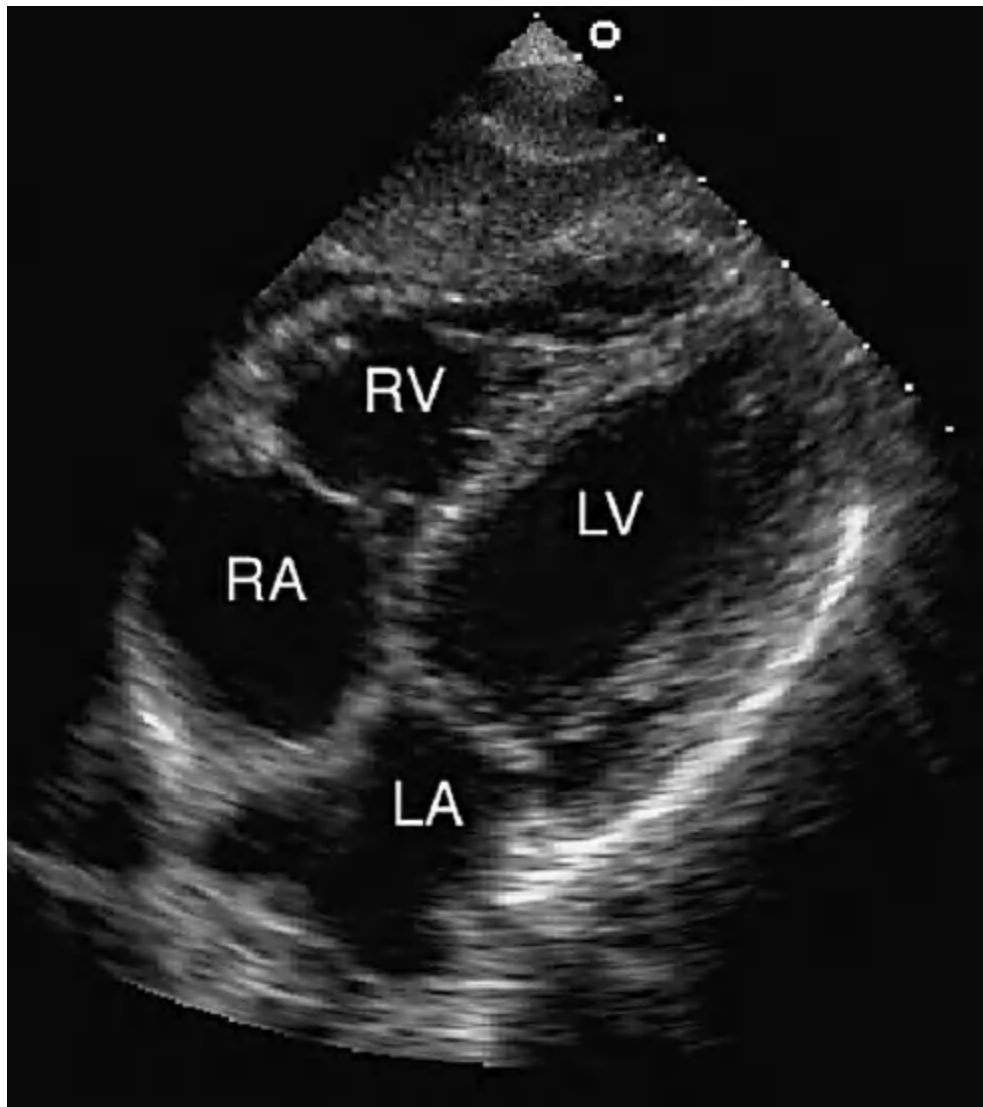


**FIGURE 4.39.** A subcostal four-chamber view.

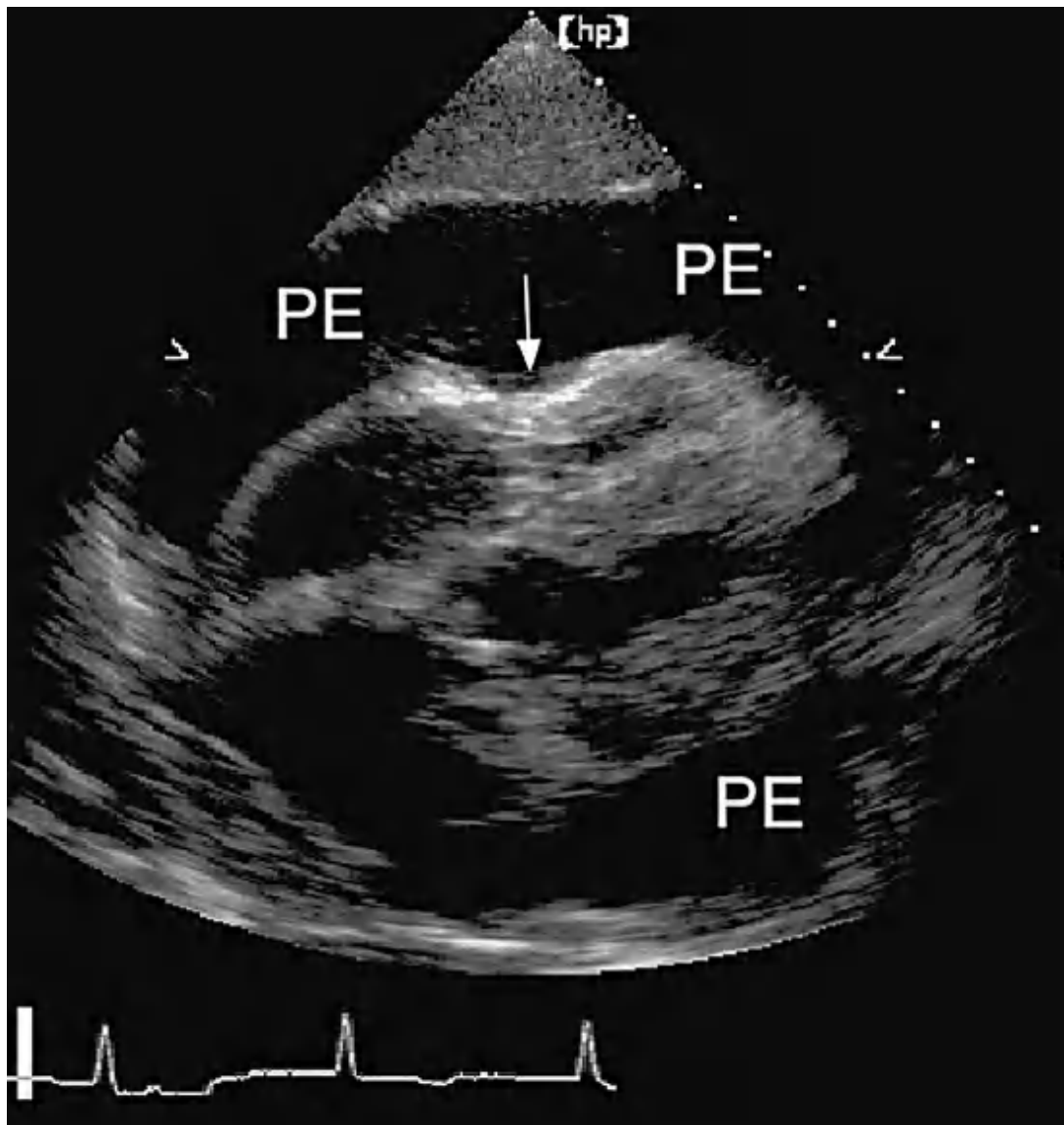
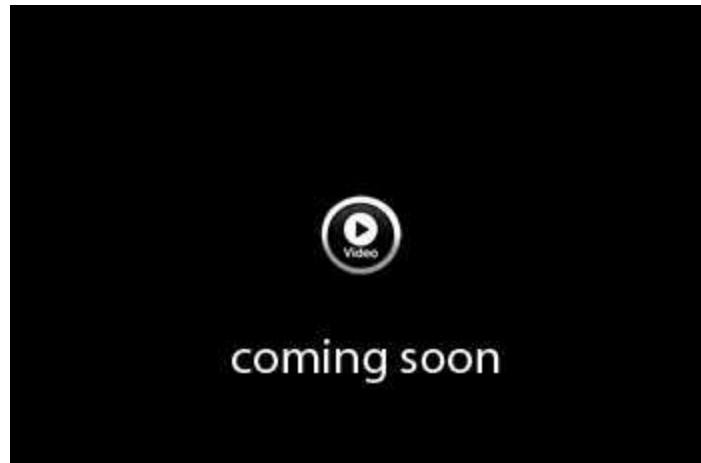


**FIGURE 4.40.** A subcostal four-chamber view from a patient with a large pericardial effusion (PE). From this window, diastolic right ventricular free wall collapse (*arrow*) can be demonstrated. 



**Video 4-40**

## **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.

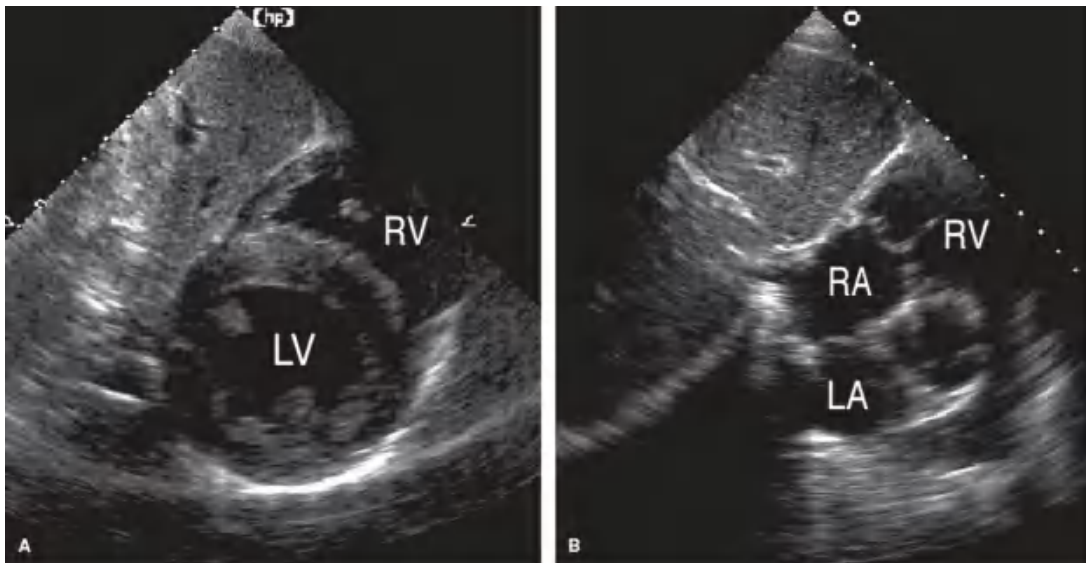
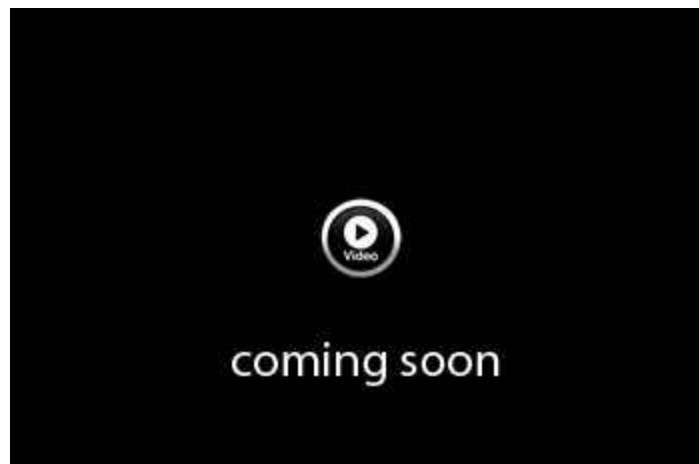


**FIGURE 4.41.** **A:** A subcostal short-axis view at the level of the papillary muscles. **B:** A short-axis view at the base. This view provides a clear recording of the interatrial septum and the right ventricular outflow tract, pulmonary valve, and main pulmonary artery. 



### 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.

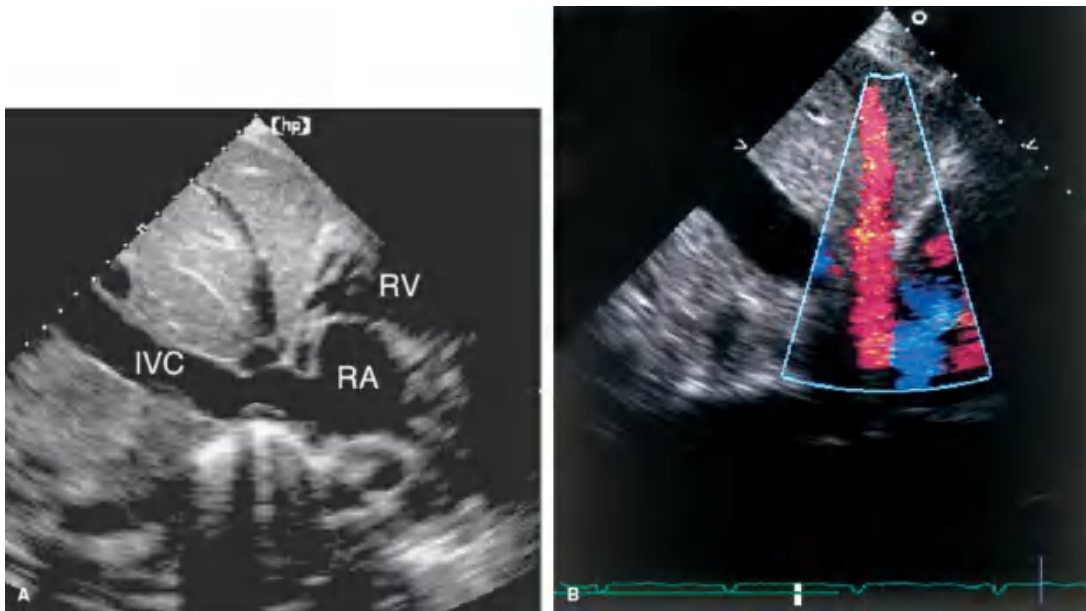
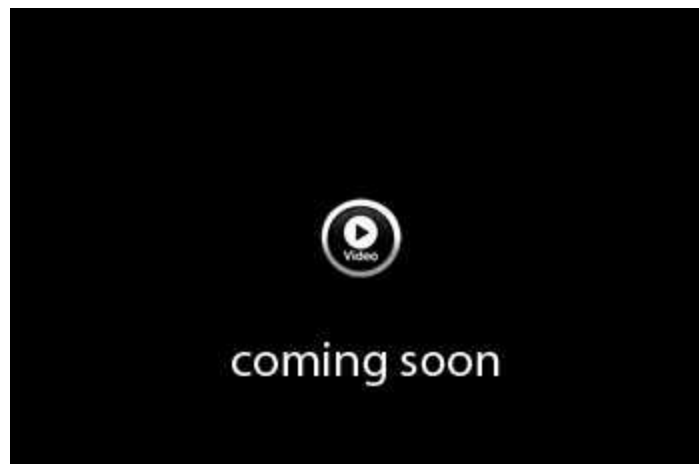


**FIGURE 4.42.** **A:** The subcostal view is adjusted to demonstrate the long axis of the IVC joining the RA. **B:** Color flow imaging of hepatic vein flow. 



**Video 4-42**

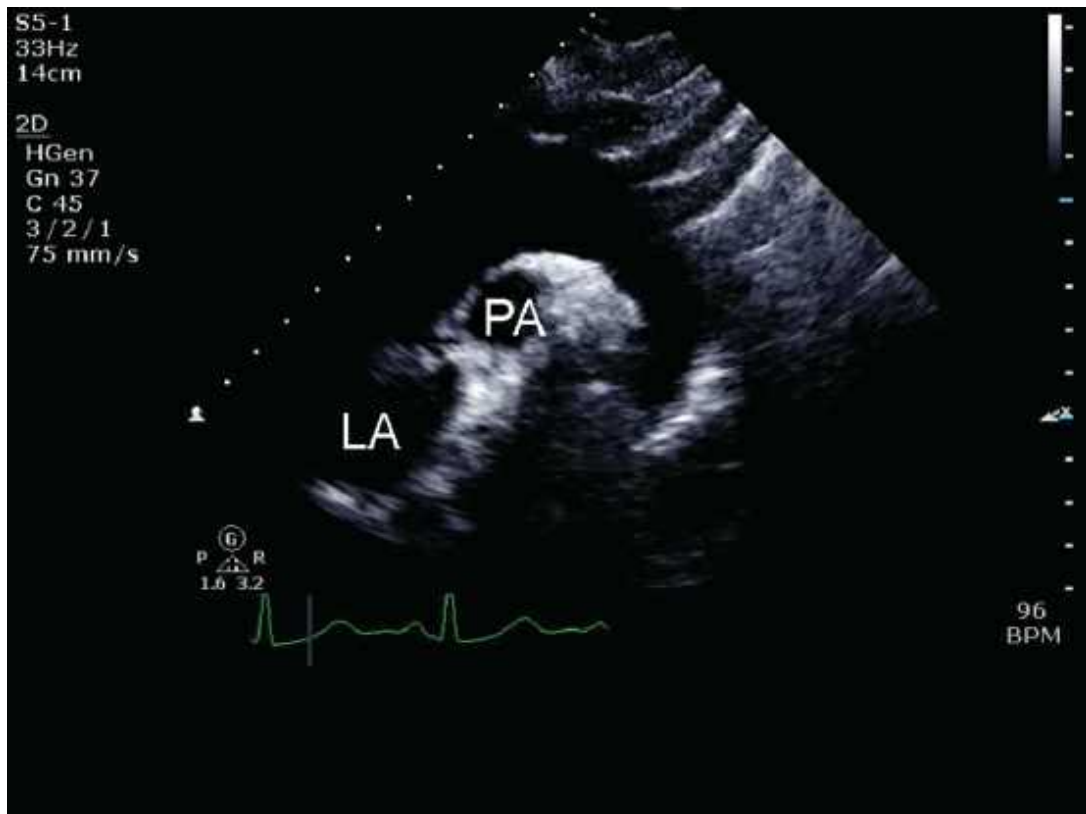


**FIGURE 4.43.** From the suprasternal notch, the imaging plane is aligned parallel to the aortic arch. The relationship among the arch, right PA, and LA is demonstrated.

## 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.



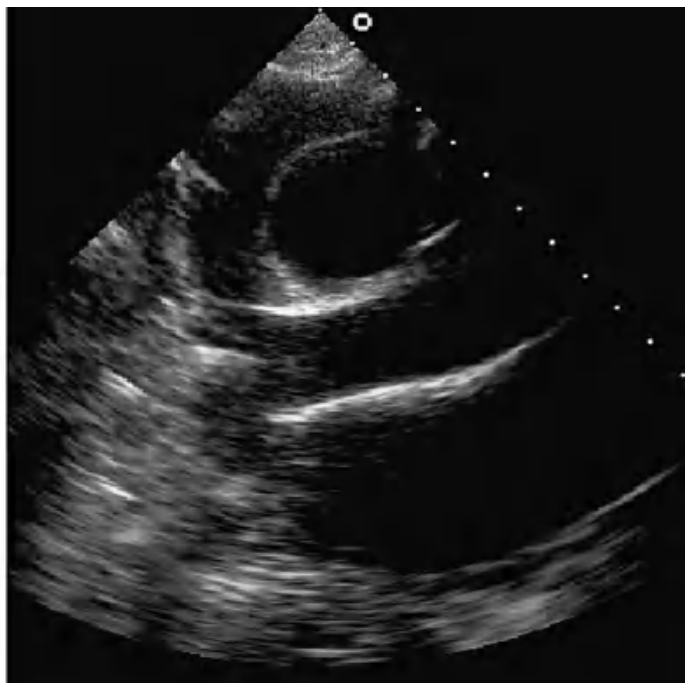
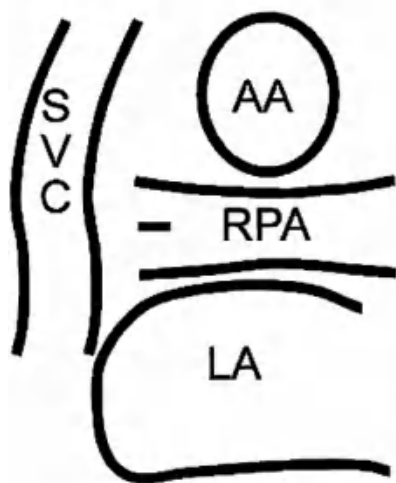
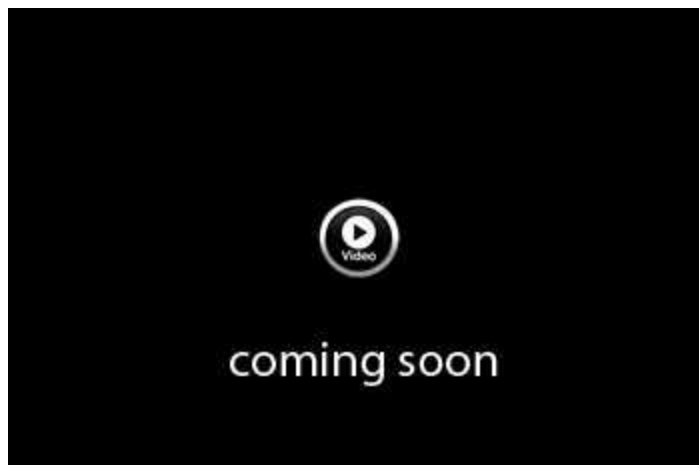


**FIGURE 4.44.** The suprasternal notch also permits the aortic arch (AA) to be recorded in cross section. This plane allows visualization of the superior vena cava and demonstrates the right pulmonary artery (RPA) coursing below the arch and above the left atrium. 📺



**Video 4-44**

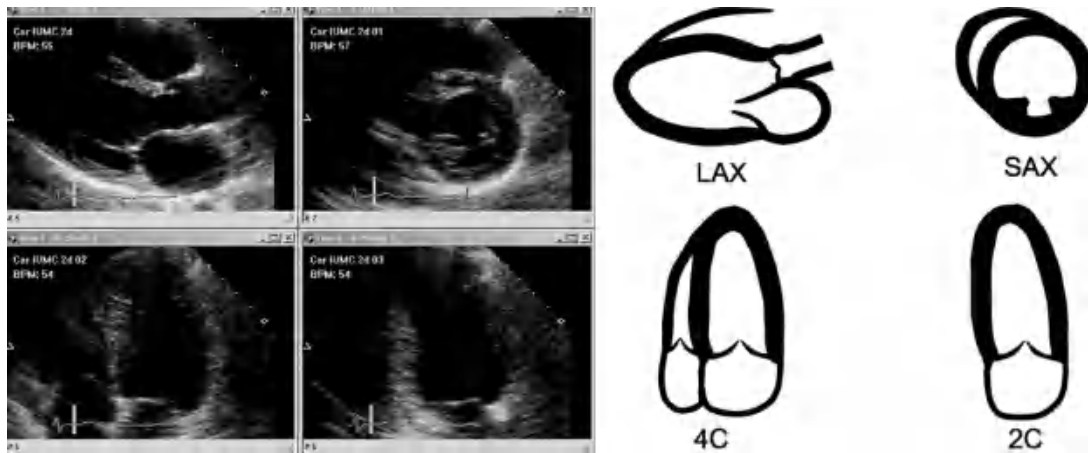


**FIGURE 4.45.** In a quad screen format, the four views most often included are the parasternal long- and short-axis and the apical four- and two-chamber views. LAX, long axis; SAX, short axis; 4C, four chamber; 2C, two chamber.

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.

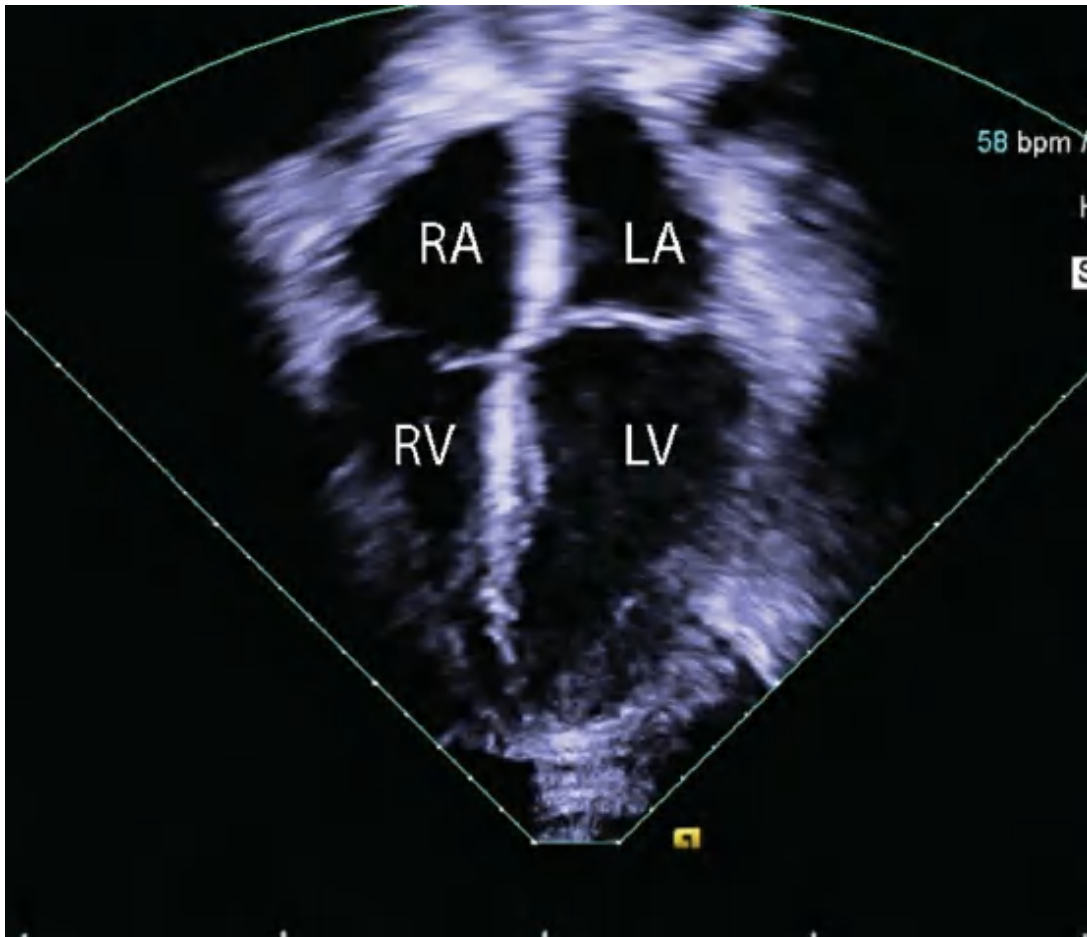
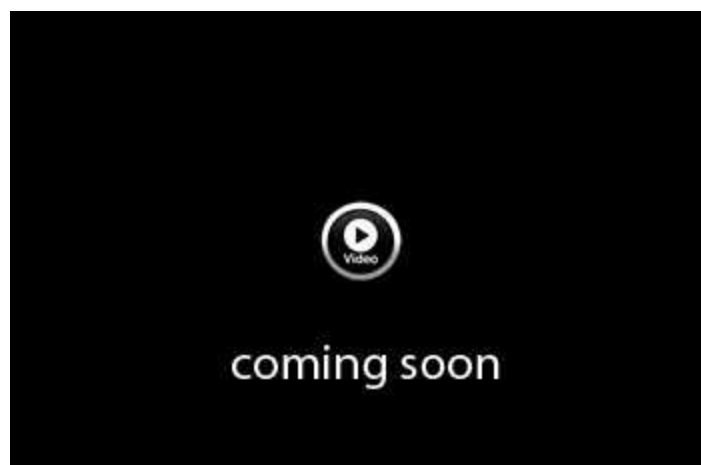


**FIGURE 4.46.** The apical four-chamber view is sometimes displayed with this “anatomically correct” orientation that places the apex down and the atria (RA and LA) above. 📺



**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

| Direct Measurement | Derived Data                                                             |
|--------------------|--------------------------------------------------------------------------|
|                    | Fractional shortening (%)<br>LV mass<br>Stroke volume, aortic valve area |

|                                                                                                                                                                                                                                                                                                                      |                            |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------|
| LVd (minor axis dimension at end diastole)<br>LVs (minor axis dimension at end systole)<br>Left atrial dimension (at end systole)<br>IVSd (interventricular septal wall thickness at end-diastole)<br>PWh (posterior LV wall thickness at end diastole)<br>LVOTd (left ventricular outflow tract dimension, systole) |                            |
| <b>Area measurements</b>                                                                                                                                                                                                                                                                                             |                            |
| SAXd (LV short-axis area at end diastole)                                                                                                                                                                                                                                                                            | Fractional area change (%) |
| SAXs (LV short-axis area at end systole)                                                                                                                                                                                                                                                                             |                            |
| Left atrial area (at end systole)                                                                                                                                                                                                                                                                                    | Left atrial volume         |
| LVVd (left ventricular volume, apical view, end diastole)                                                                                                                                                                                                                                                            |                            |
| LVVs (left ventricular volume, apical view, end systole)                                                                                                                                                                                                                                                             | LV ejection fraction (%)   |
| MVa (mitral valve area, early diastole)                                                                                                                                                                                                                                                                              | Mitral stenosis severity   |

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.

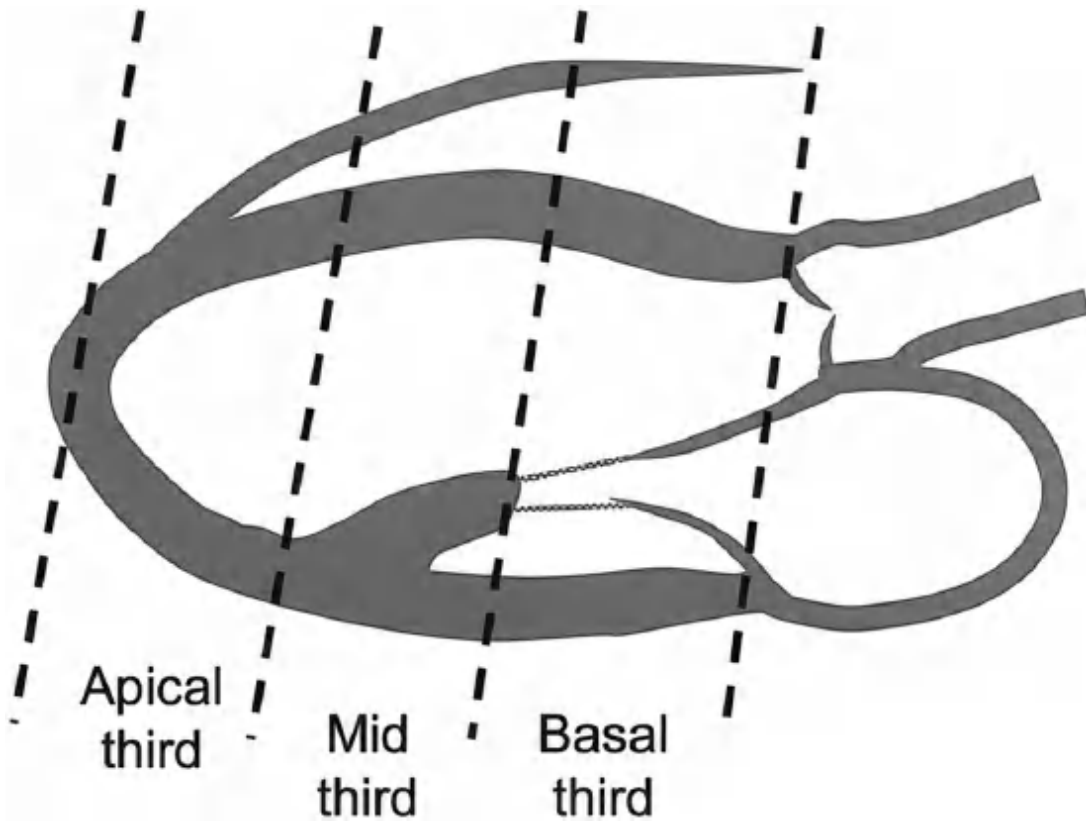


**FIGURE 4.48.** To define the left ventricular segments, it is first necessary to divide the left ventricle into apical, mid, and basal thirds, as shown in the schematic.

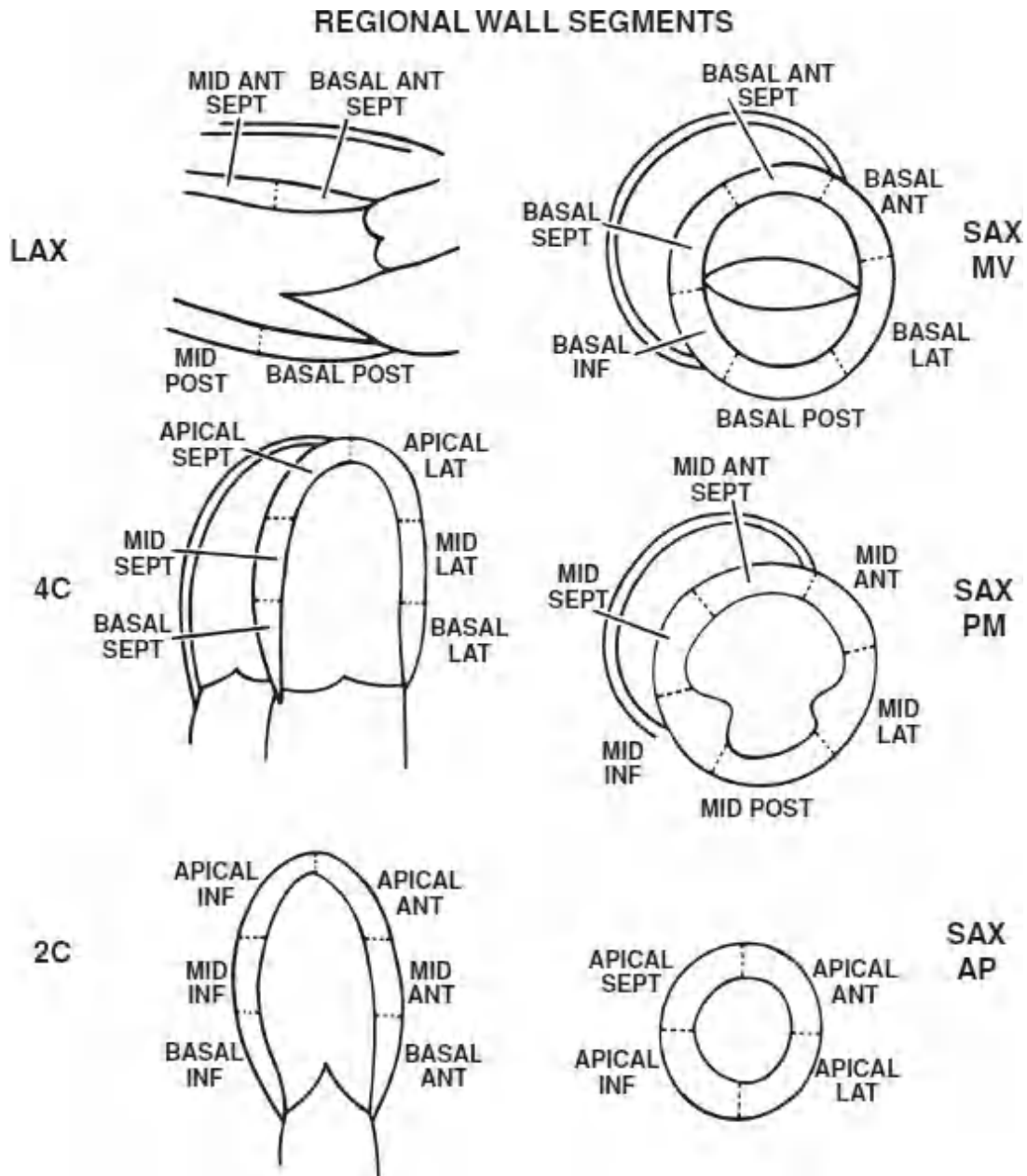


**FIGURE 4.49.** The 16-segment model for left ventricular segmentation. See text for details. LAX, parasternal long axis; 4C, apical four chamber; 2C, apical two chamber; SAX MV, parasternal short-axis view at the level of the mitral valve; SAX PM, parasternal short-axis view at the papillary muscle level; SAX AP, apical short-axis view.

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.

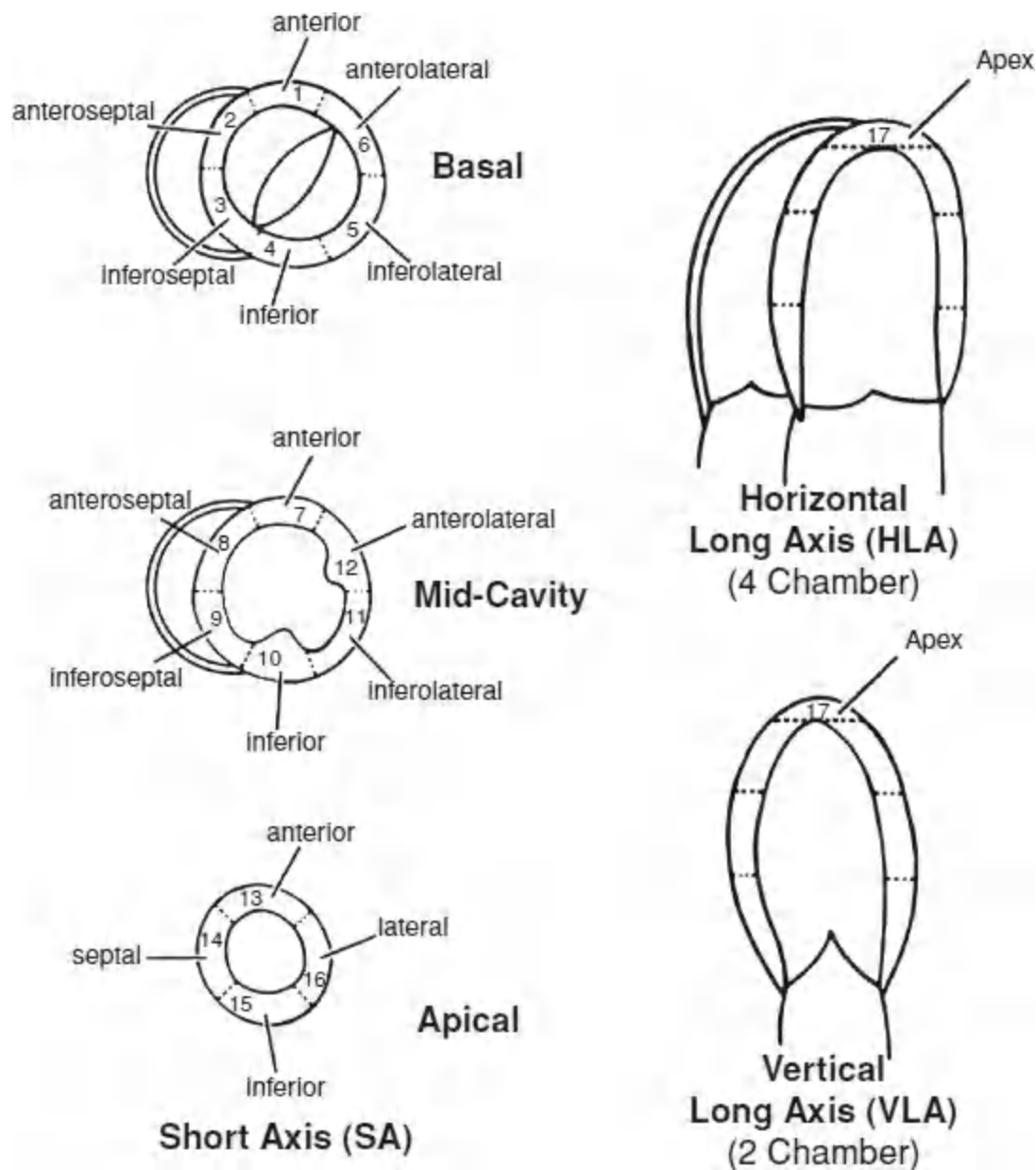


**FIGURE 4.50.** An alternative approach to segmenting the left ventricle suggests identifying the apex as a separate (17th) segment. See text for details. (Reprinted with permission from Cerqueira MD, Weissman NJ, Dilsizian V, et al. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: a statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation*. 2002;105(4):539–542. Copyright © 2002 American Heart Association.

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.

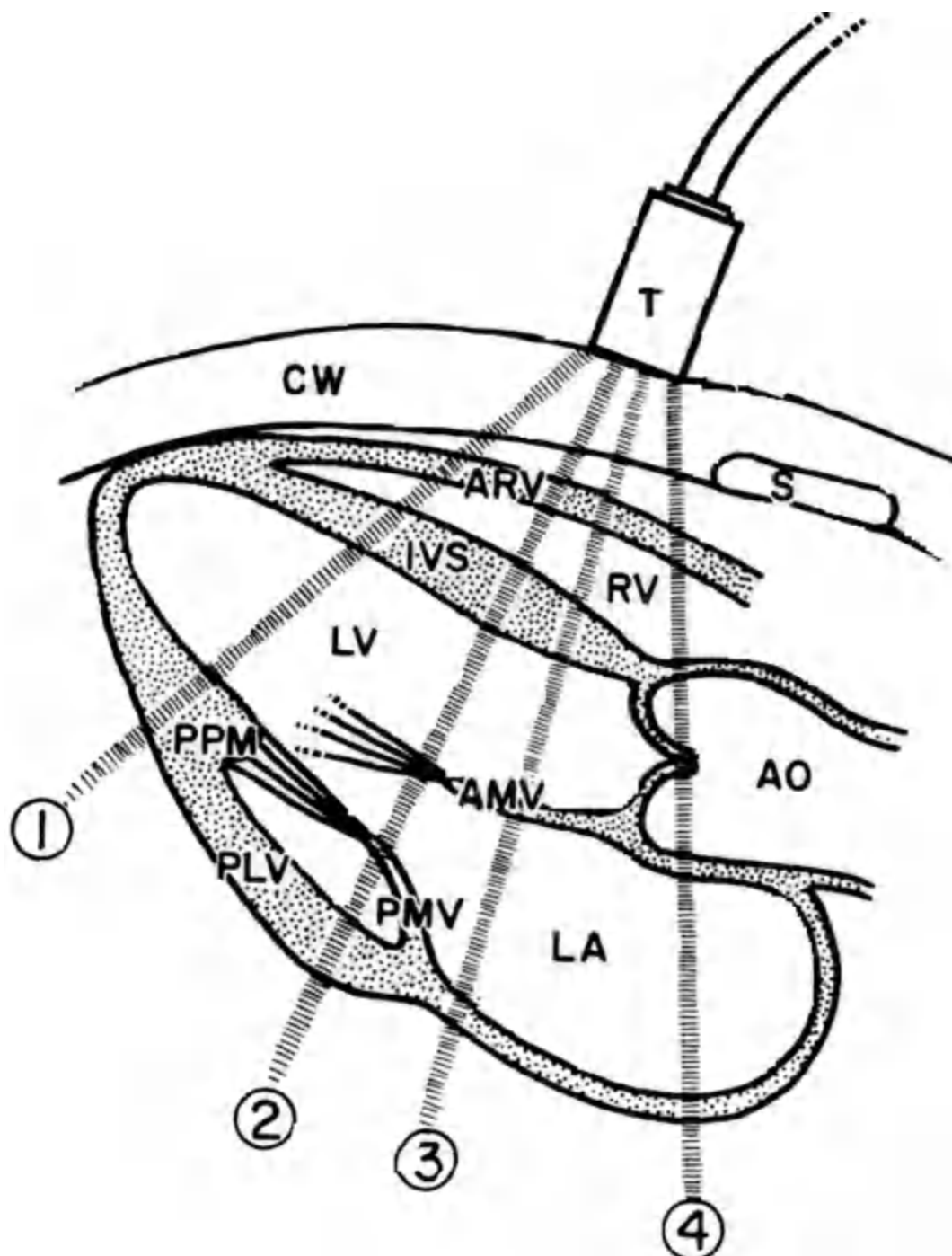


**FIGURE 4.51.** With a transducer (T), placed on the chest wall (CW) in the parasternal window, a variety of possible M-mode views can be recorded. See text for details. ARV, anterior right ventricular wall; AMV, anterior mitral valve leaflet; PMV, posterior mitral valve leaflet; PLV, posterior left 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.)

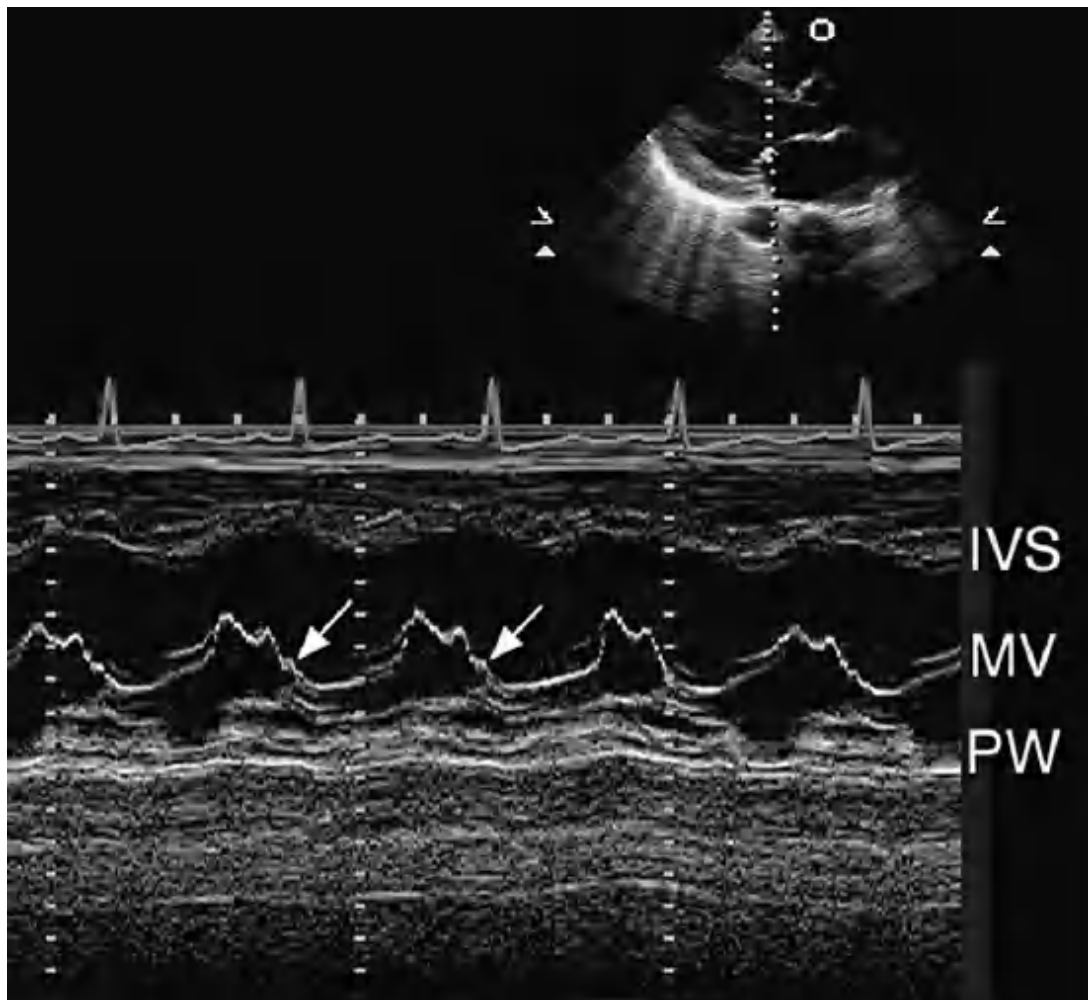


**FIGURE 4.52.** M-mode recording at the level of the mitral valve. A B bump is indicated by the *arrows*. PW, posterior wall.

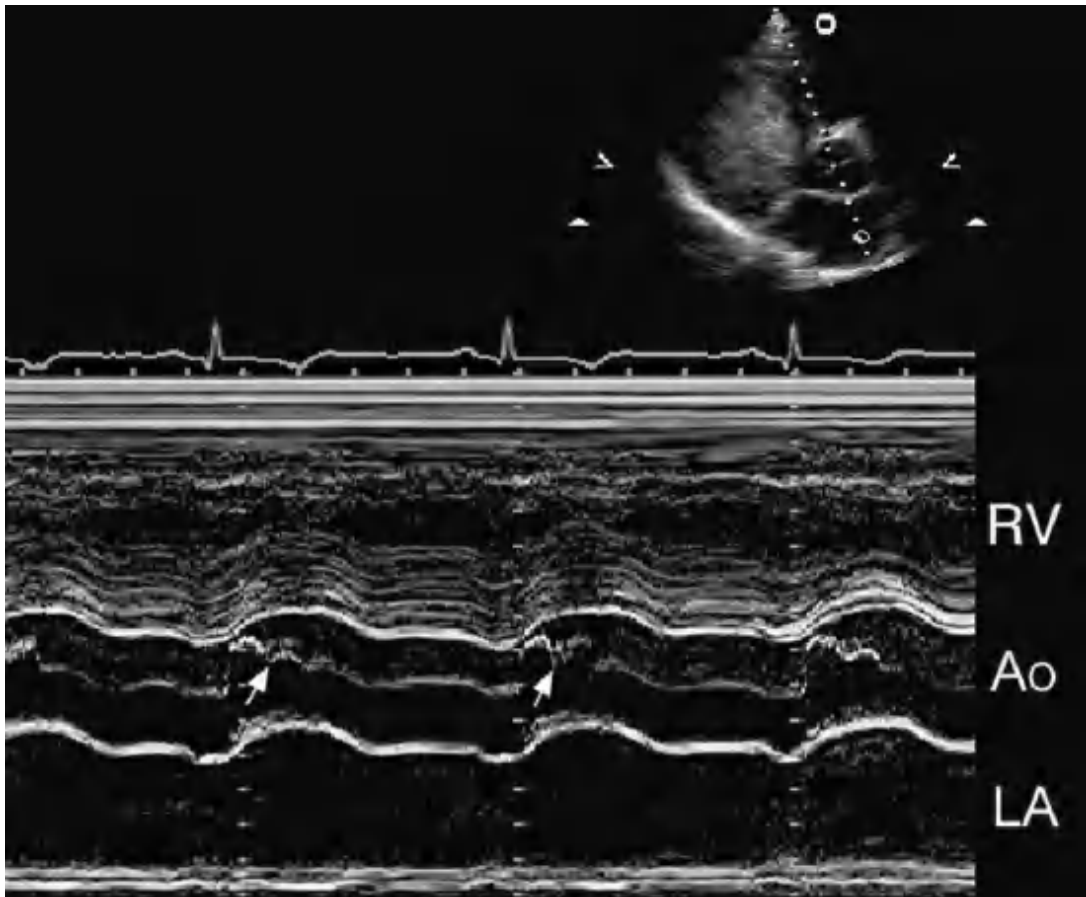


**FIGURE 4.53.** An M-mode recording of the aortic valve, demonstrating partial midsystolic closure of the valve due to subaortic obstruction.

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.

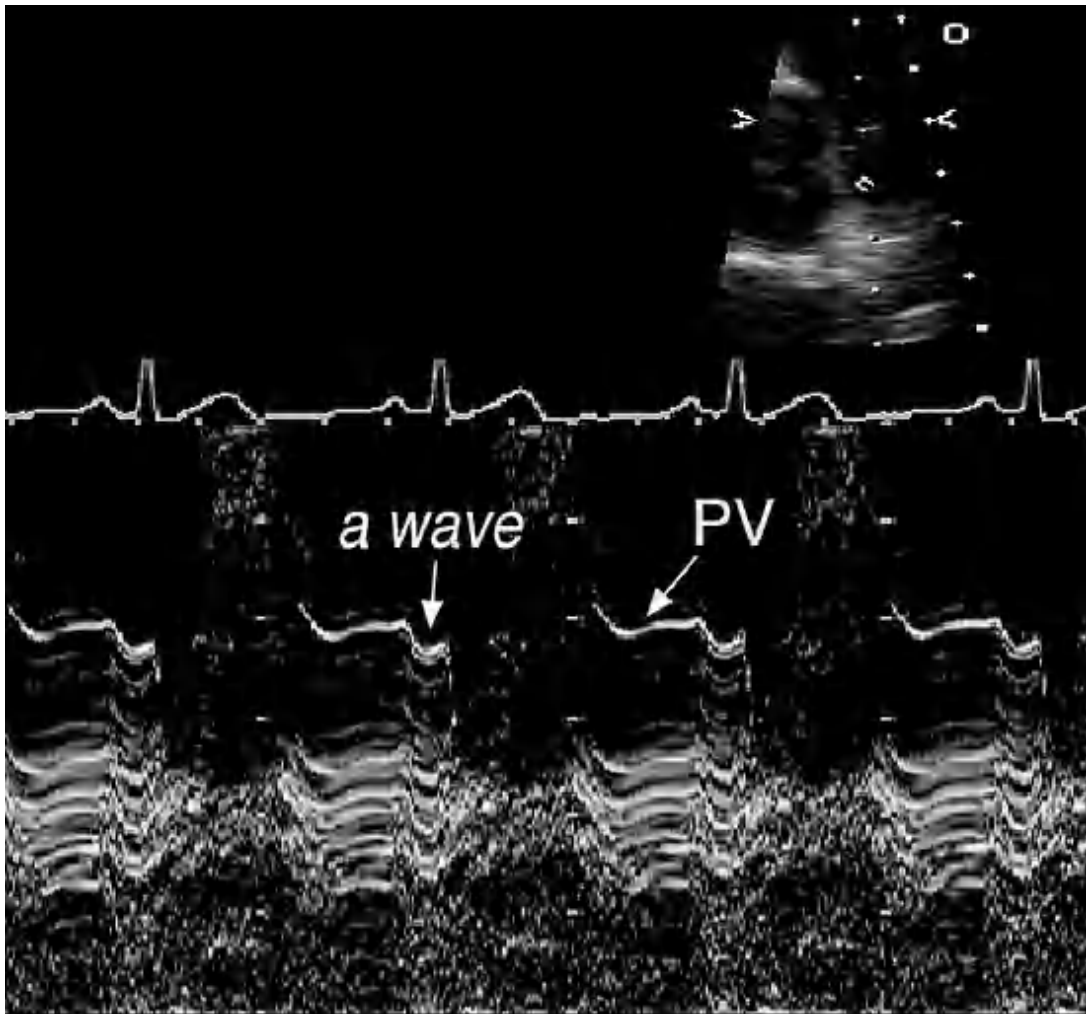


**FIGURE 4.54.** An M-mode recording of pulmonary valve (PV) motion. The A-wave, corresponding to right atrial systole, is indicated.

## TRANSESOPHAGEAL ECHOCARDIOGRAPHY

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

published jointly by the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists (Hahn et al., 2013). The document also discusses training requirements, patient selection, and 3D imaging.

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 4.4**

#### UNIQUE DATA FROM TRANSESOPHAGEAL

#### ECHOCARDIOGRAPHY

##### **Atrial thrombi/masses**

- Left atrial appendage clot
- Left atrial appendage spontaneous contrast
- Clot in body of left atrium
- Right atrial thrombus
- Thrombus/mass on pacemaker wire or indwelling catheter

##### **Mitral valve**



- 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

### **Table 4.5**

## **RISKS AND CONTRAINDICATIONS FOR TRANSESOPHAGEAL ECHOCARDIOGRAPHY**

#### **Topical anesthesia**

- Allergic reactions
- Toxic methemoglobinemia

#### **Conscious sedation**

- Respiratory compromise/hypoxia
- Hypotension
- Paradoxical hypertension
- Paradoxical agitation
- Idiosyncratic reactions

#### **Probe insertion: immediate**

- Oral trauma
- Dental trauma
- Esophageal perforation
- Vagal reaction

#### **Probe insertion: delayed**

- Aspiration

|                                                                                                                                                                            |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Tachycardia<br>Paroxysmal supraventricular tachycardia<br>Ventricular tachycardia                                                                                          |
| <b>Contradictions</b><br>Absolute<br>Recent esophageal trauma/surgery<br>Recent esophageal bleed<br>Relative<br>Poorly cooperative patient<br>Remote esophageal procedures |

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.



**FIGURE 4.55.** Patient, sonographer, nurse, and physician positioning for performing transesophageal echocardiography. See text for details.

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.

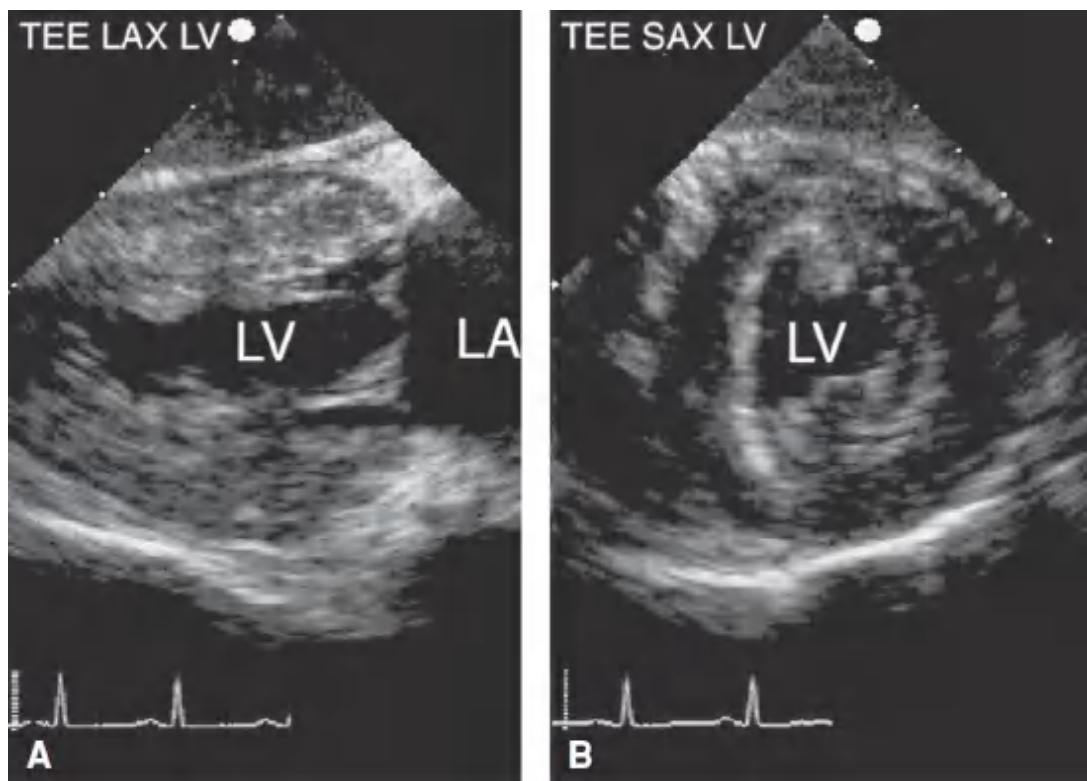


**FIGURE 4.56.** A biplane transesophageal echocardiogram. **A:** The long-axis view of the left ventricle with the transducer located in the patient’s stomach. **B:** The orthogonal view demonstrating a short-axis plane. TEE, transesophageal echocardiography. 📺



**Video 4-56**



**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.

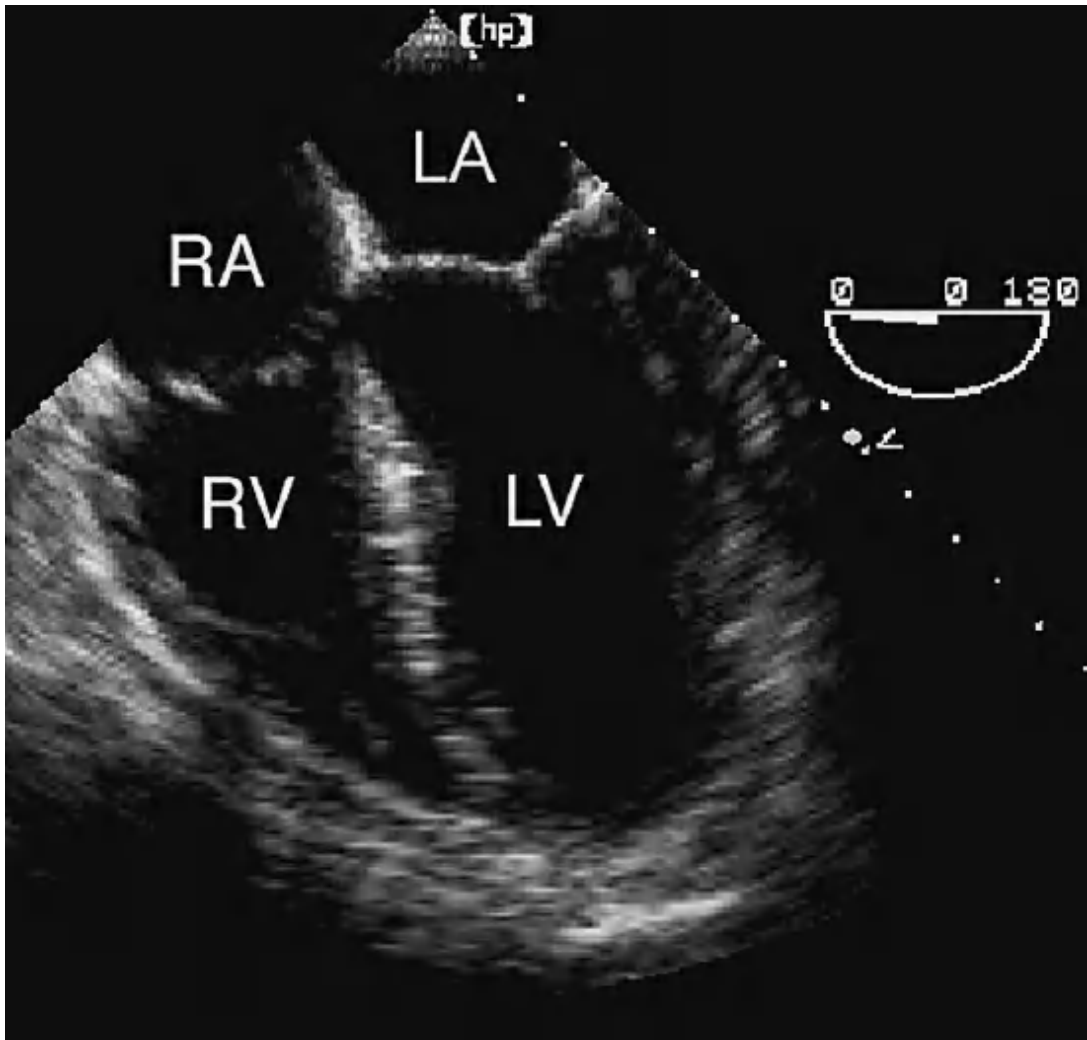
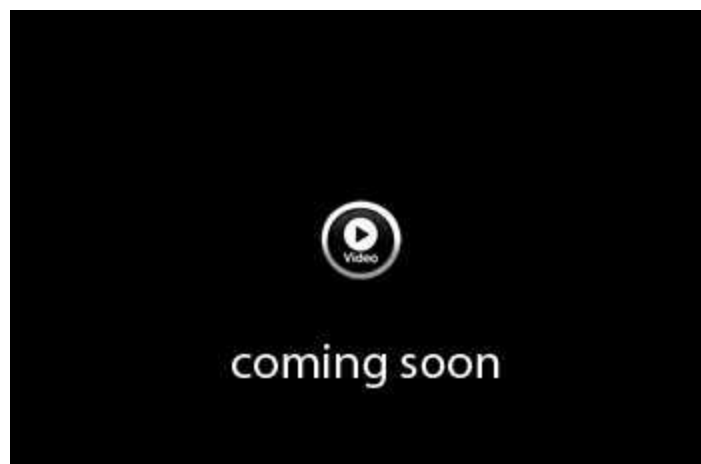


**FIGURE 4.58.** A four-chamber view from the mid-esophagus is displayed, illustrating the close proximity of the esophagus to the LA. 🇧🇪



**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).

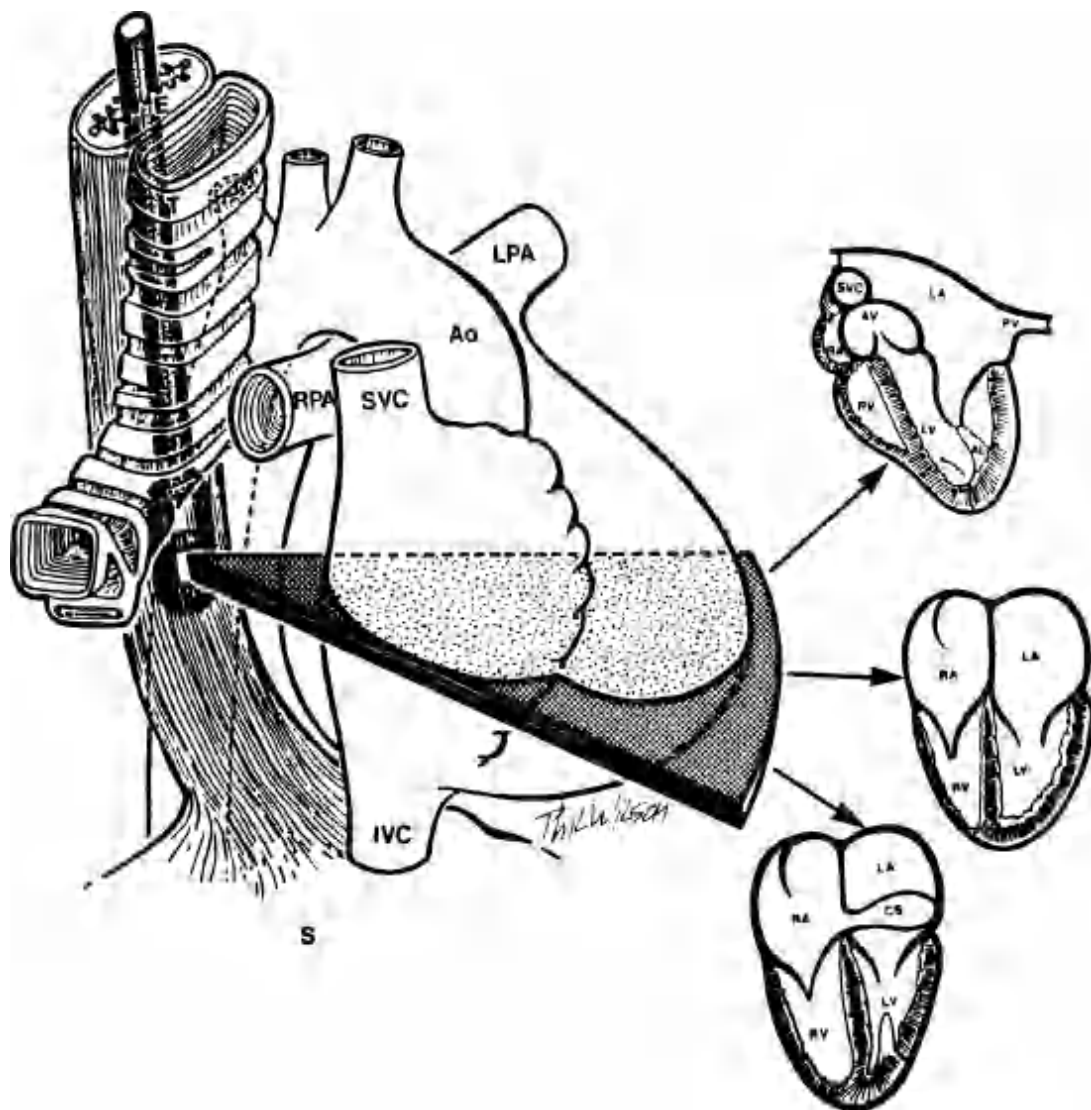


**FIGURE 4.59.** Three of the echocardiographic views that can be obtained with the horizontal probe in the midesophageal location. LPA, left pulmonary artery; RPA, right pulmonary artery; S, stomach; PV, pulmonary vein; CS, coronary sinus.

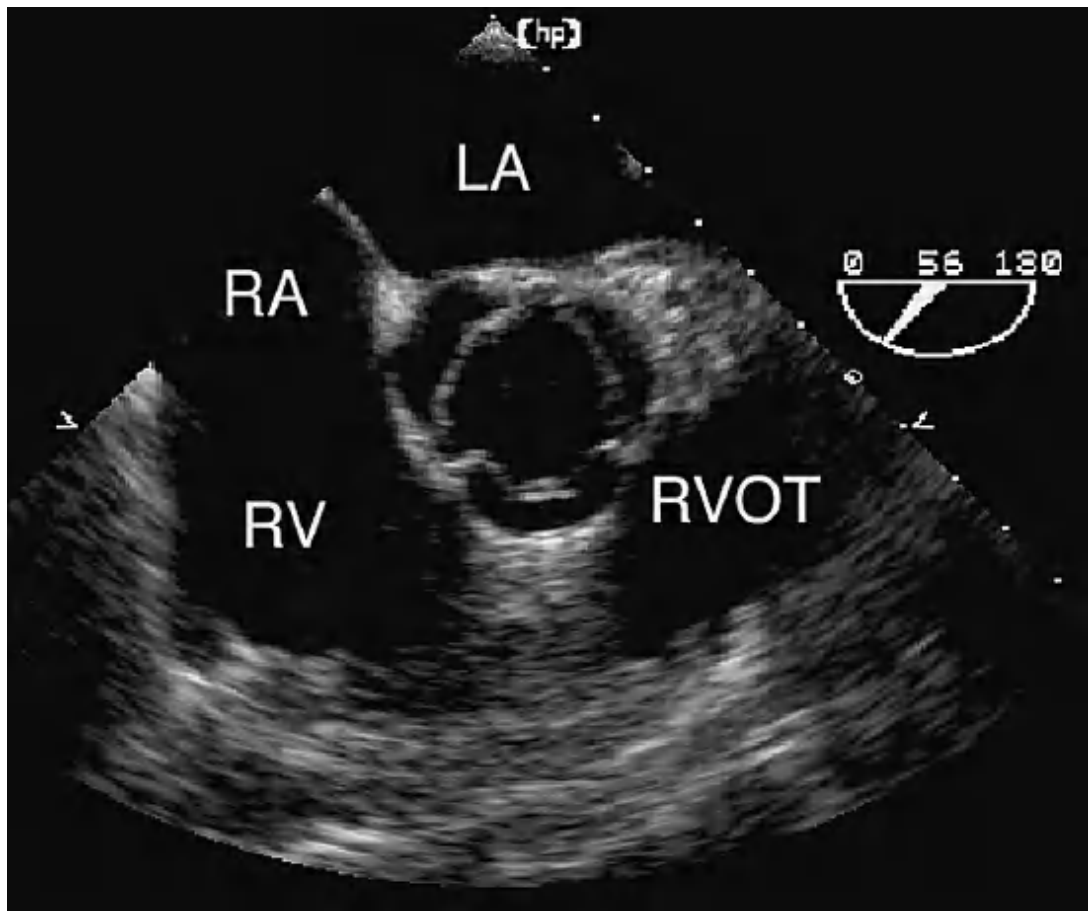
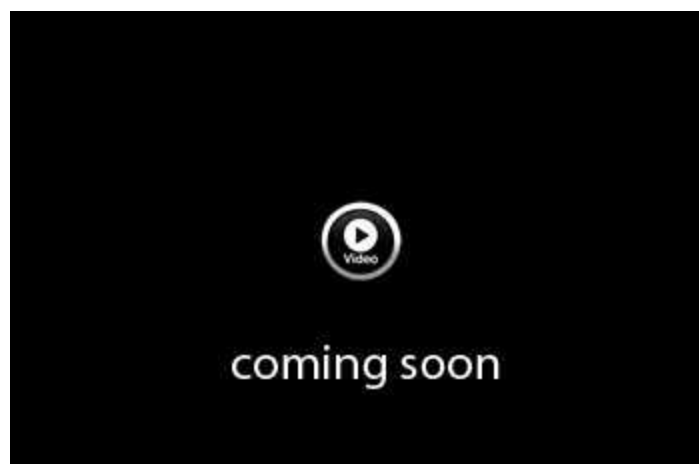


**FIGURE 4.60.** From the esophagus, the probe can be flexed to yield a basal short-axis projection. 



**Video 4-60**

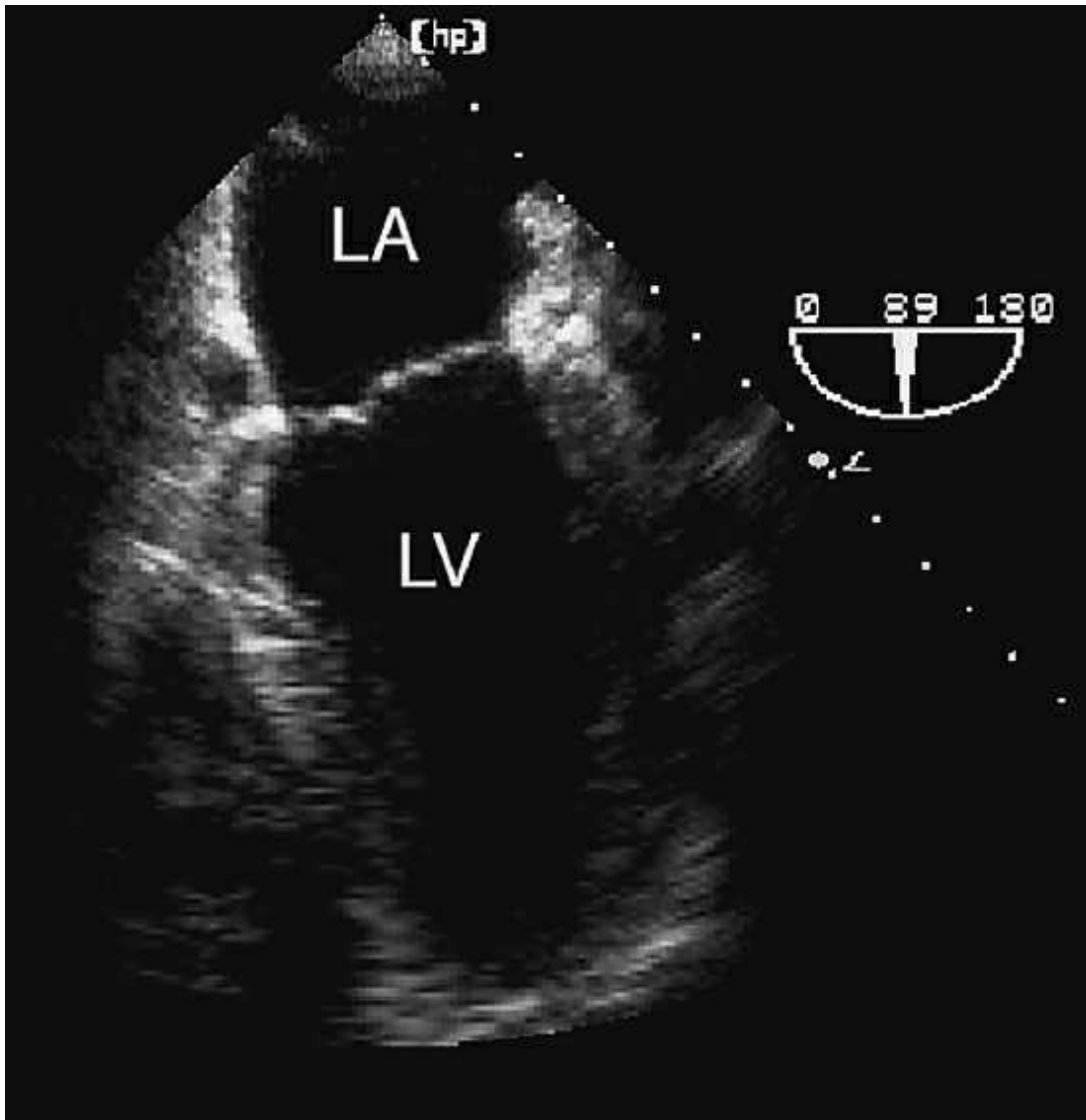


**FIGURE 4.61.** Four of the short-axis views that can be obtained with the horizontal probe in the upper esophagus. S, stomach; LUPV, left upper pulmonary vein; RUPV, right upper pulmonary vein; LAA, left atrial appendage; RAA, right atrial appendage; RLPV, right lower pulmonary vein; LLPV, left lower pulmonary vein; LCA, left coronary artery; RCA, right coronary artery; FO, foramen ovale; N, noncoronary cusp; R, right coronary cusp.

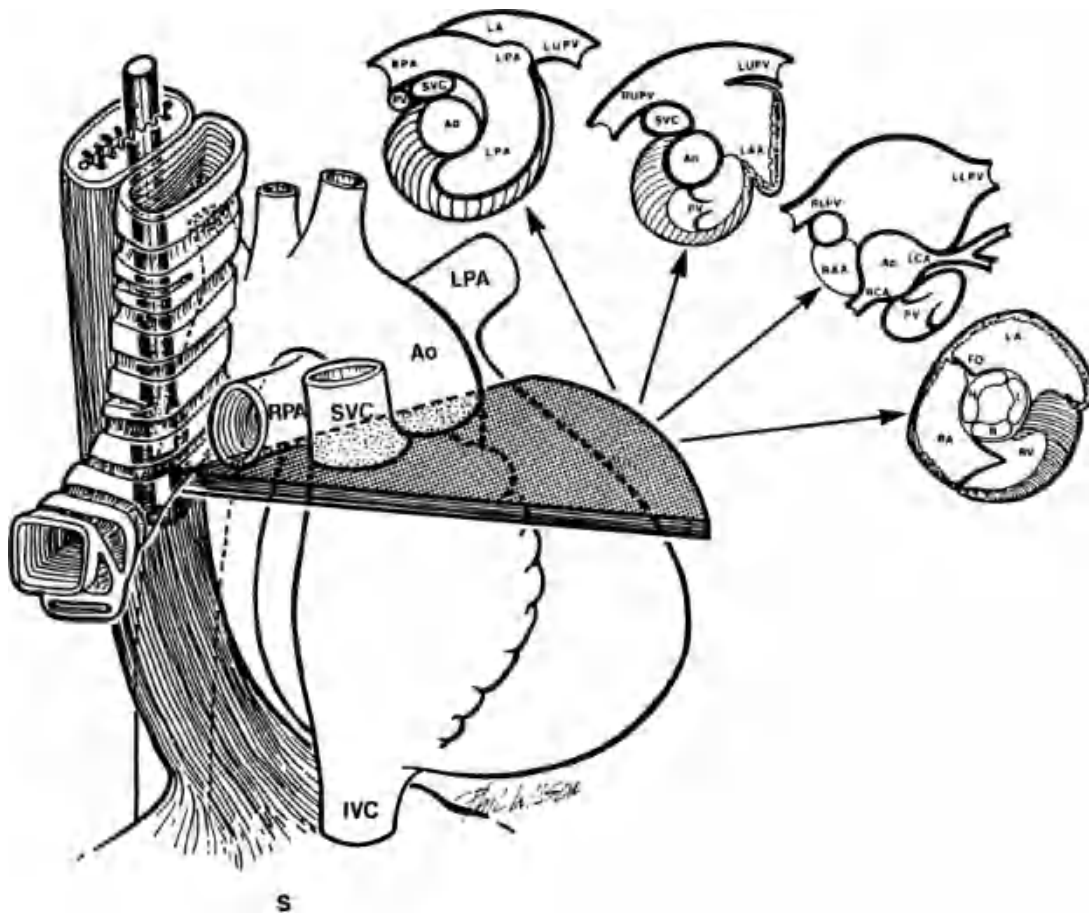
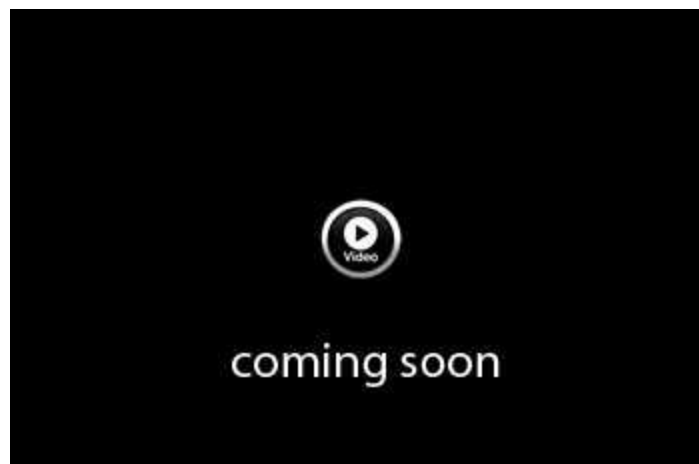


**FIGURE 4.62.** By adjusting the array angle to approximately 90 degrees, a two-chamber view is recorded. 



### 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.

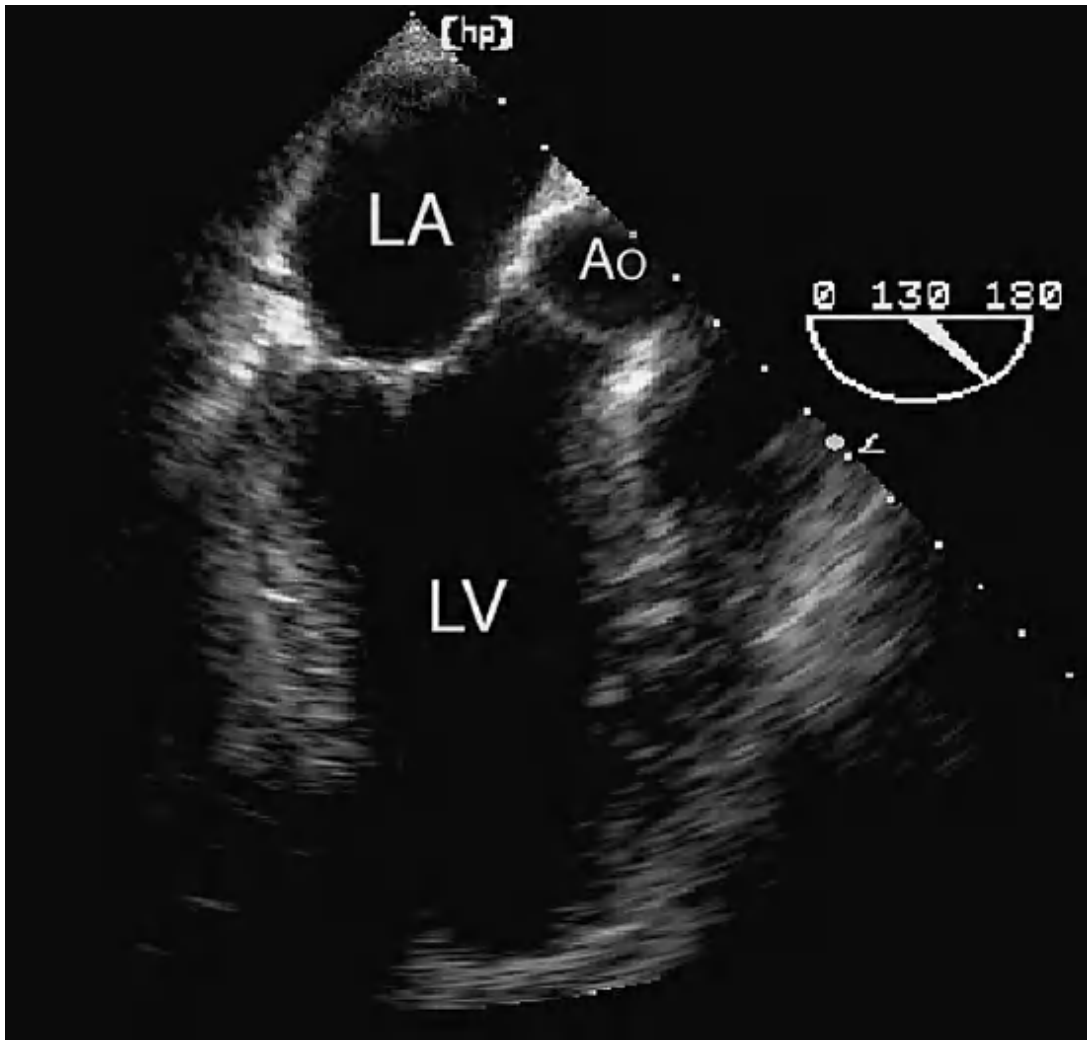


**FIGURE 4.63.** By increasing the array angle to approximately 130 degrees, the left ventricular outflow tract, aortic valve, and proximal aorta can be included in the plane, yielding a long-axis view.

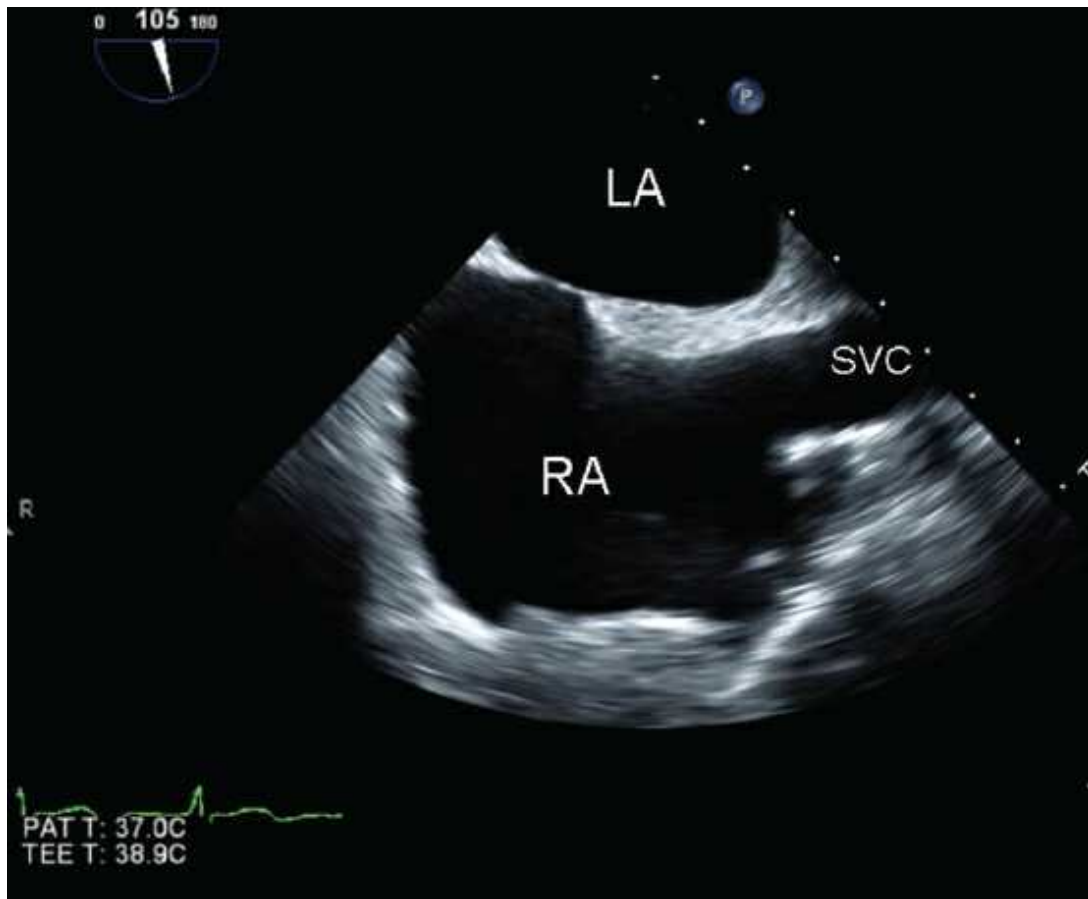


**FIGURE 4.64.** With the probe relatively high within the esophagus, a vertical plane allows both atria and the interatrial septum to be recorded. This plane is called the bicaval view and also records the entrance of the superior vena cava (SVC) into the RA.

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.

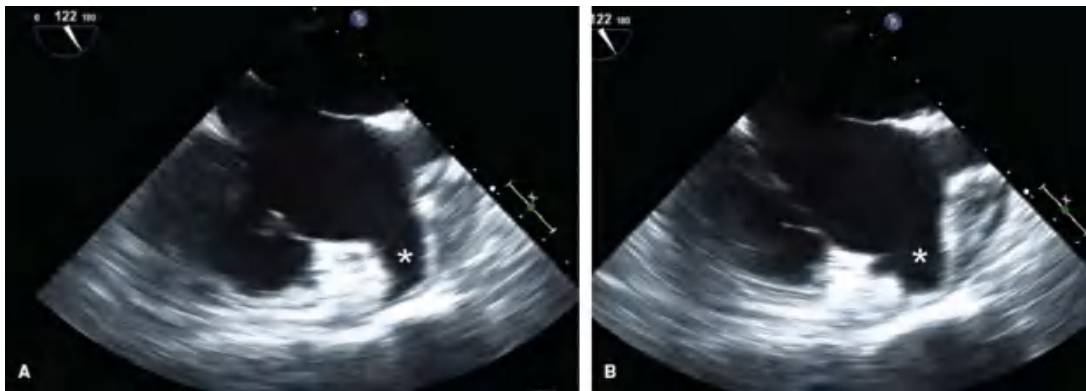
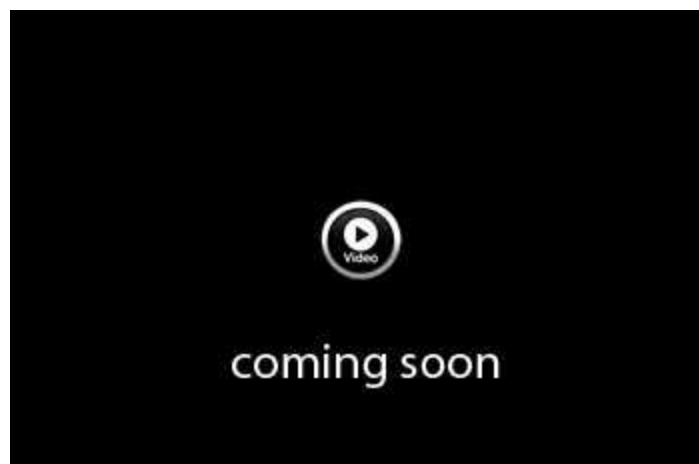


**FIGURE 4.65.** From the vertical plane, the right atrial appendage (\*) can be visualized (A). By slight adjustment of the probe (B), the multilobed shape of the structure is demonstrated. 



**Video 4-65**

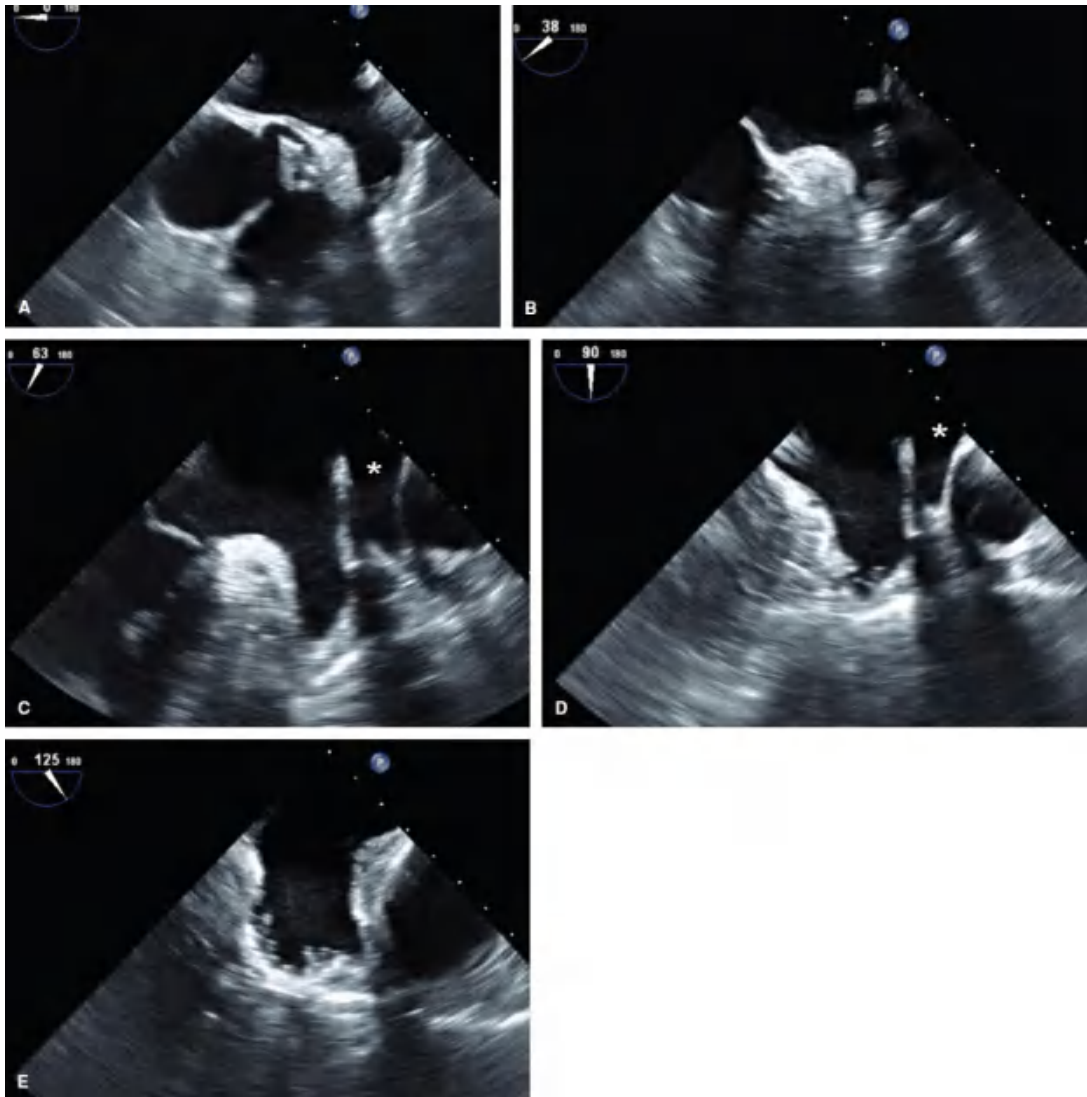


**FIGURE 4.66.** Five views (A–E) of the left atrial appendage are recorded at various angles of rotation of the scan plane. Note how the appearance of the appendage varies in the different views. The relationship of the appendage to the left upper pulmonary vein (\*) is also demonstrated.

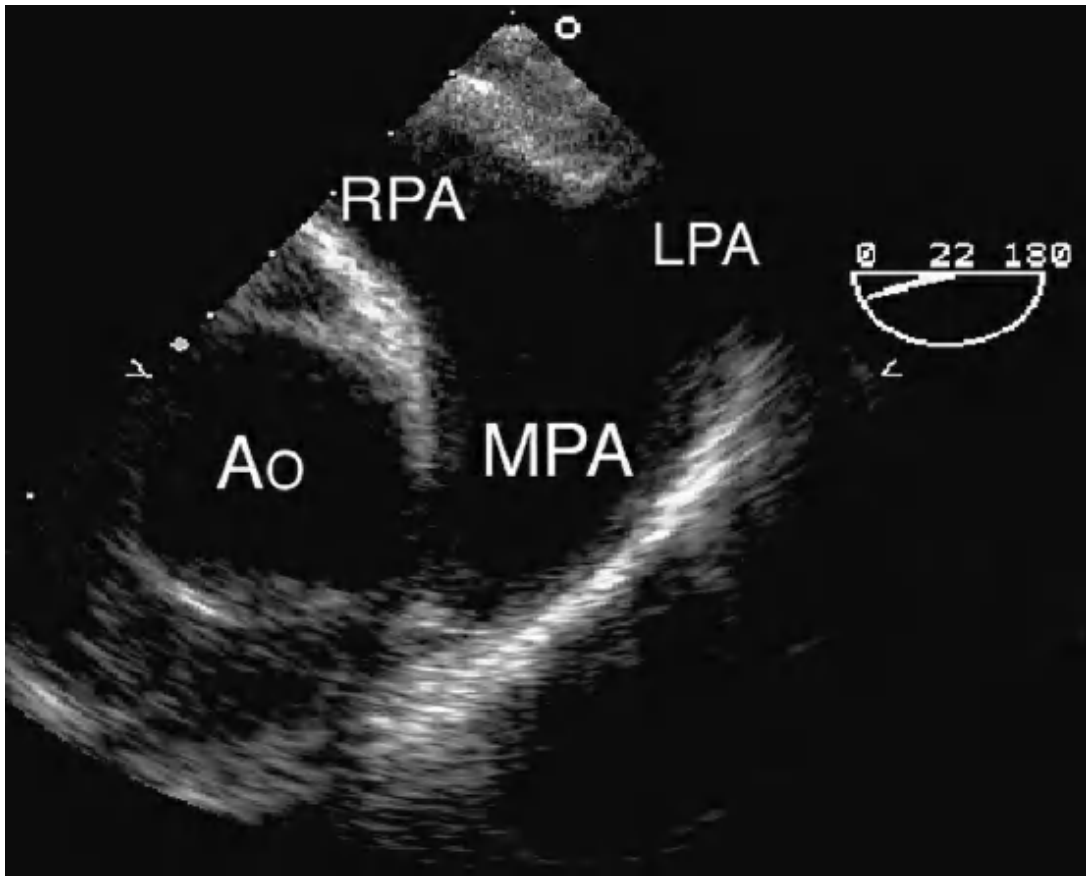


**FIGURE 4.67.** From a high esophageal position, the horizontal plane will permit the relationship between the main pulmonary artery (MPA) and aorta to be recorded. This view also allows the bifurcation of the MPA into the right pulmonary artery (RPA) and left pulmonary artery (LPA) to be demonstrated. 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](#)).

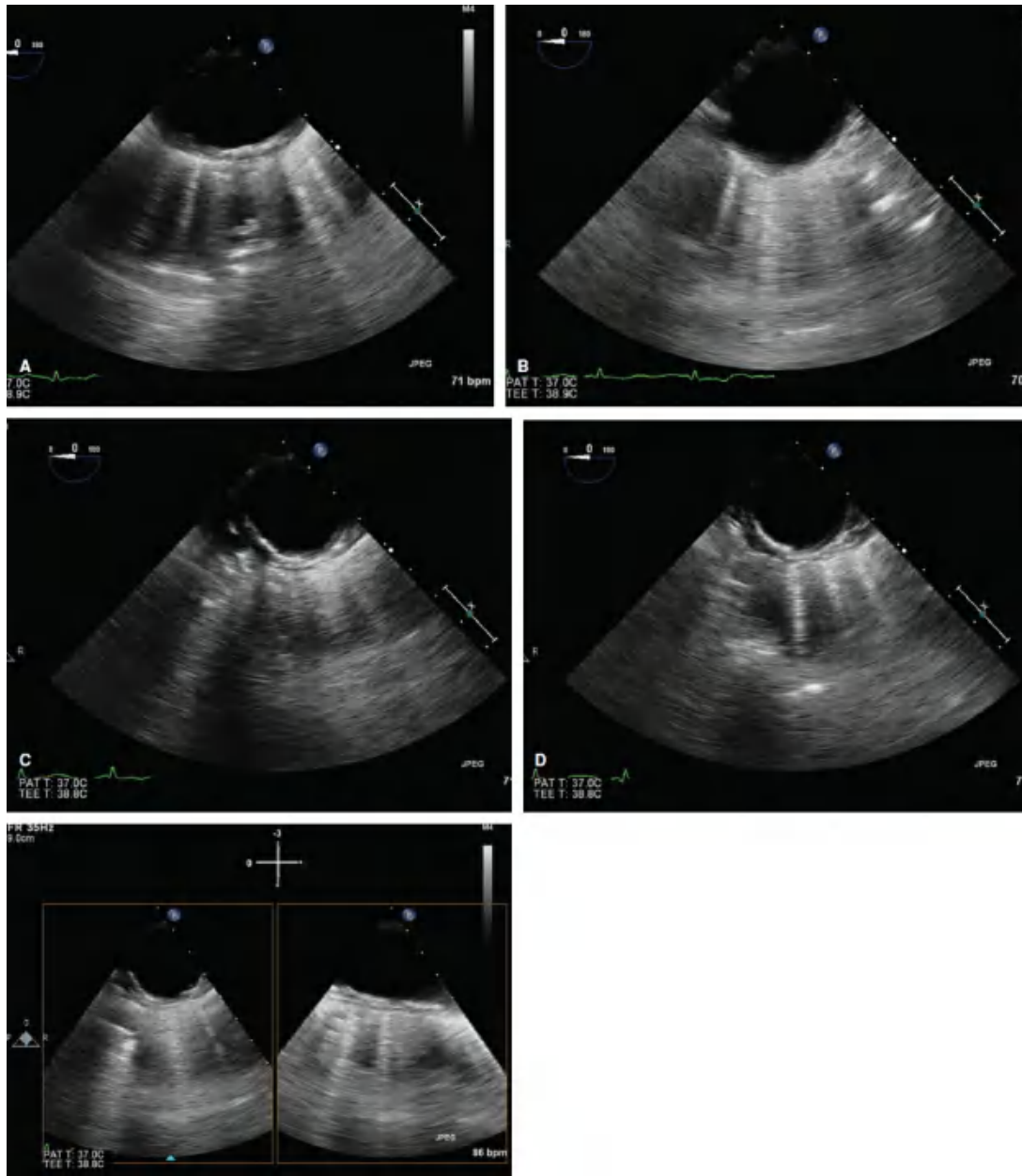


**FIGURE 4.68.** The descending aorta is recorded in a series of short-axis views at various levels by withdrawing the transducer from the level of the diaphragm to the distal arch (panels **A–D**). In panel **E**, a biplane recording of the proximal thoracic aorta is shown, demonstrating short- and 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.

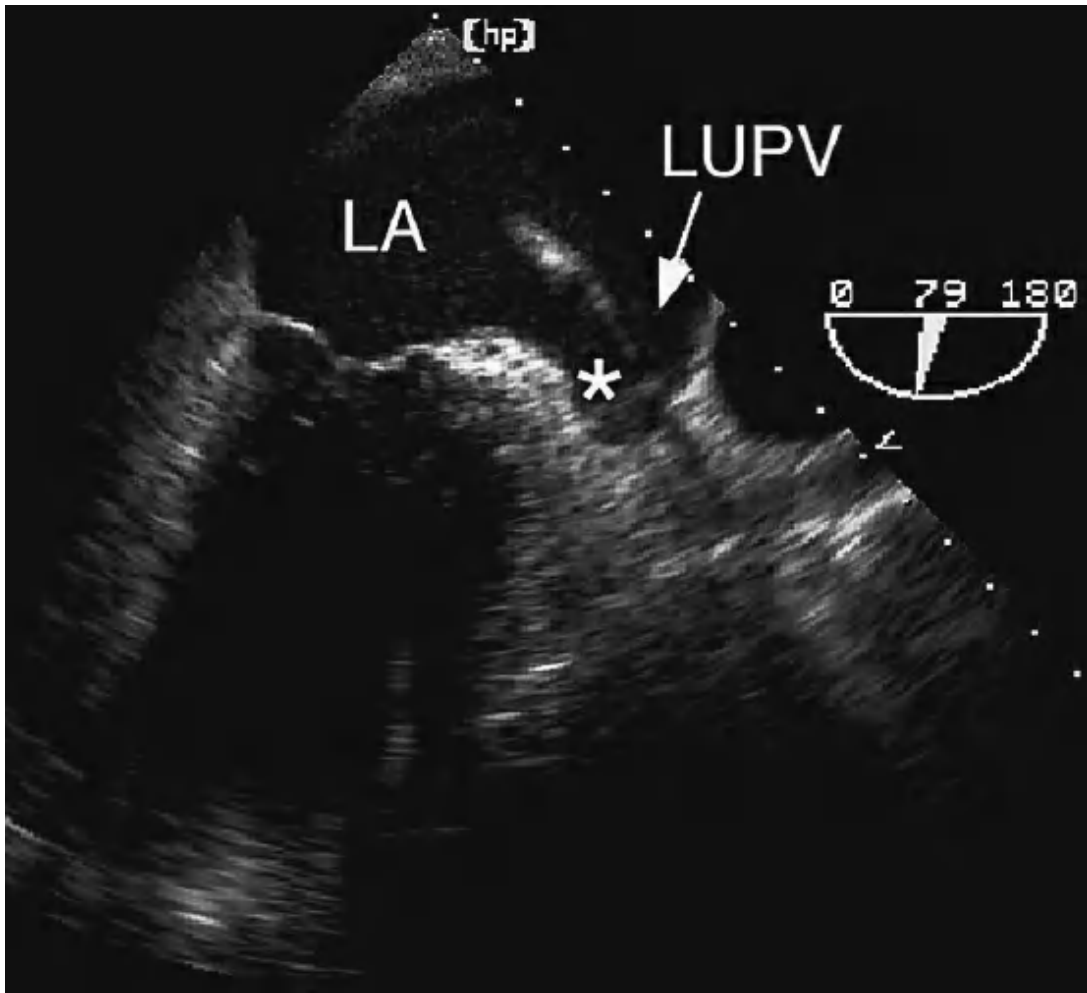
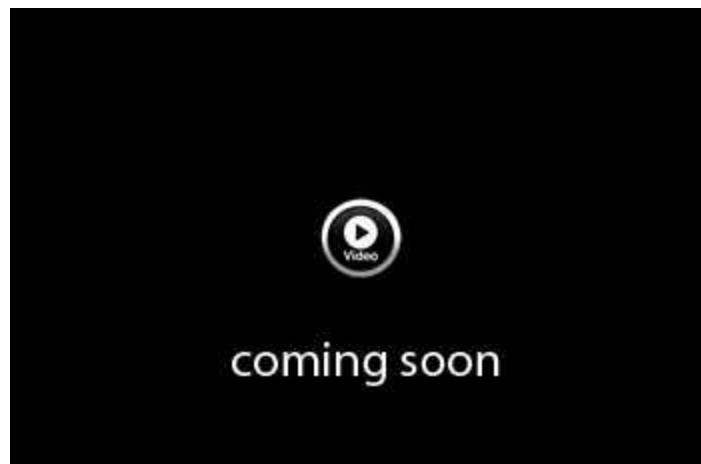


**FIGURE 4.70.** This view demonstrates the relationship between the left atrial appendage (\*) and the left upper pulmonary vein (LUPV) from the two-chamber view. 



**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 multibeam 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.



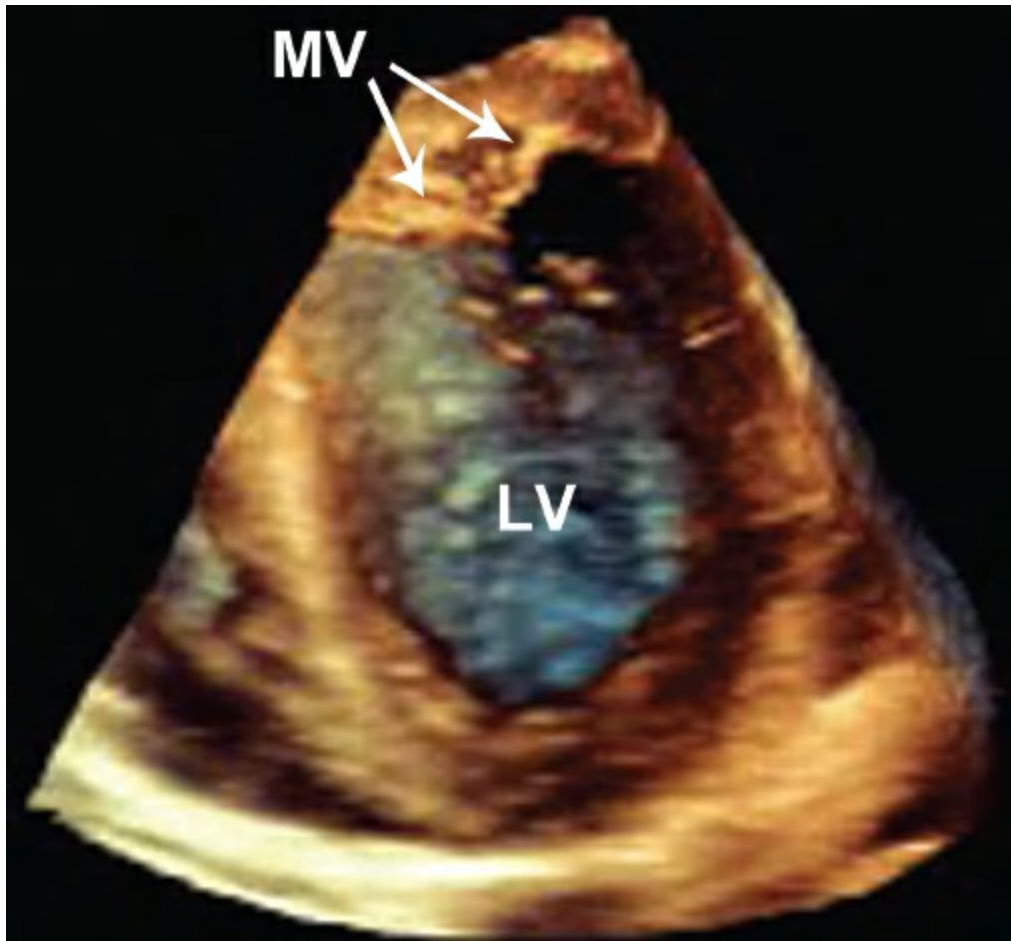
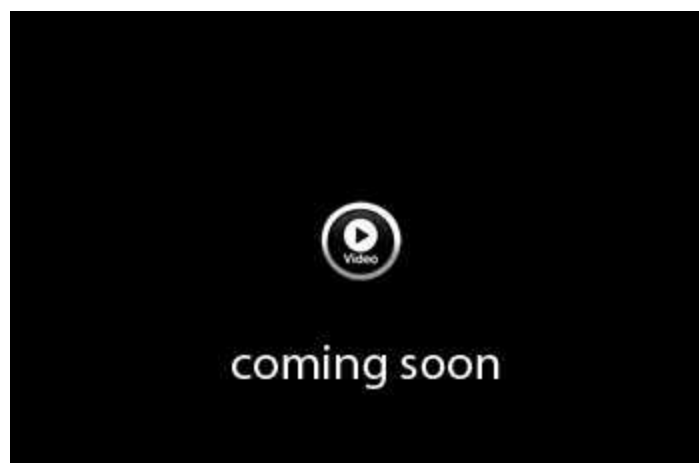


**FIGURE 4.71.** An example of a transesophageal three-dimensional echocardiogram. The MV and left ventricle are shown. A portion of the left ventricular wall is removed to show the cavity of the chamber. 



**Video 4-71**

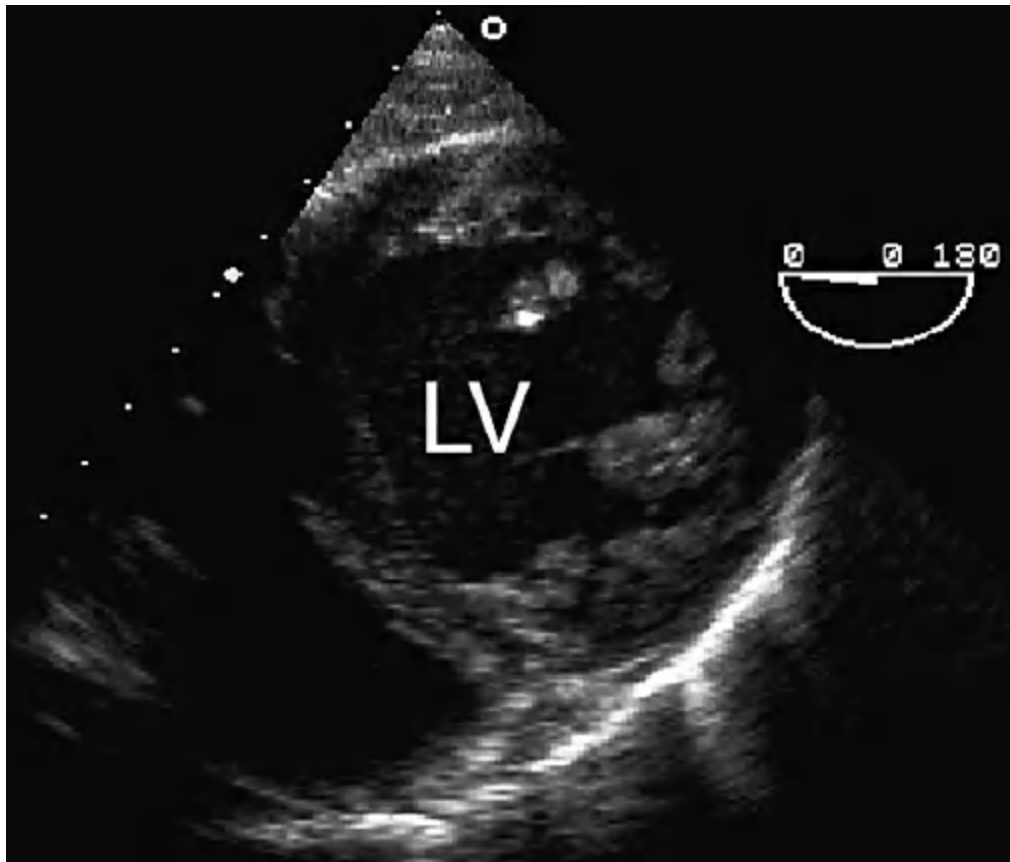


**FIGURE 4.72.** Transgastric imaging demonstrates a short-axis view at the mid left ventricular level.

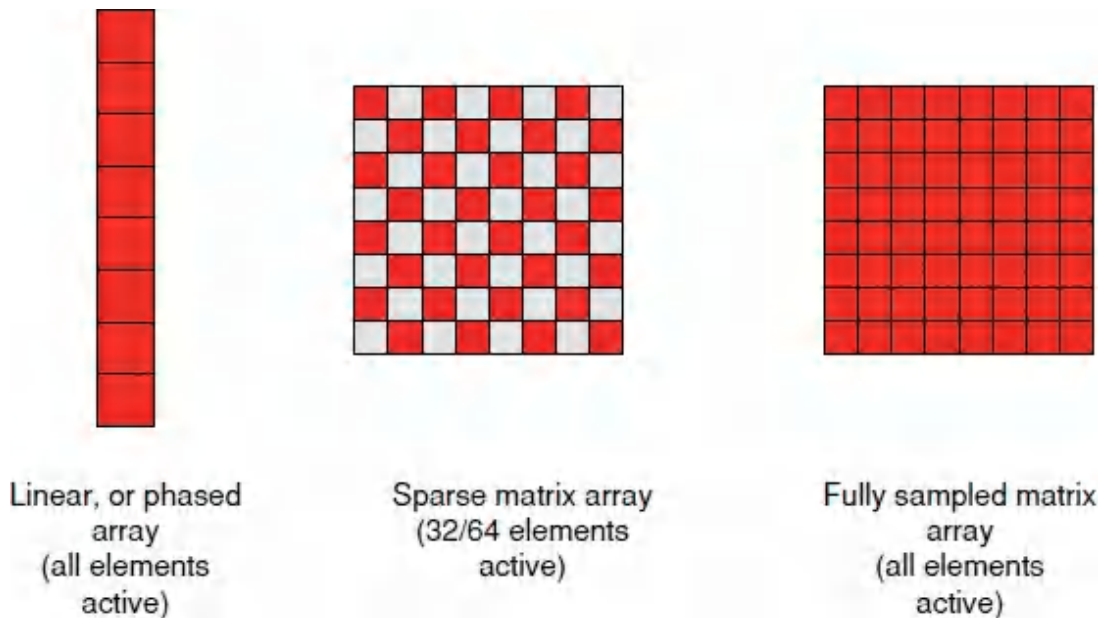


**FIGURE 4.73.** The piezoelectric crystals in an ultrasound transducer can be arranged linearly (**left panel**), or in a two-dimensional array. The middle panel demonstrates a sparsely sampled matrix array in which half of the elements are

active. In the right panel, a fully sampled array, in which all elements are active, is shown. See text for details.

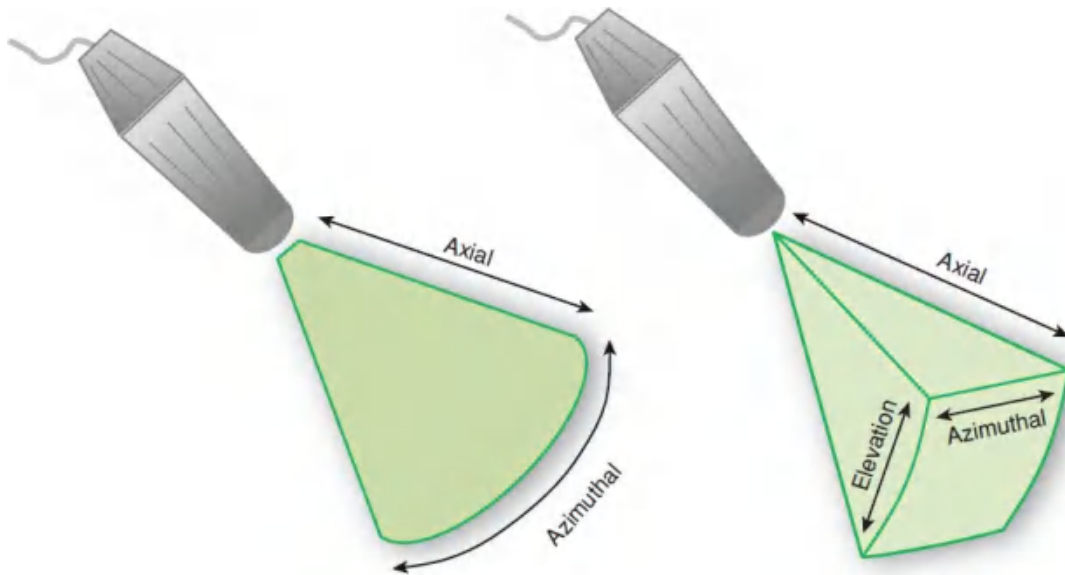


**FIGURE 4.74.** The fundamental difference between two-dimensional and three-dimensional scanning is illustrated. On the **left**, a phased array probe creates a 2D sector scan. On the **right**, the third dimension is added to create a volumetric data set, from which real-time 3D imaging is derived. See text for details.

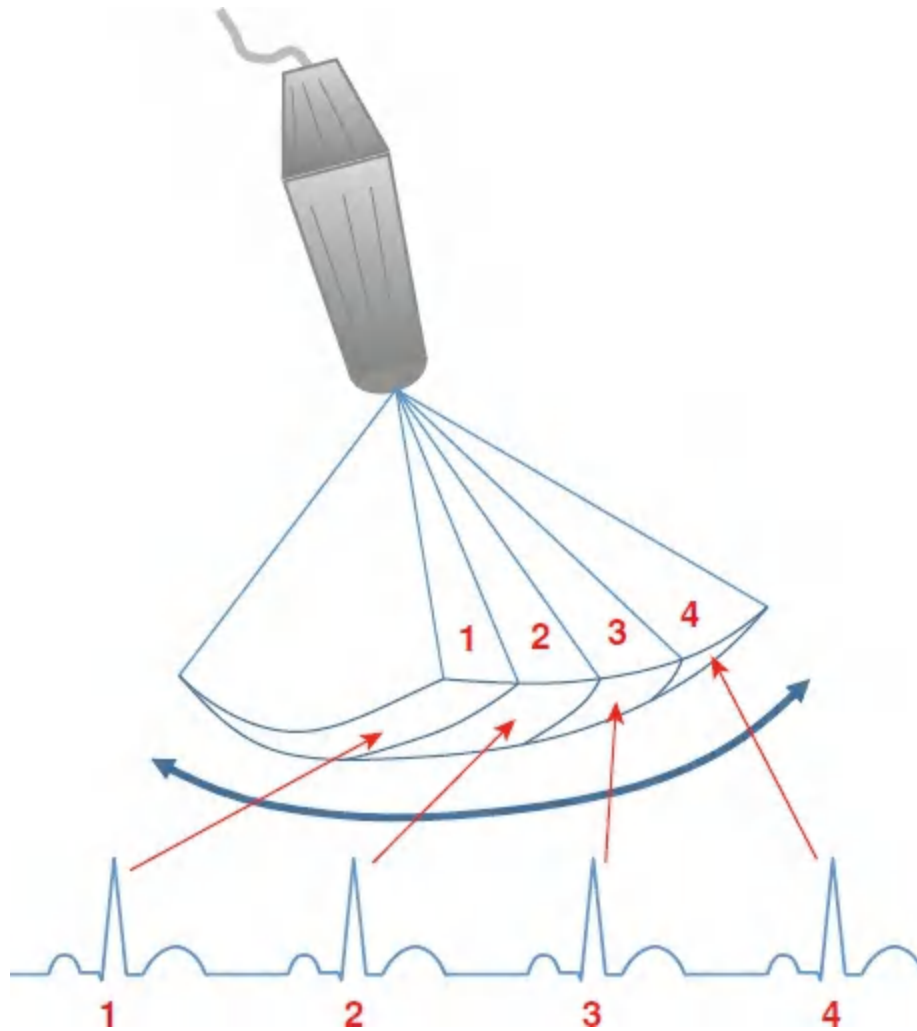


**FIGURE 4.75.** The concept of multibeam acquisition is demonstrated, in which the pyramidal volume of ultrasound data is built up from partial sampling of four sequential cardiac cycles.

A second option is multibeam 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.

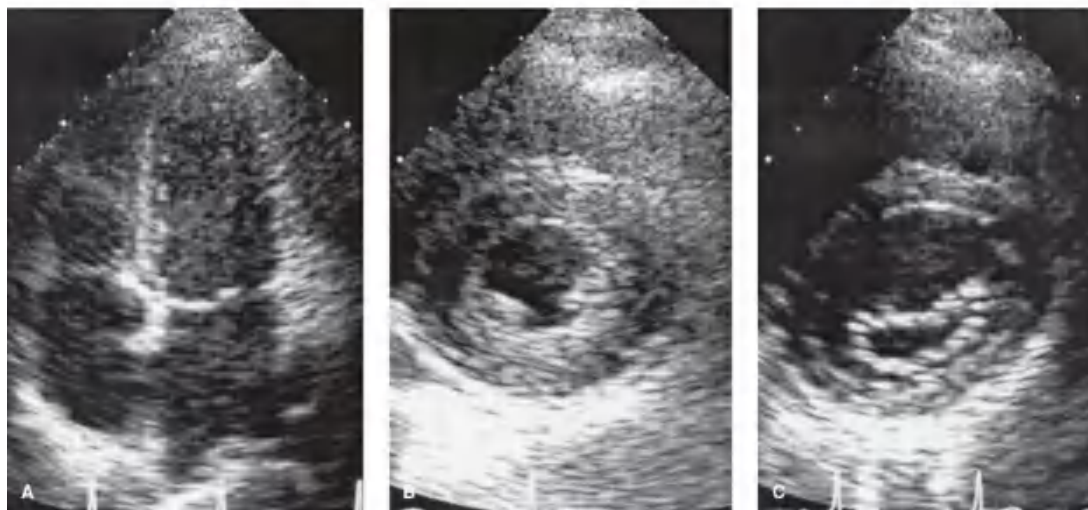
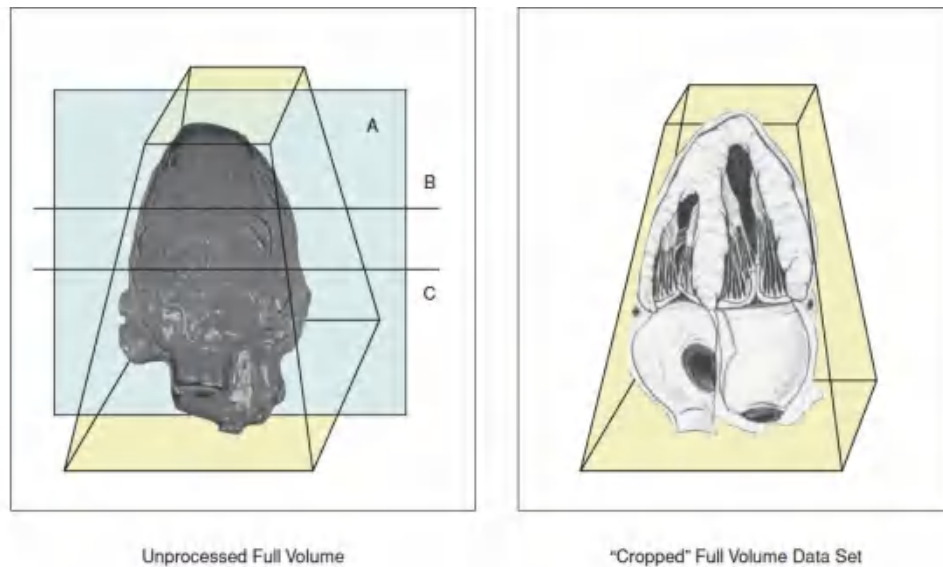


**FIGURE 4.76.** Schematic demonstrates various methods for processing real-time three-dimensional full-volume data sets. **A:** Schematic represents the total volume in a pyramidal scan that contains all four cardiac chambers. Note that distinct cardiac structures are not visualized. **B:** A “cropped” 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  $\times$  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.

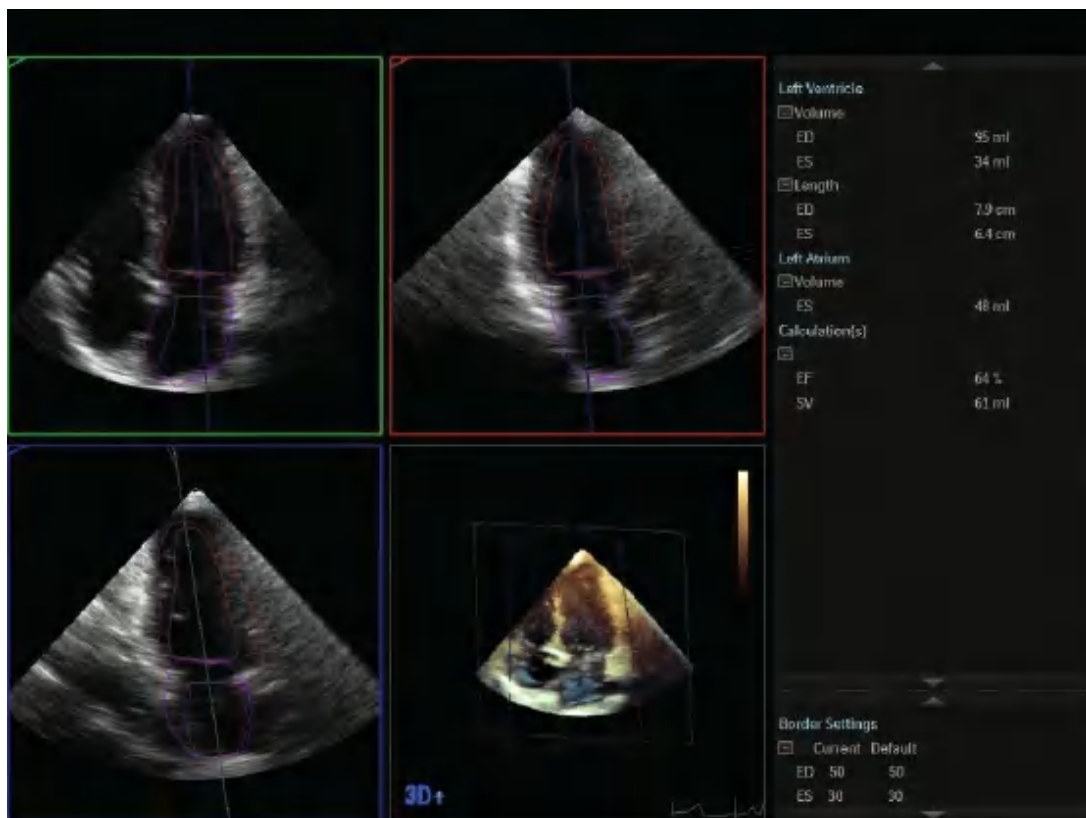
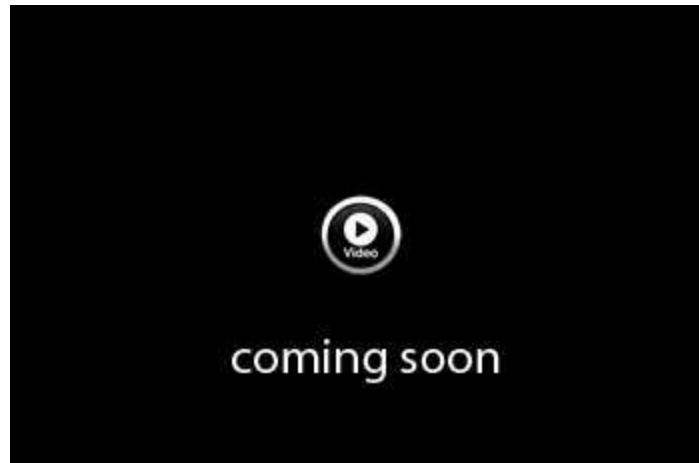


**FIGURE 4.77.** Multiplane imaging involves the simultaneous recording of (in this case) three two-dimensional images, from which a volumetric reconstruction of the left ventricle is created. Volumes and ejection fraction can then be calculated.







### **Video 4-77**

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.

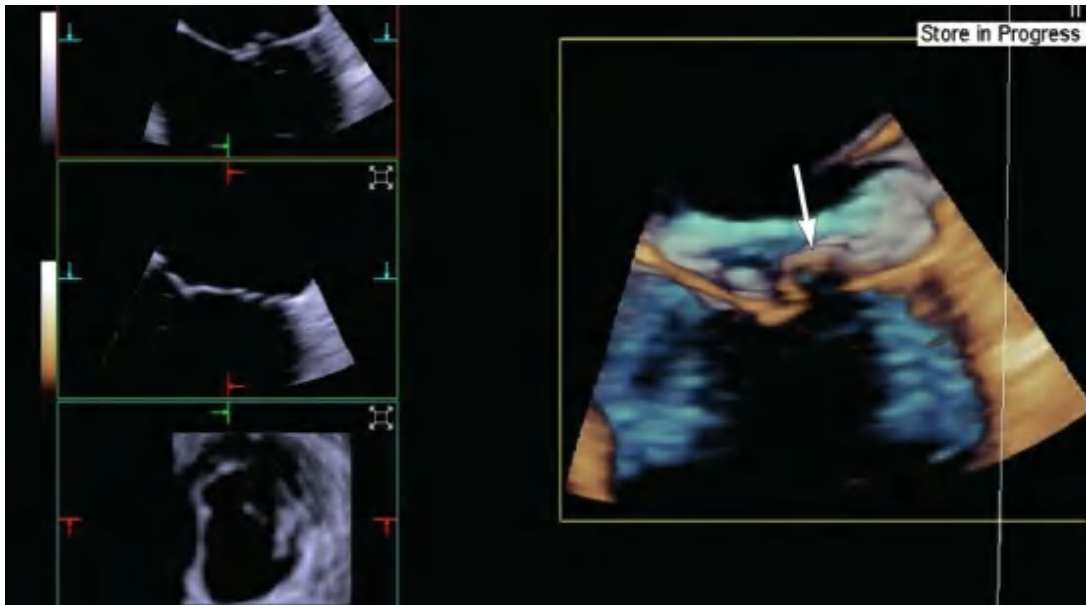
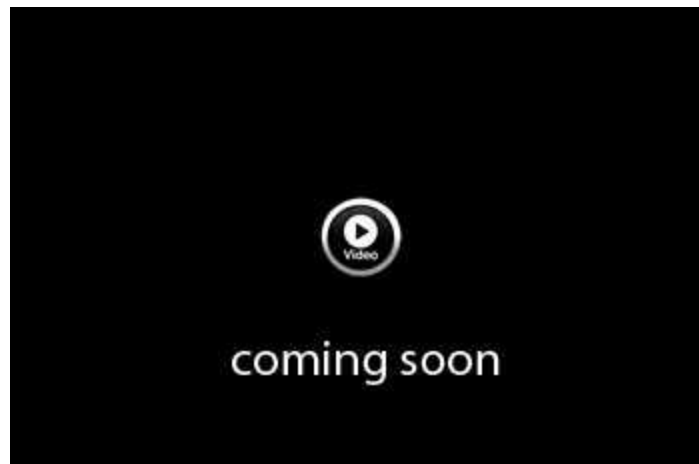


**FIGURE 4.78.** Once a three-dimensional data set is recorded, the displayed planes can be manipulated to show a specific area of interest. In this case, a 3D display of a prolapsed mitral valve (*arrow*) is shown, along with the corresponding two-dimensional planes. 



**Video 4-78**



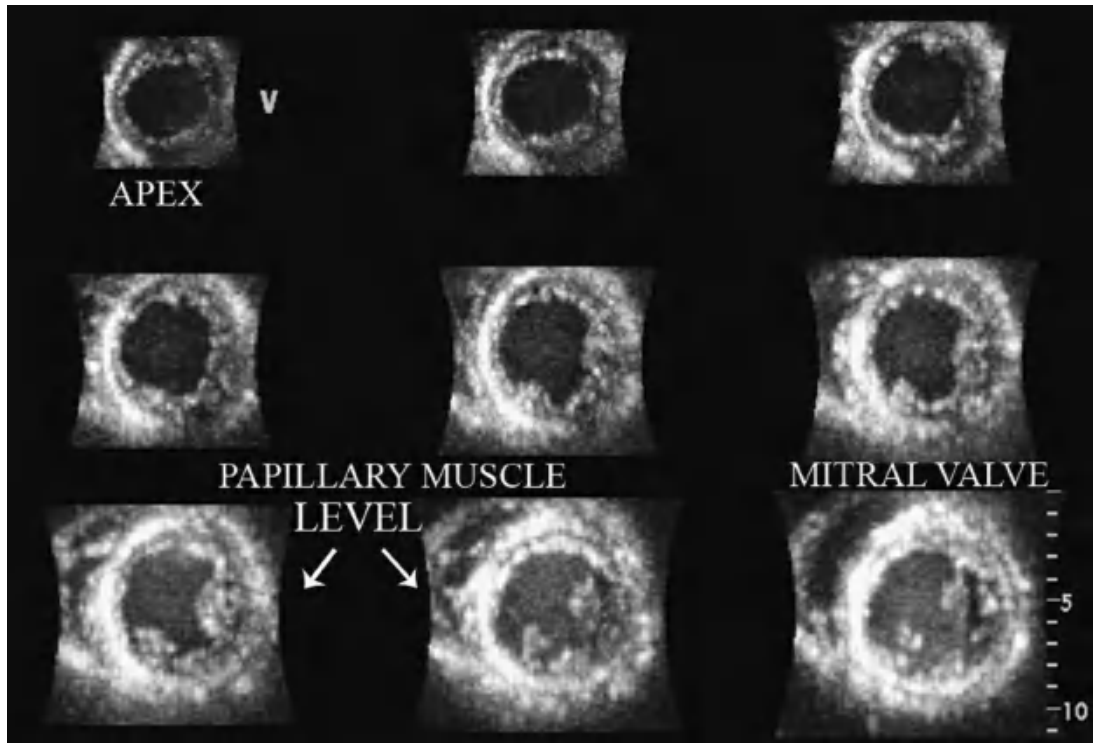
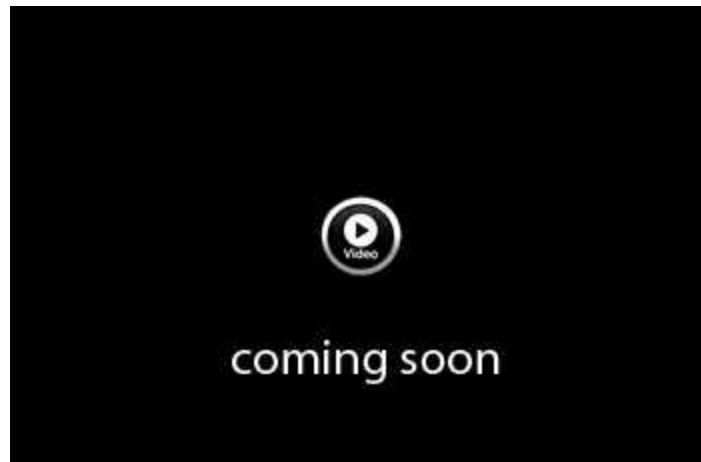


**FIGURE 4.79.** Series of nine equidistant short-axis images automatically extracted from a three-dimensional data set. Moving left-to-right and top-to-bottom are recorded at the apex through the papillary muscle and, finally, mitral valve level. Note that in the real-time image, wall motion can be accurately analyzed at each of these imaging planes in real time. 📺



**Video 4-79**

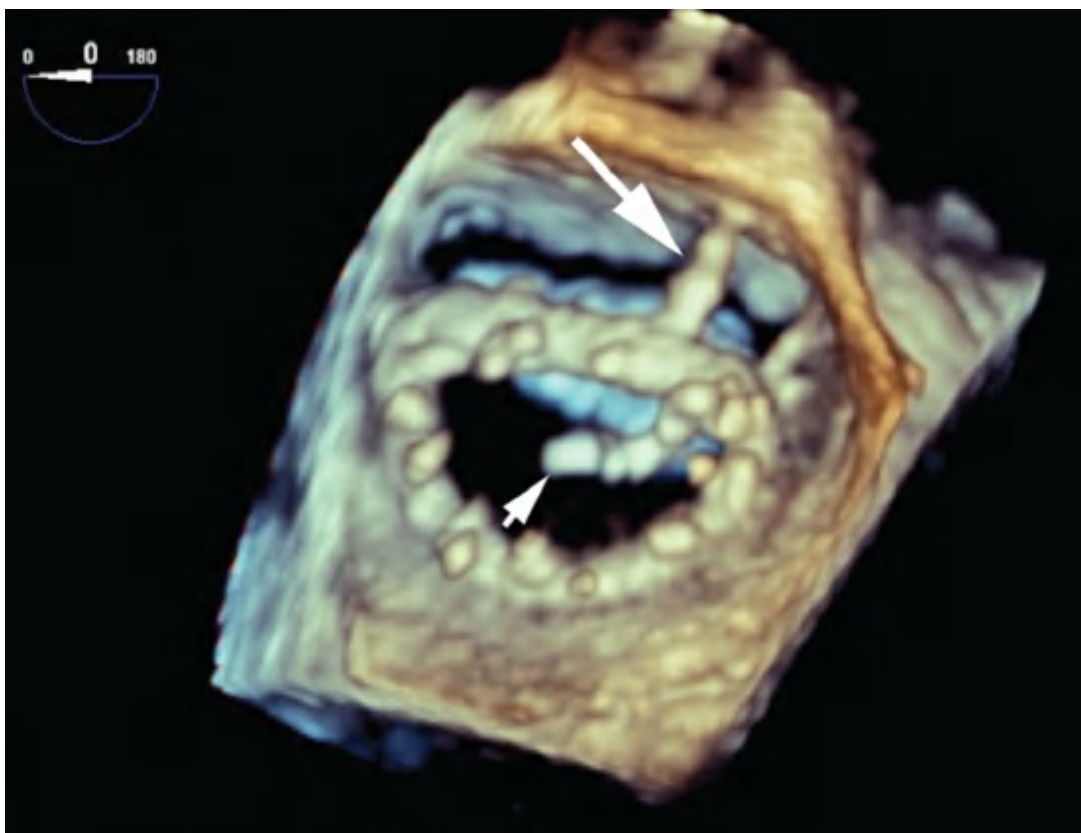
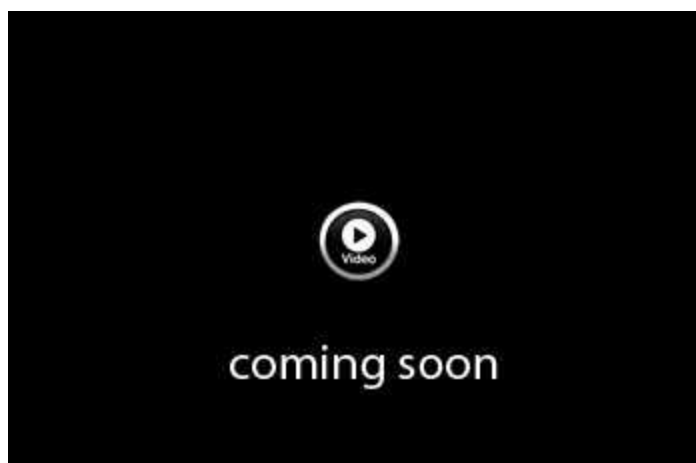


**FIGURE 4.80.** Using live 3D, a dehisced mitral ring from a patient with endocarditis is displayed. The ring is shown from the perspective of the left atrium. A large region of dehiscence is seen superior to the ring, with the *large arrow* demonstrating an intact suture. The *small arrowhead* points 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.

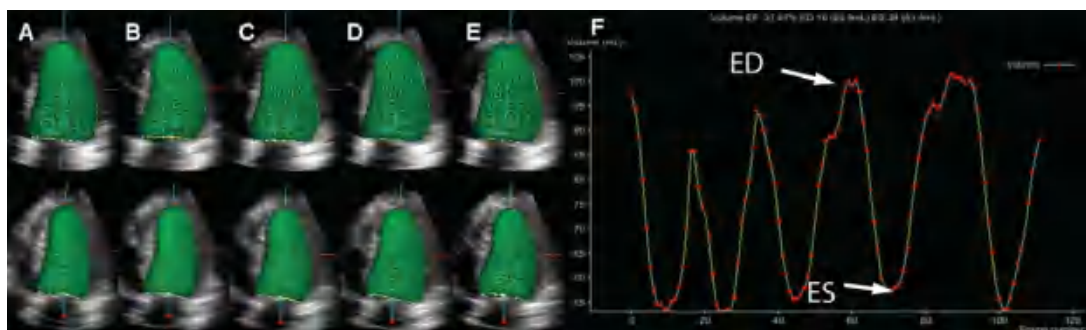


**FIGURE 4.81.** Real-time three-dimensional echocardiography is recorded from a patient with atrial fibrillation. On the **left**, five consecutive cardiac cycles are shown, with the end-diastolic (ED) left ventricular volumes on top and the corresponding end-systolic (ES) volumes directly below. On the **right**, the instantaneous left ventricular volumes are plotted, illustrating the beat-to-beat variability and range of ejection fraction.

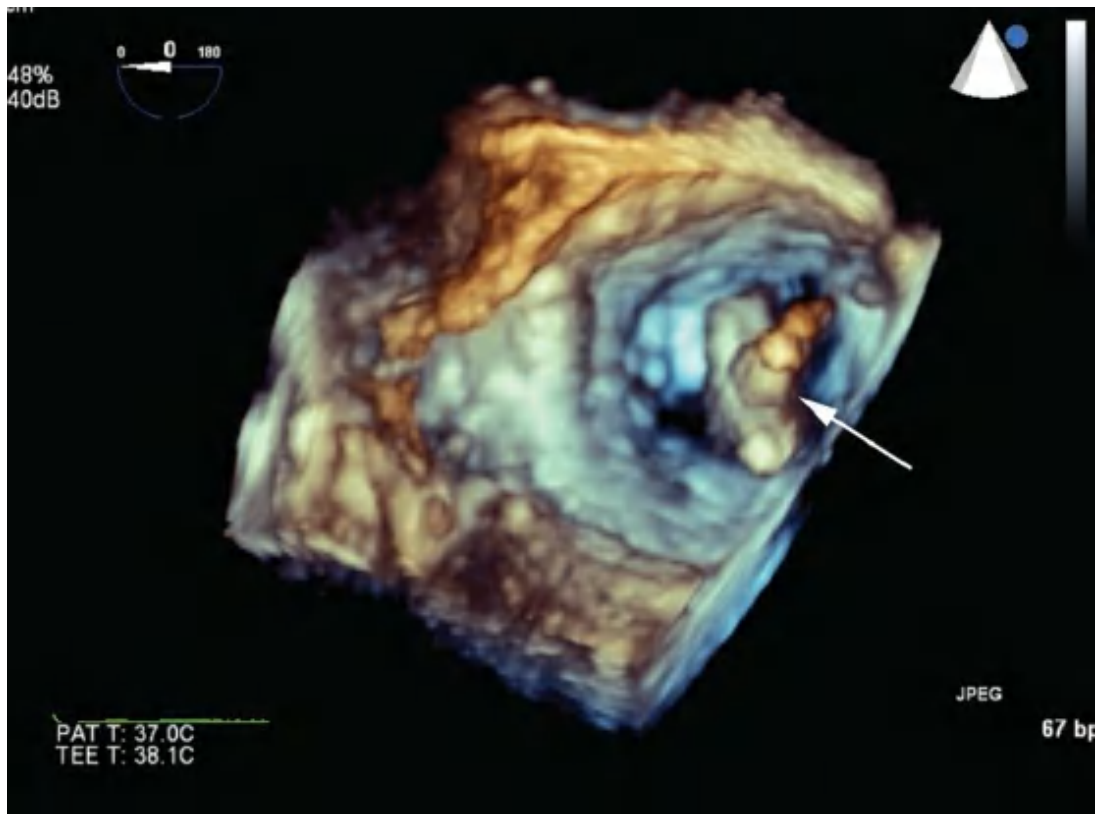


**FIGURE 4.82.** A Mitra Clip device (*arrow*) is seen positioned between the two leaflets of a myxomatous mitral valve using three-dimensional echocardiography.

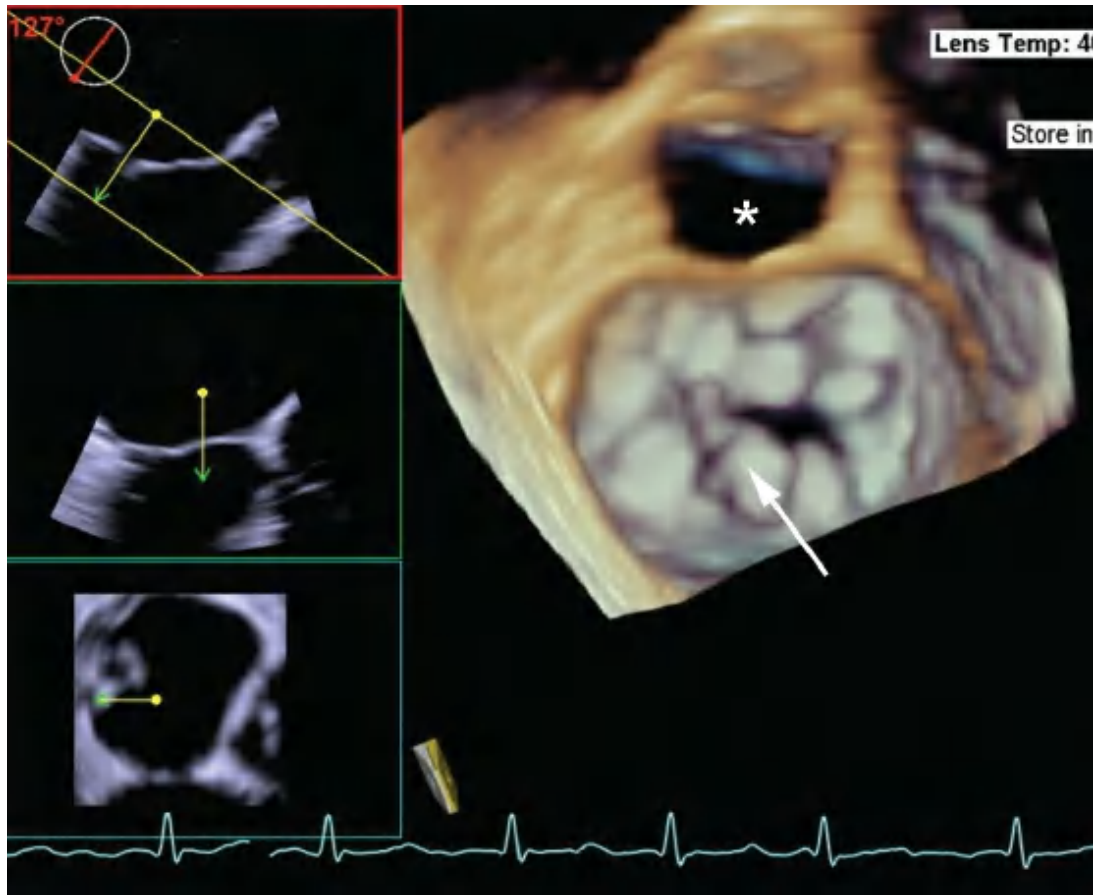
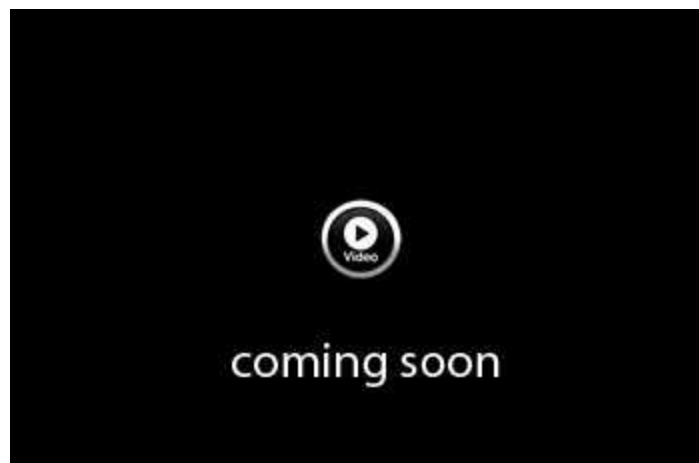


**FIGURE 4.83.** Detailed anatomic information on intracardiac pathology is possible with real-time three-dimensional echocardiography. In this case, from a patient with mitral valve prolapse, the specific prolapsing scallop (*arrow*) is shown clearly, along with the relationship of the mitral valve to the aorta (\*). 📺



**Video 4-83**

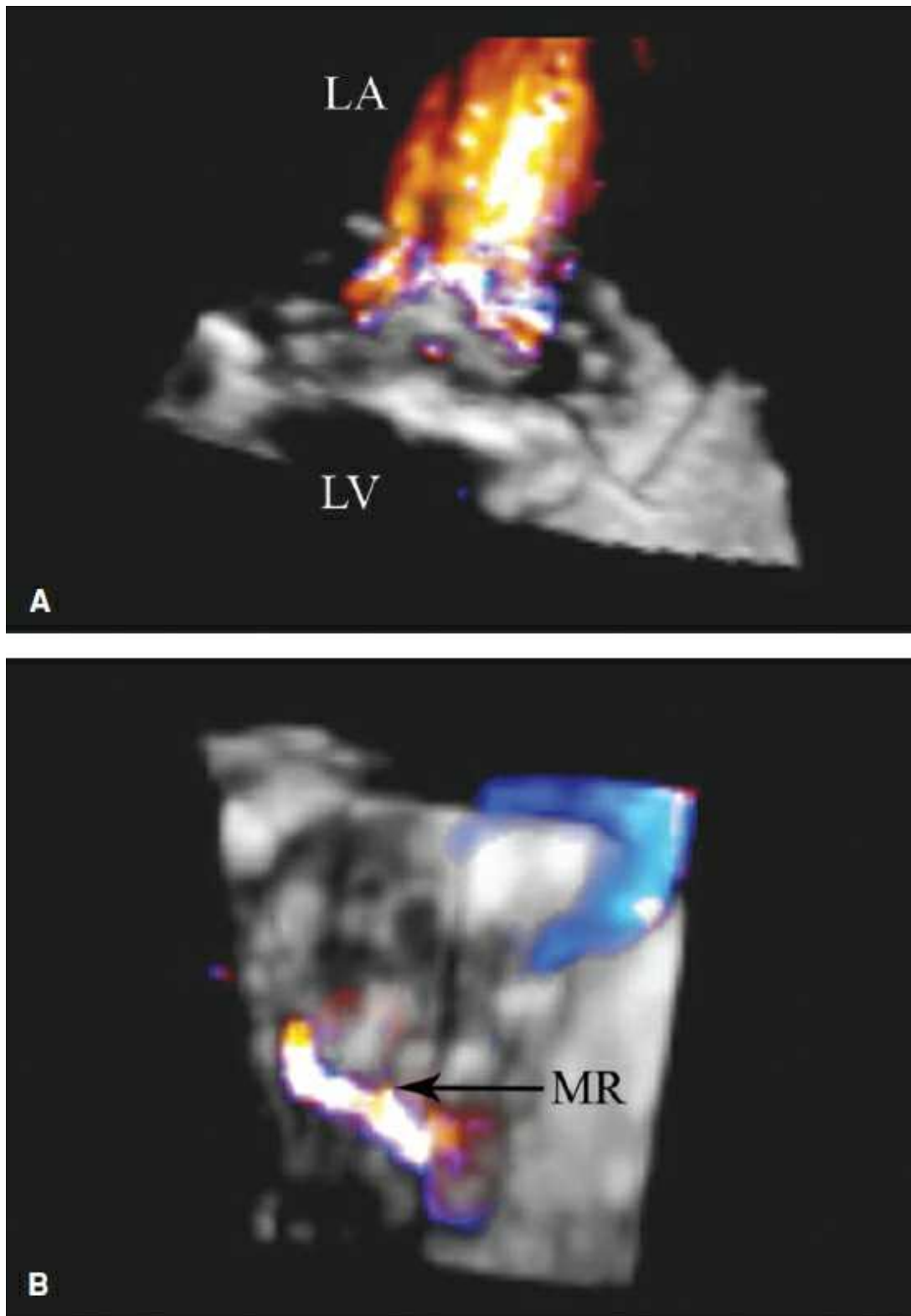
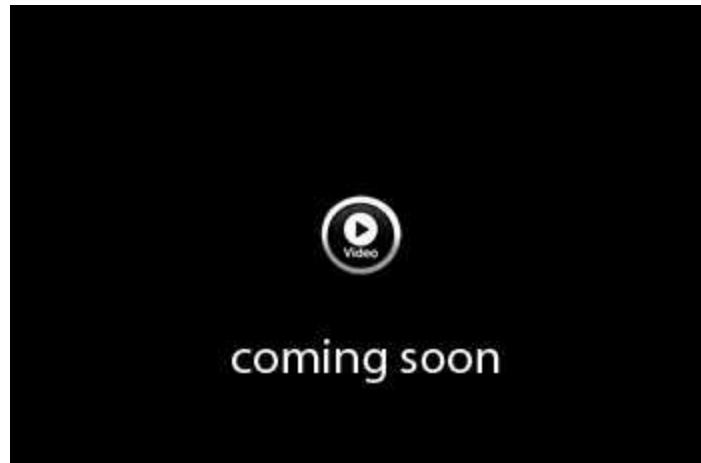


**FIGURE 4.84.** A, B: Real-time three-dimensional transesophageal echocardiographic image of mitral regurgitation. B: This image can be rotated and cropped to provide a short-axis image of the actual regurgitant orifice. MR, mitral regurgitation. 



#### **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.



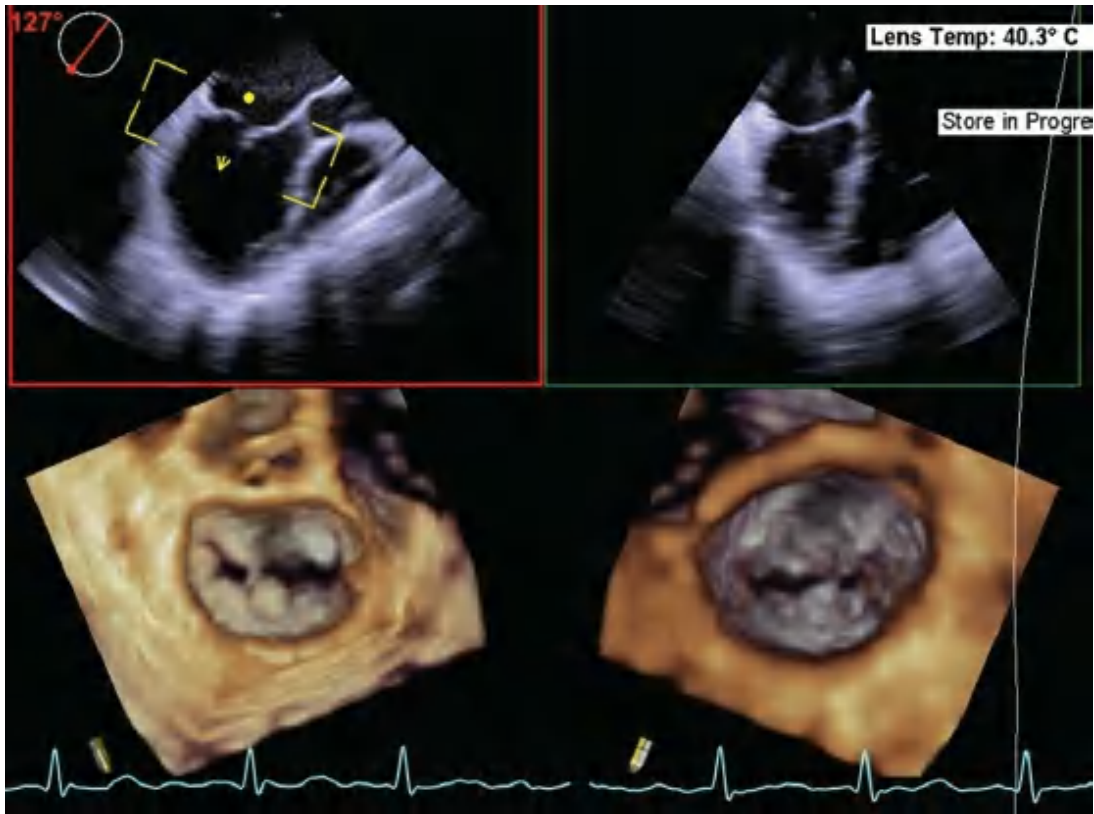
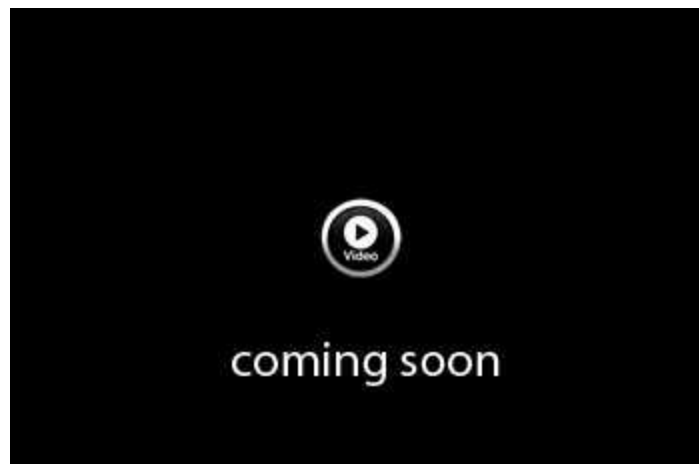


**FIGURE 4.85.** An advantage of live-3D imaging is the ability to display anatomic information during surgery or interventions. In this example, from a patient with mitral valve prolapse, the detailed anatomy of the mitral leaflets can be shown from both the left atrial and left ventricular perspective. 



**Video 4-85**



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 smart phone. Two-dimensional and color Doppler images can be recorded and stored on the phone or tablet.

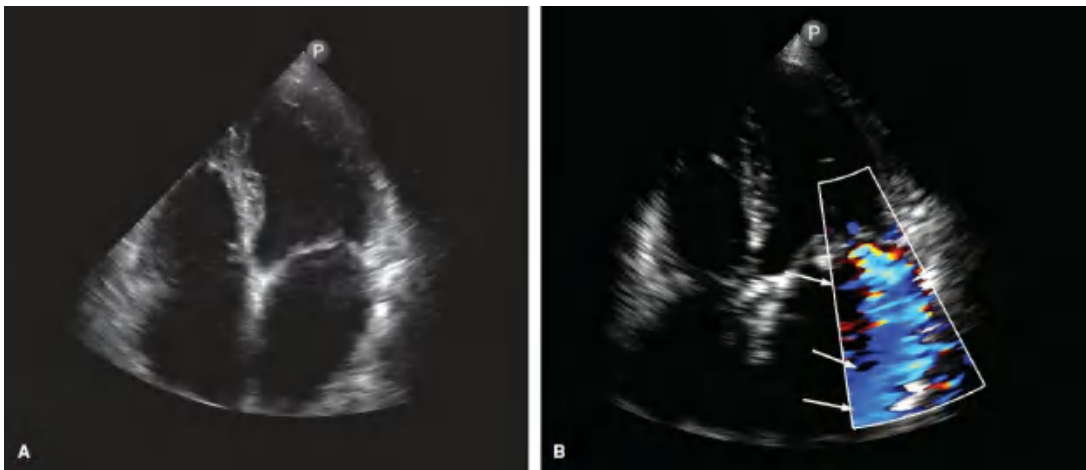
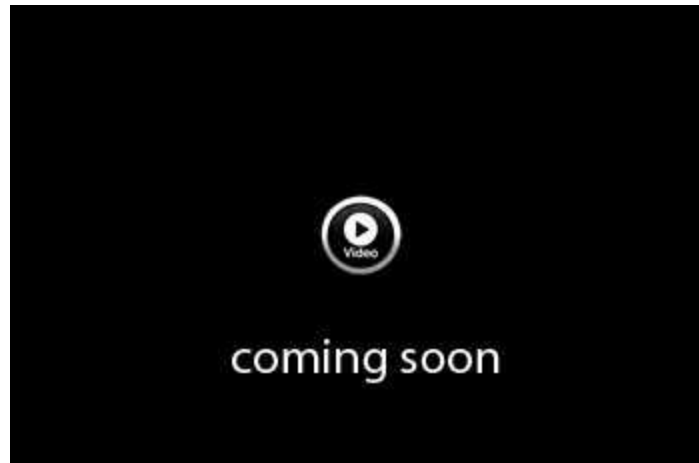
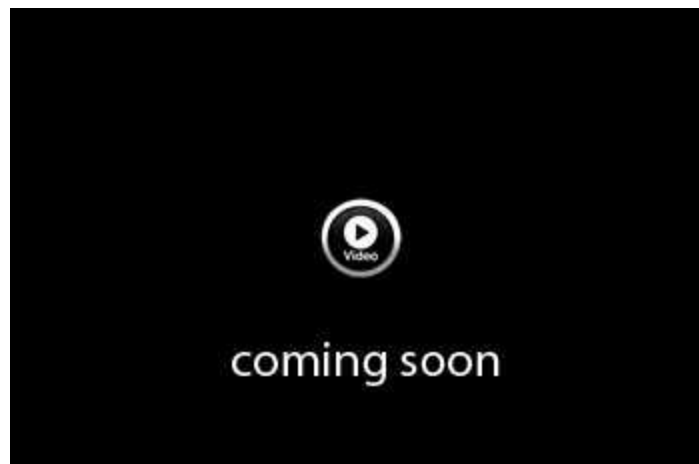


**FIGURE 4.87.** These images (A, B) from a patient with severe mitral regurgitation were recorded using the point-of-care device shown in [Figure 4.86](#).





**Video 4-87a**



**Video 4-87b**

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**

**INDICATIONS FOR ECHOCARDIOGRAPHY TO SCREEN FOR THE PRESENCE OF CARDIOVASCULAR DISEASE**

|                                                                                                                                                                | Class |
|----------------------------------------------------------------------------------------------------------------------------------------------------------------|-------|
| 1. Patients with a family history of genetically transmitted cardiovascular disease                                                                            | I     |
| 2. Potential donors for cardiac transplantation                                                                                                                | I     |
| 3. Patients with phenotypic features of Marfan syndrome or related connective tissue diseases                                                                  | I     |
| 4. Baseline and reevaluations of patients undergoing chemotherapy with cardiotoxic agents                                                                      | I     |
| 5. First-degree relatives of patients with unexplained dilated cardiomyopathy in whom no etiology has been identified                                          | I     |
| 6. Patients with systemic disease that may affect the heart                                                                                                    | IIb   |
| 7. The general population                                                                                                                                      | III   |
| 8. Competitive athletes without clinical evidence of heart disease                                                                                             | III   |
| 9. Routine screening echocardiogram for participation in competitive sports in patients with normal cardiovascular history, electrocardiogram, and examination | III   |

Adapted with permission from Cheitlin MD, Alpert JS, Armstrong WF, et al. ACC/AHA guidelines for the clinical application of echocardiography: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Clinical Application of Echocardiography) developed in collaboration with the American Society of Echocardiography. *Circulation* 1997;95:1686–1744. Copyright © 1997 American Heart Association.



## 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.

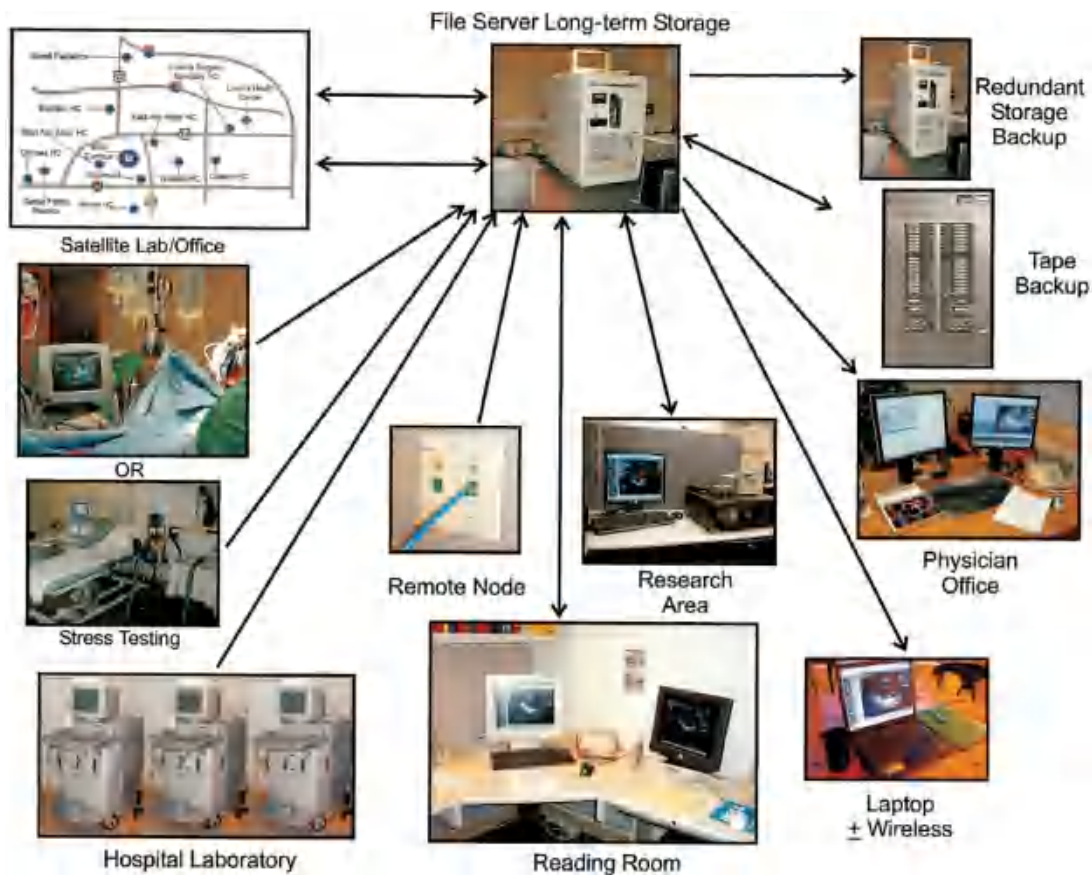


**FIGURE 4.88.** Diagrammatic representation of a modern digital echocardiography laboratory denoting the multiple components for image analysis and storage as well as the diversity of sites from which images can be transferred or viewed.

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 4.7** SUMMARY OF TRAINING REQUIREMENTS IN ECHOCARDIOGRAPHY

| Level | Duration of Training (months) | Cumulative Duration of Training (months) | Minimal Total no. of TTE Examinations Performed | Minimal Total no. of TTE Examinations Interpreted | TEE and Special Procedures |
|-------|-------------------------------|------------------------------------------|-------------------------------------------------|---------------------------------------------------|----------------------------|
| 1     | 3                             | 3                                        | 75                                              | 150                                               | Yes <sup>a</sup>           |
| 2     | 3                             | 6                                        | 150                                             | 300                                               | Yes <sup>b</sup>           |
| 3     | 3                             | 9                                        | 300                                             | 750                                               | Yes                        |

<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.

Adapted with permission from Ryan T, Berlacher K, Lindner JR, et al. COCATS 4 Task Force 5: Training in Echocardiography. *J Am Coll Cardiol* 2015;65:1786–1799. Copyright © 2015 American College of Cardiology Foundation.



## 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

Douglas PS, Garcia MJ, Haines DE, et al. ACCF/ASE/AHA/ASNC/HFSA/HRS/SCAI/SCCM/SCCT/SCMR. 2011 appropriate use criteria for echocardiography. A Report of the American College of Cardiology Foundation Appropriate Use Criteria Task Force, American Society of Echocardiography, American Heart Association, American Society of Nuclear Cardiology, Heart Failure Society of America, Heart Rhythm Society, Society for Cardiovascular Angiography and Interventions, Society of Critical Care Medicine, Society of Cardiovascular Computed Tomography, and Society for Cardiovascular Magnetic Resonance Endorsed by the American College of Chest Physicians. *J Am Coll Cardiol* 2011;57:1126–1166.

Samad Z, Minter S, Armour A, et al. Implementing a continuous quality improvement program in a high-volume clinical echocardiography laboratory: improving care for patients with aortic stenosis. *Circ Cardiovasc Imaging* 2016;9:e003708.

### APPROPRIATE USE

Bhatia RS, Ivers NM, Yin XC, et al. Improving the appropriate use of transthoracic echocardiography: the Echo WISELY Trial. *J Am Coll Cardiol* 2017;70:1135–1144.

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echocardiography. A Report of the American College of Cardiology Foundation Appropriate Use Criteria Task Force, American Society of Echocardiography, American Heart Association, American Society of Nuclear Cardiology, Heart Failure Society of America, Heart Rhythm Society, Society for Cardiovascular Angiography and Interventions, Society of Critical Care Medicine, Society of Cardiovascular Computed Tomography, Society for Cardiovascular Magnetic Resonance American College of Chest Physicians. *J Am Soc Echocardiogr* 2011;24:229–267.

## **APPLICATIONS**

Cheitlin MD, Alpert JS, Armstrong WF, et al. ACC/AHA guidelines for the clinical application of echocardiography: a report of the American College of Cardiology/ American Heart Association Task Force on Practice Guidelines (Committee on Clinical Application of Echocardiography) developed in collaboration with the American Society of Echocardiography. *Circulation* 1997;95:1686–1744.

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Hu K, Liu D, Niemann M, et al. Methods for assessment of left ventricular systolic function in technically difficult patients with poor imaging quality. *J Am Soc Echocardiogr* 2013;26:105–113.

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## 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.



### LINEAR MEASUREMENTS

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**

| Parameter                              | Formula                                                              | Abbreviation      | Units                         |
|----------------------------------------|----------------------------------------------------------------------|-------------------|-------------------------------|
| LV internal dimension in diastole      |                                                                      | LVID <sub>d</sub> | mm (or cm)                    |
| LV internal dimension in systole       |                                                                      | LVID <sub>s</sub> | mm (or cm)                    |
| Fractional shortening                  | $(\text{LVID}_d - \text{LVID}_s) / \text{LVID}_d$                    | FS                | % or 0.XX                     |
| Meridional wall stress in systole      | PR/h                                                                 | $\sigma_m$        | mm Hg or dyne-cm <sup>2</sup> |
| Cubed LV volume in diastole            | $(\text{LVID}_d)^3$                                                  |                   | cm <sup>3</sup> or mL         |
| Cubed LV + myocardial volume           | $(\text{IVS} + \text{LVID}_d + \text{PW})^3$                         |                   | cm <sup>3</sup> or mL         |
| Velocity of circumferential shortening | $(\text{LVID}_d - \text{LVID}_s) / (\text{LVID}_d \times \text{ET})$ | VCf               | Circumference/s               |

ET, ejection time; h, wall thickness; PR, pressure × radius; PW, posterior wall.

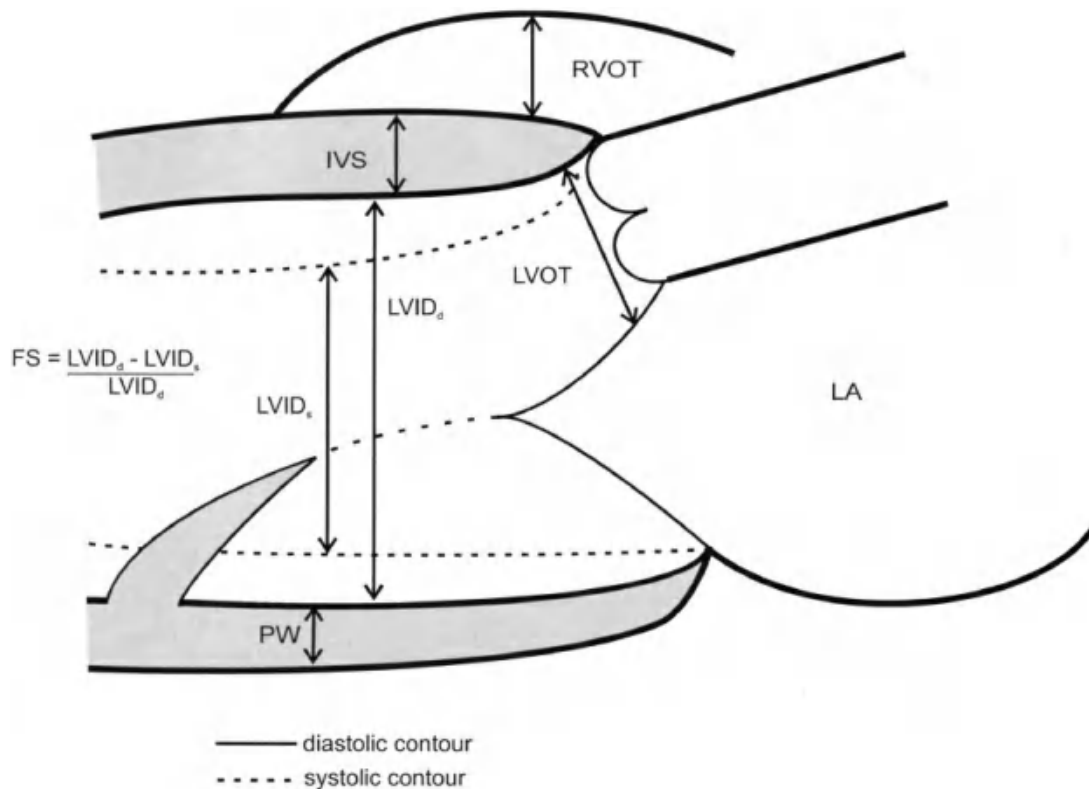


**FIGURE 5.1.** Schematic of a parasternal long-axis view of the left ventricle depicting linear measurements. By convention, linear measurements of the left ventricle are made at the level of the mitral chordae. From the linear internal dimension of the left ventricle in diastole and systole, fractional 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<sub>d</sub>, left ventricular internal dimension in diastole; LVID<sub>s</sub>, left ventricular internal dimension in systole; PW, posterior wall.

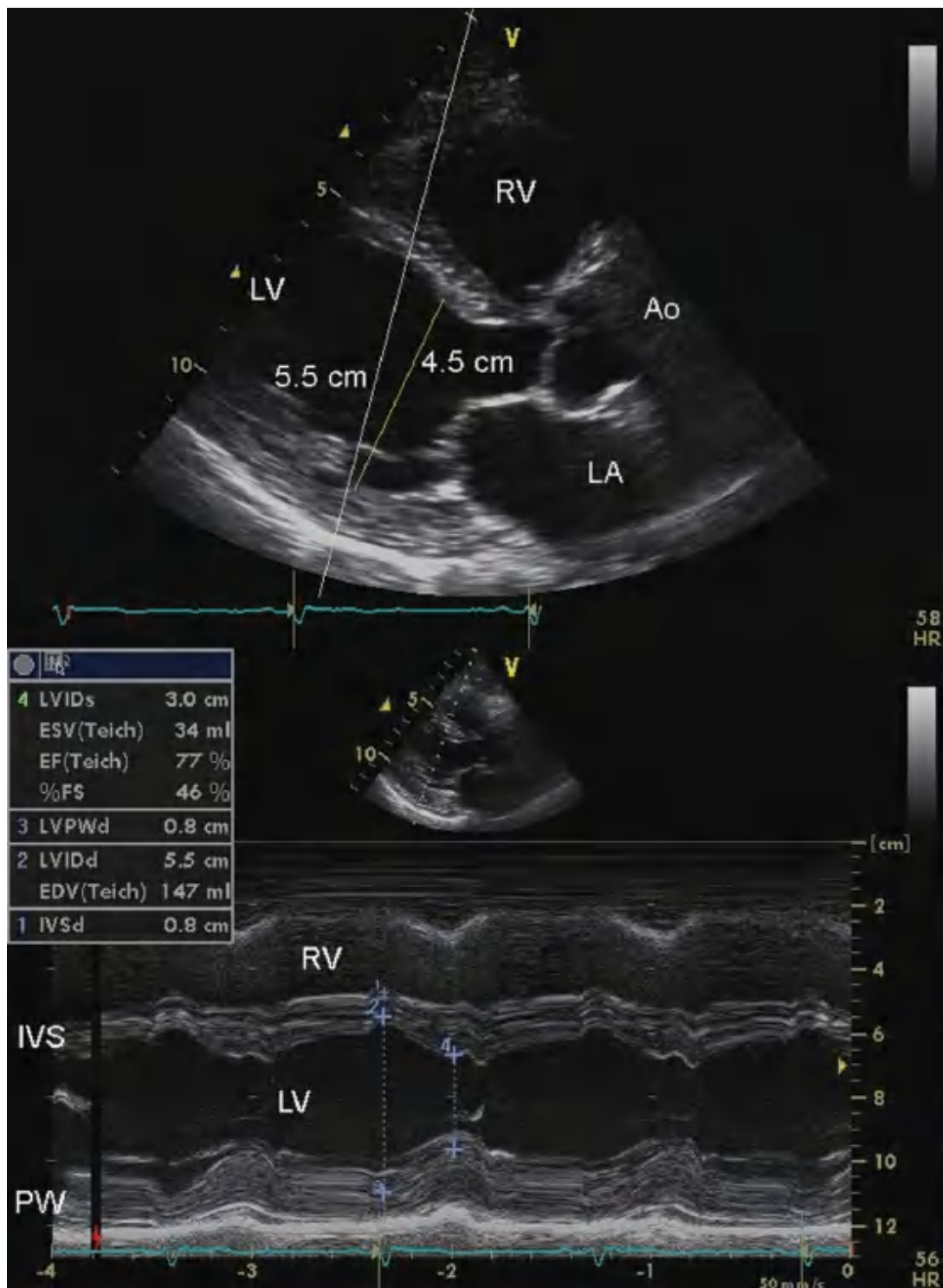


**FIGURE 5.2.** Parasternal long-axis echocardiogram and two-dimensional-derived M-mode echocardiogram in a patient with normal ventricular function. On the M-mode echocardiogram, note the internal dimension of the left ventricle of 5.5 cm and the derived values. On the two-dimensional echocardiogram, 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. 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.

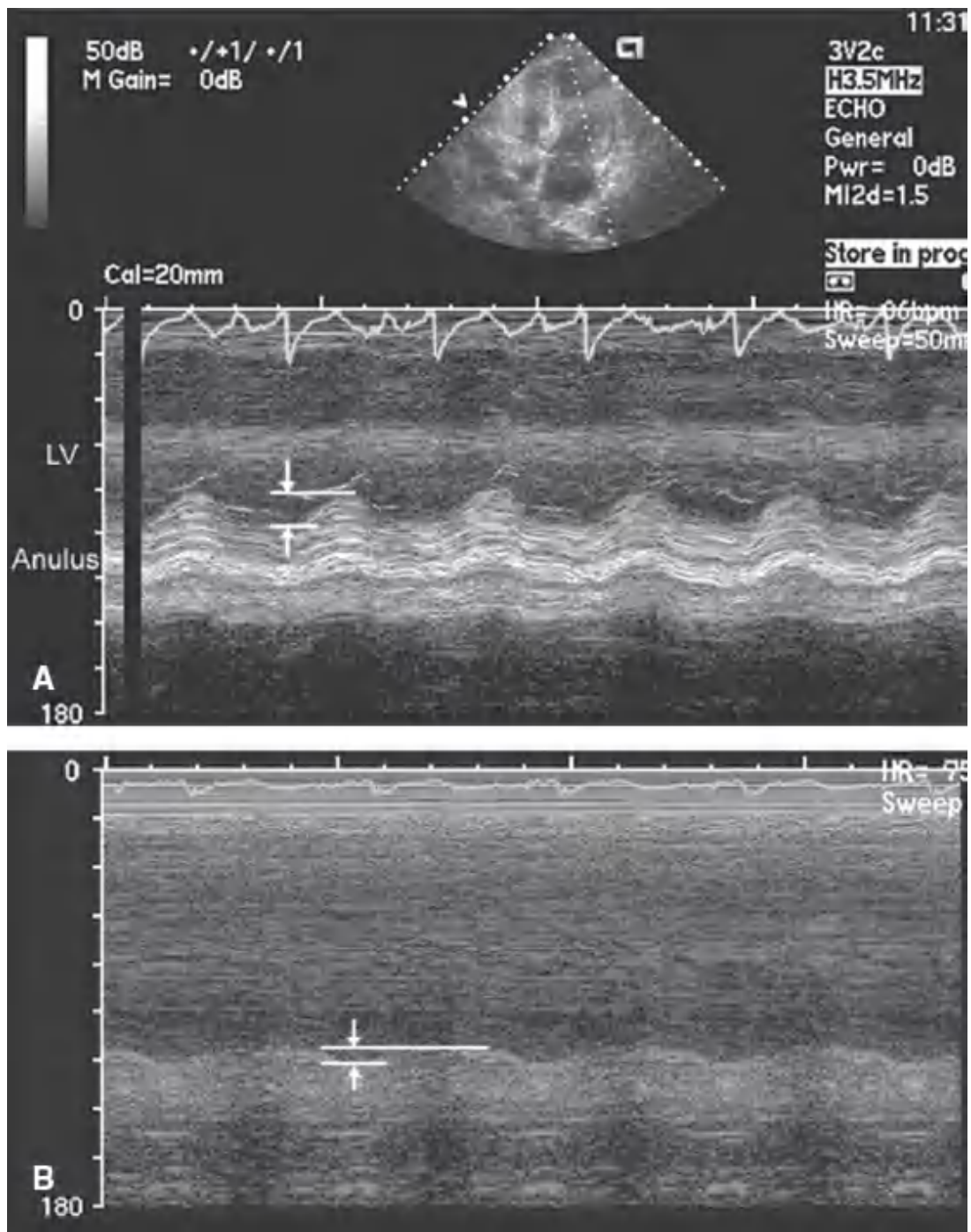


**FIGURE 5.3.** Apical view recorded in two patients demonstrates the measurement of the descent of the base with M-mode echocardiography (arrows). The M-mode interrogation beam has been directed from the apex of the heart through the lateral annulus. **A:** Note the approximate 1.6 cm of annular motion 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](#).

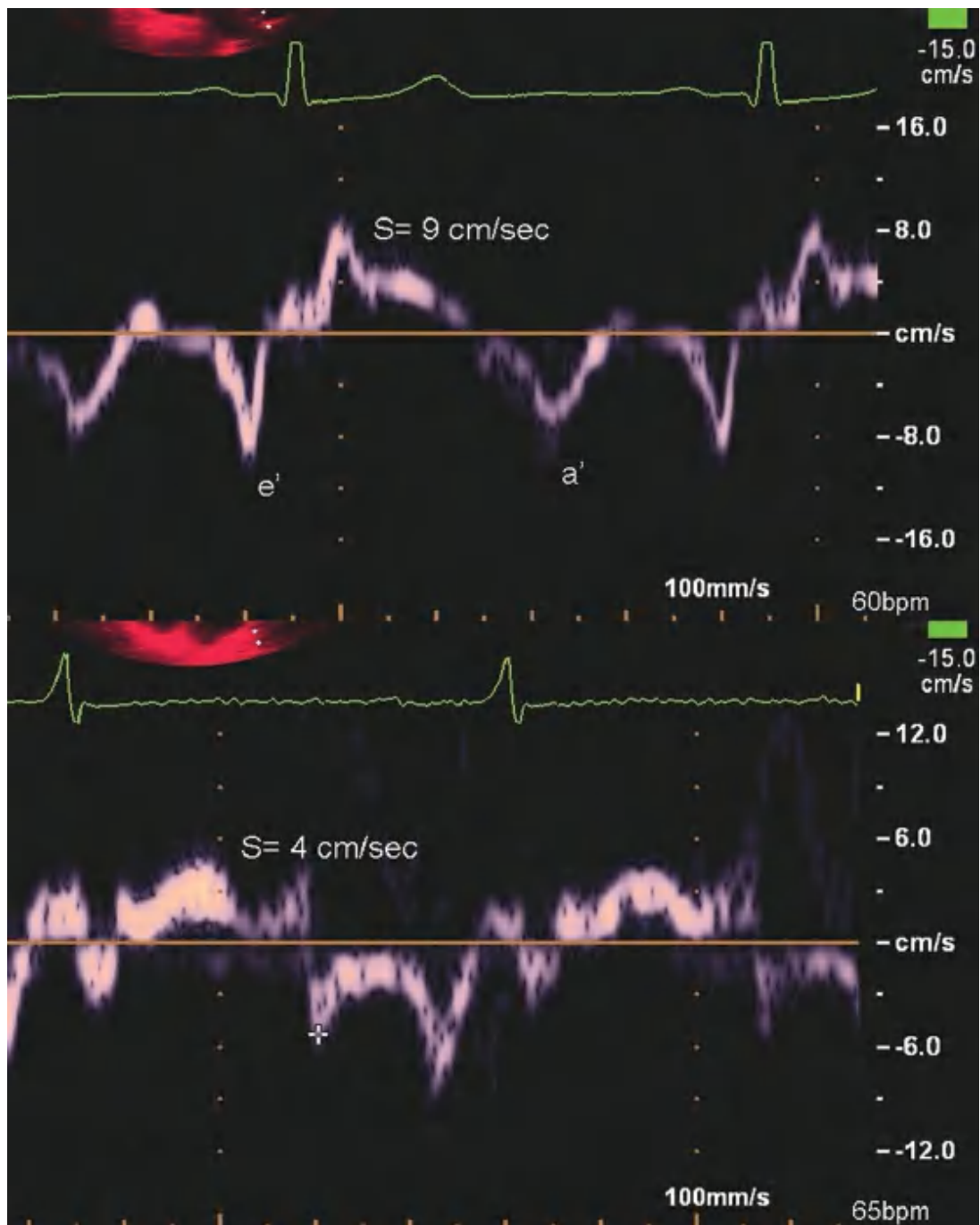


**FIGURE 5.4.** Doppler tissue imaging of the lateral annulus performed in two patients. The upper panel was recorded in a patient with normal systolic function and an ejection fraction of 60%. Notice the S wave of 9 cm/s. Also noted are the diastolic e' and a' velocities. The lower panel was recorded 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.

## Indirect M-Mode Markers of Left Ventricular Function







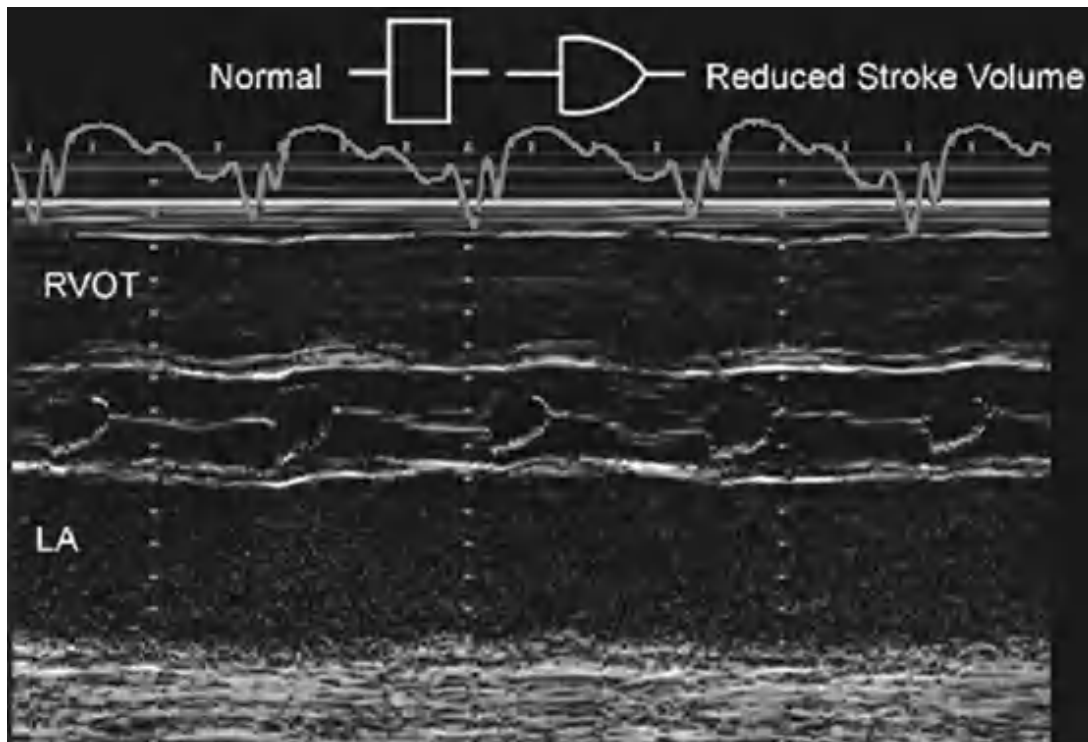


**FIGURE 5.6.** M-mode echocardiogram recorded through the aortic valve in a patient with reduced cardiac function and decreased forward stroke volume. Note the rounded closure of the aortic valve, indicating decreasing forward flow at the end of systole. Normal and abnormal aortic valve opening patterns 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 ( $\text{area} = \pi r^2$ ). 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 5.2** AREA/VOLUME-BASED MEASUREMENTS FOR VENTRICULAR SIZE AND FUNCTION<sup>a</sup>

| Parameter                             | Abbreviations       | Formula                 | Units           |
|---------------------------------------|---------------------|-------------------------|-----------------|
| Short-axis diastolic area (at mid LV) | ASx <sub>d</sub>    |                         | cm <sup>2</sup> |
| Short-axis systole area (at mid LV)   | ASx <sub>s</sub>    |                         | cm <sup>2</sup> |
| Fractional area change                | FAC                 | $(ASx_d - ASx_s)/ASx_d$ | % or 0.XX       |
| Four-chamber LV area in diastole      | ALV <sub>4c-d</sub> |                         | cm <sup>2</sup> |
| Four-chamber LV area in systole       | ALV <sub>4c-s</sub> |                         | cm <sup>2</sup> |
| LV volume in diastole <sup>a</sup>    | LVV <sub>d</sub>    |                         | mL              |
| LV volume in systole <sup>a</sup>     | LVV <sub>s</sub>    |                         | mL              |
| Stroke volume                         | SV                  | $LVV_d - LVV_s$         | mL              |
| Ejection fraction                     | EF                  | $SV/LVV_d$              | % or 0.XX       |

<sup>a</sup>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

| Parameter                    | Male           |            | Female         |            |
|------------------------------|----------------|------------|----------------|------------|
|                              | Mean $\pm$ SD  | 2-SD Range | Mean $\pm$ SD  | 2-SD Range |
| LV internal dimension        |                |            |                |            |
| Diastolic dimension (mm)     | 50.2 $\pm$ 4.1 | 42.0–58.4  | 45.0 $\pm$ 3.6 | 37.8–52.2  |
| Systolic dimension (mm)      | 32.4 $\pm$ 3.7 | 25.0–39.8  | 28.2 $\pm$ 3.3 | 21.6–34.8  |
| LV volumes (biplane)         |                |            |                |            |
| LVEDV (mL)                   | 106 $\pm$ 22   | 62–150     | 76 $\pm$ 15    | 46–106     |
| LFESV (mL)                   | 41 $\pm$ 10    | 21–61      | 28 $\pm$ 7     | 14–42      |
| LV volumes normalized by BSA |                |            |                |            |
| LVEDV (mL/m <sup>2</sup> )   | 54 $\pm$ 10    | 34–74      | 45 $\pm$ 8     | 29–61      |
| LVESV (mL/m <sup>2</sup> )   | 21 $\pm$ 5     | 11–31      | 16 $\pm$ 4     | 8–24       |
| LVEF (biplane)               | 62 $\pm$ 5     | 52–72      | 64 $\pm$ 5     | 54–74      |

BSA, body surface area; EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; SD, standard deviation. Reprinted with permission from Lang RM, Badano LP, Mor-Avi V. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr* 2015;28:1–39. e-14. Copyright © 2015 Elsevier.

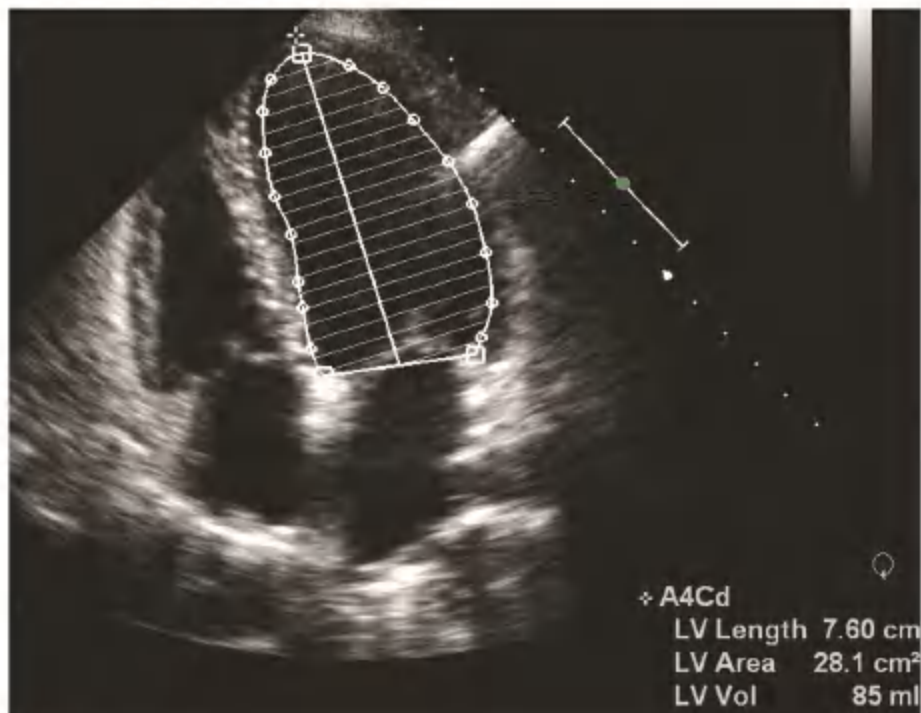
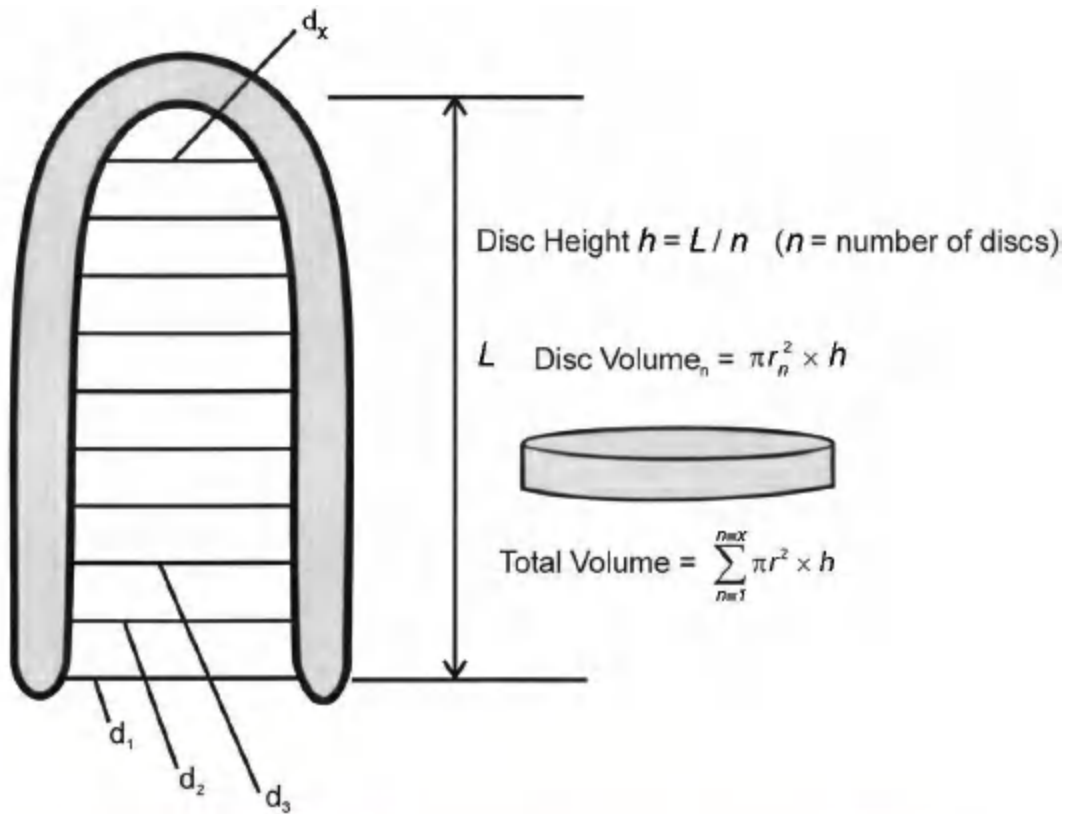


**FIGURE 5.7.** Schematic illustration of Simpson rule or the rule of discs for calculating left ventricular volume. In the upper panel, a schematized left ventricular volume has been subdivided into 10 sections, each of which is presumed to represent a disc of equal diameter at its top and bottom margins.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

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:  $\text{stroke volume} \div \text{end-diastolic volume}$ . 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](#)).



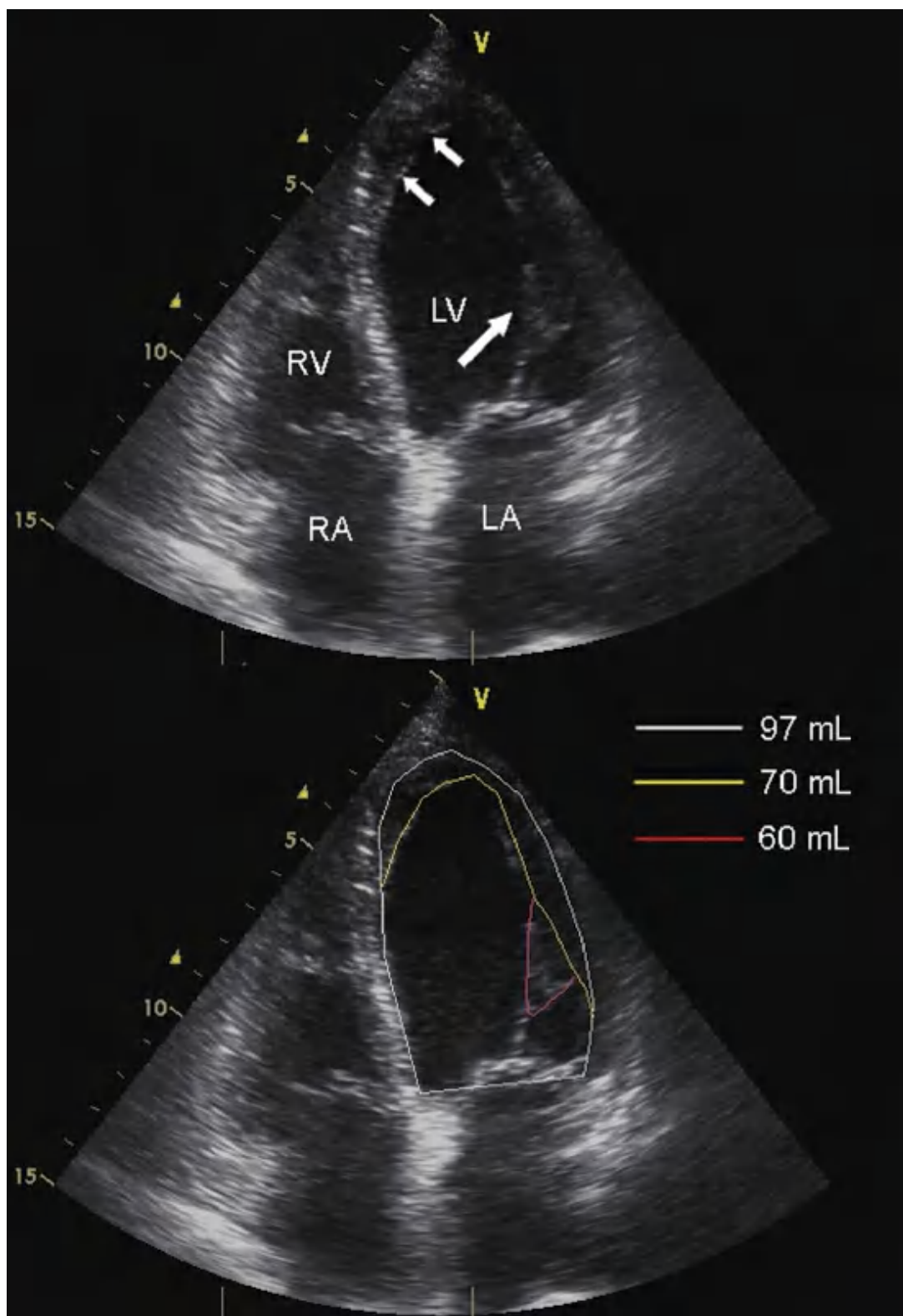


**FIGURE 5.8.** Apical four-chamber view recorded in a patient with normal ventricular size and function. The upper panel is the apical four-chamber view from which volume can be calculated. Notice the vague echoes at the apical septal and apical lateral wall due to a combination of beam width imaging 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.

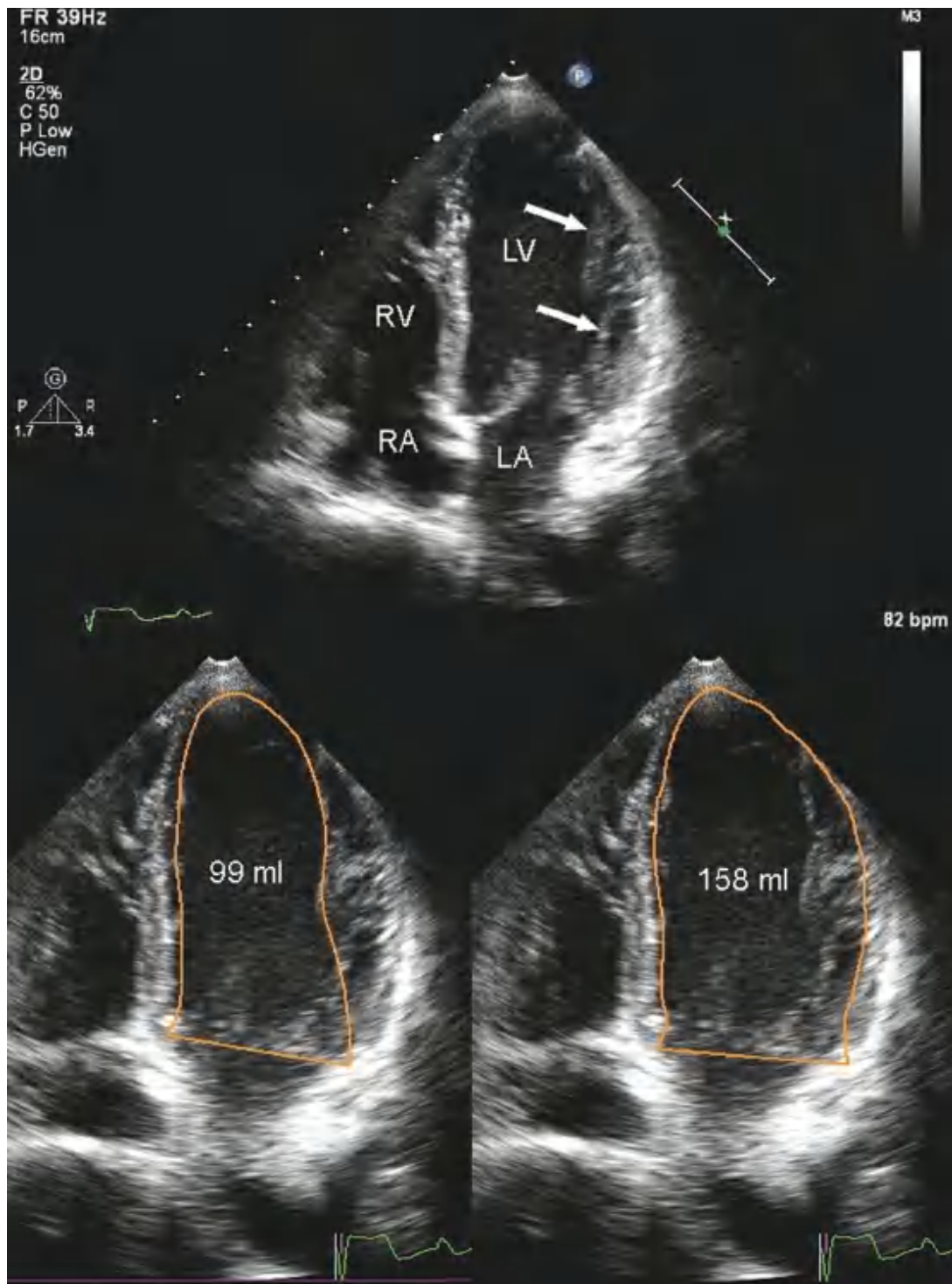
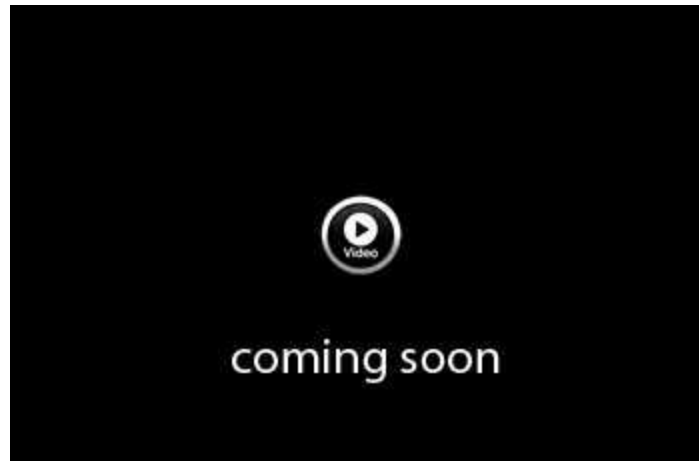


**FIGURE 5.9.** Apical four-chamber view recorded in a young patient with normal ventricular function and fairly prominent trabeculae along the lateral ventricular wall. The upper panel is an apical four-chamber view in which the papillary muscle and trabeculae can be seen on the lateral wall (*arrows*). 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. 📺

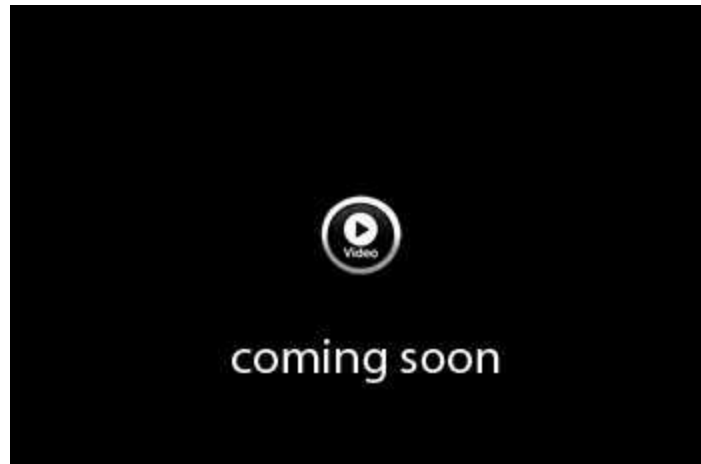


**Video 5-9**



**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**

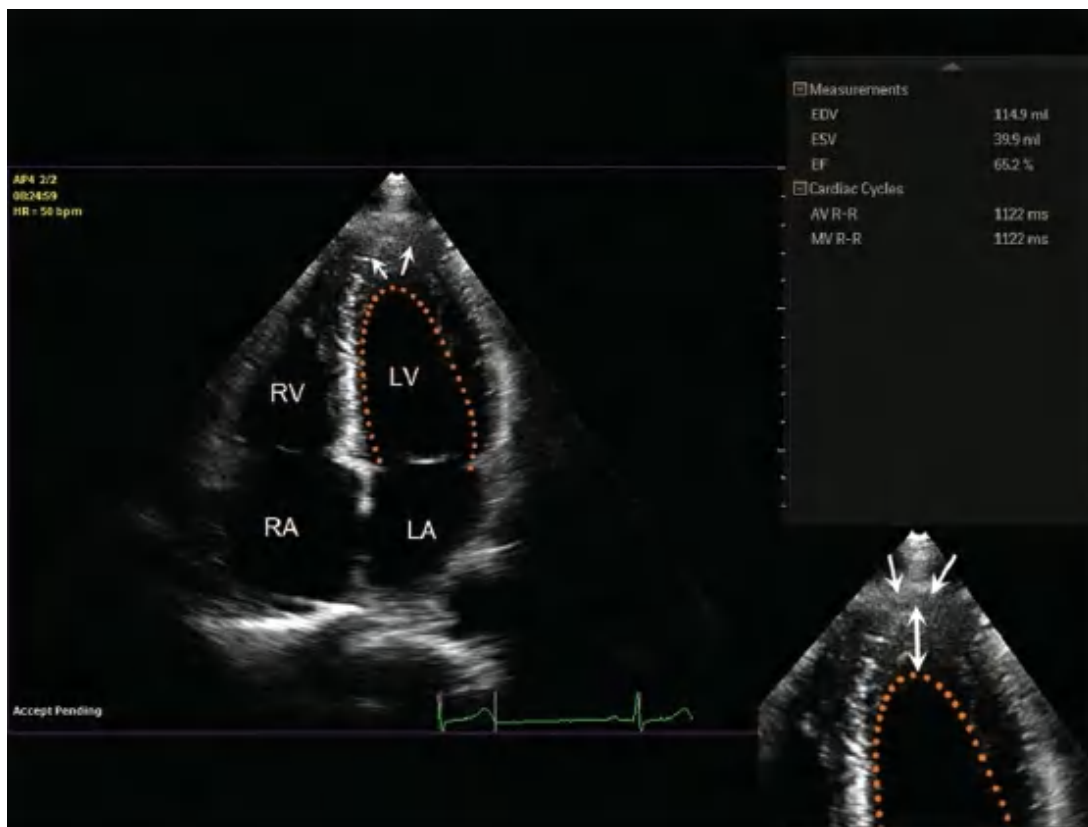
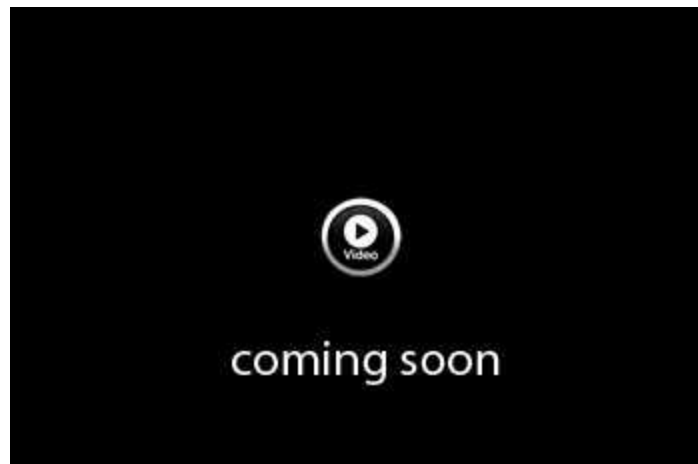


**FIGURE 5.11.** Apical four-chamber view recorded in the same patient depicted in [Figure 5.10](#). This figure represents the first approximation of the ventricular boundary by the automated edge detection algorithm. Note that the automatically detected boundary (*dotted lines*) has foreshortened the 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.

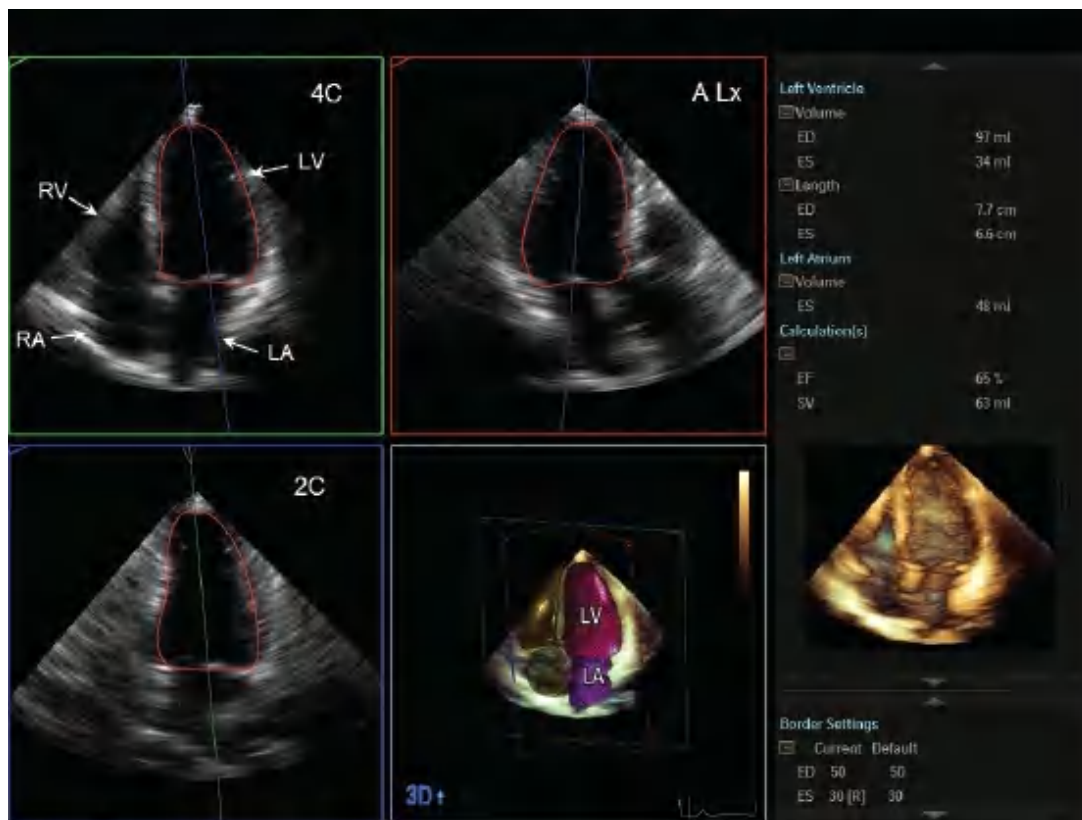


**FIGURE 5.12.** Composite image derived from a real-time three-dimensional full-volume dataset. The image at the far lower right is the cropped three-dimensional volume set in which the details of the left ventricular cavity are identified. The images at the left include apical four-chamber (4C), apical 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.

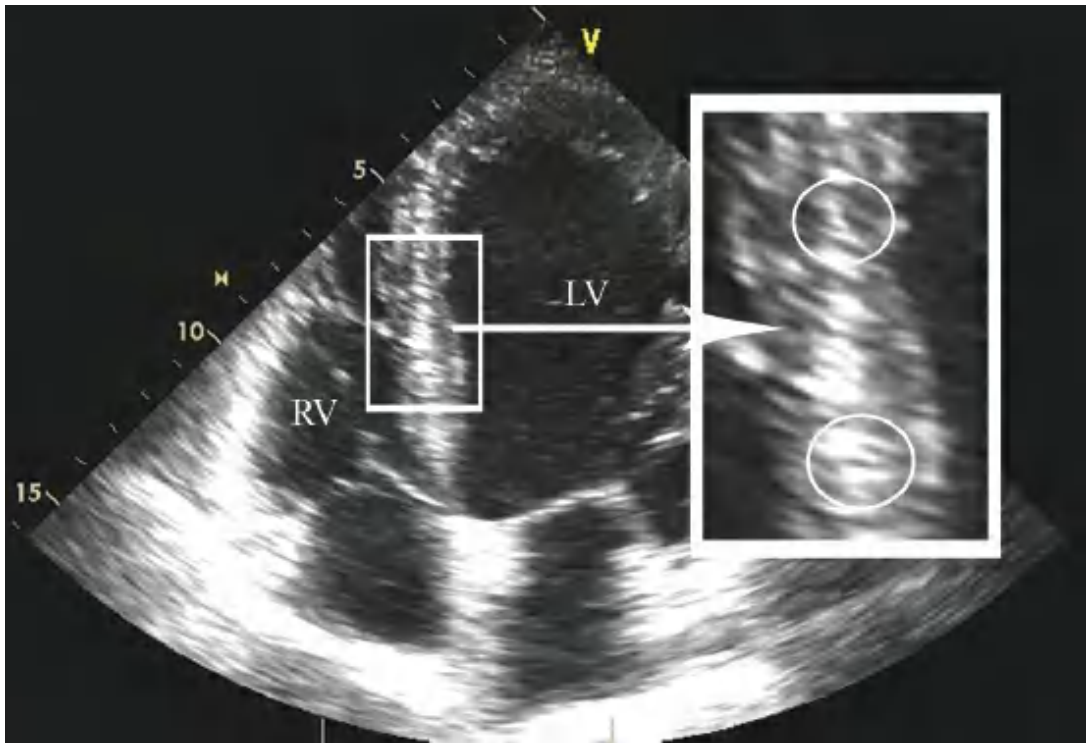


**FIGURE 5.13.** Illustration of the basic principles of speckle tracking. An apical four-chamber view is presented from which a section of the ventricular septum has been expanded (*bordered area*). Within the expanded area, two circular regions of interest are identified. Note the distinctly different 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.

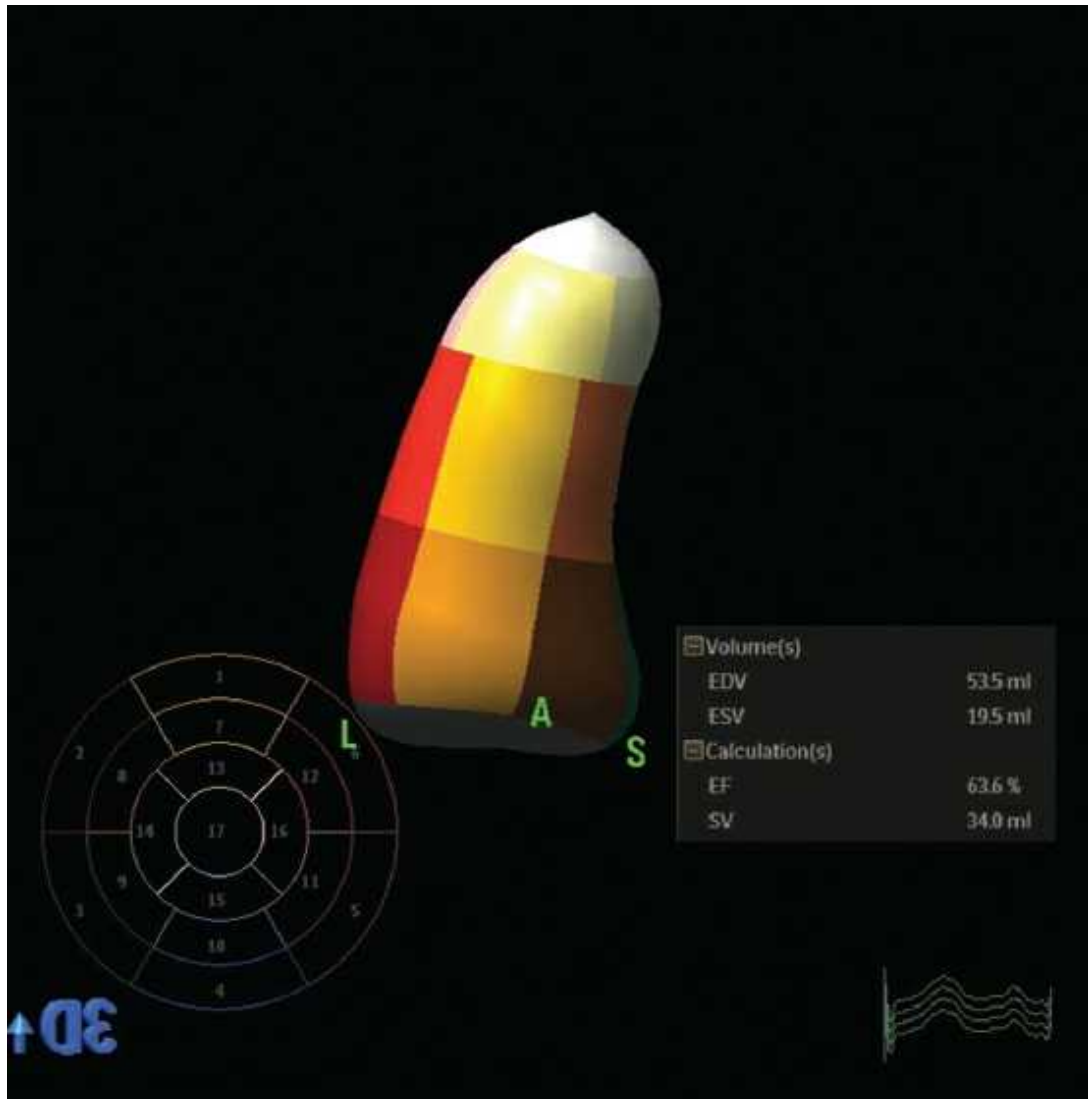
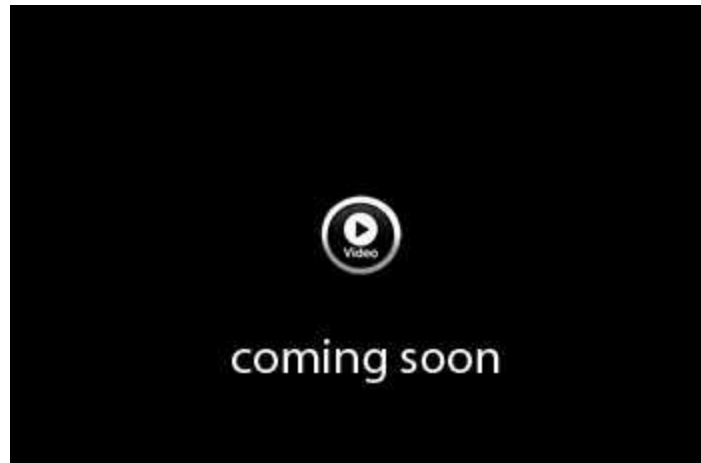


**FIGURE 5.14.** Stylized shell rendering of a normal left ventricle recorded using full-volume three-dimensional echocardiography. Note the calculated end-diastolic and end-systolic volumes as well as ejection fraction and stroke volume. In the accompanying video note the normal motion of all visualized 17 segments. 



### **Video 5-14**

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.

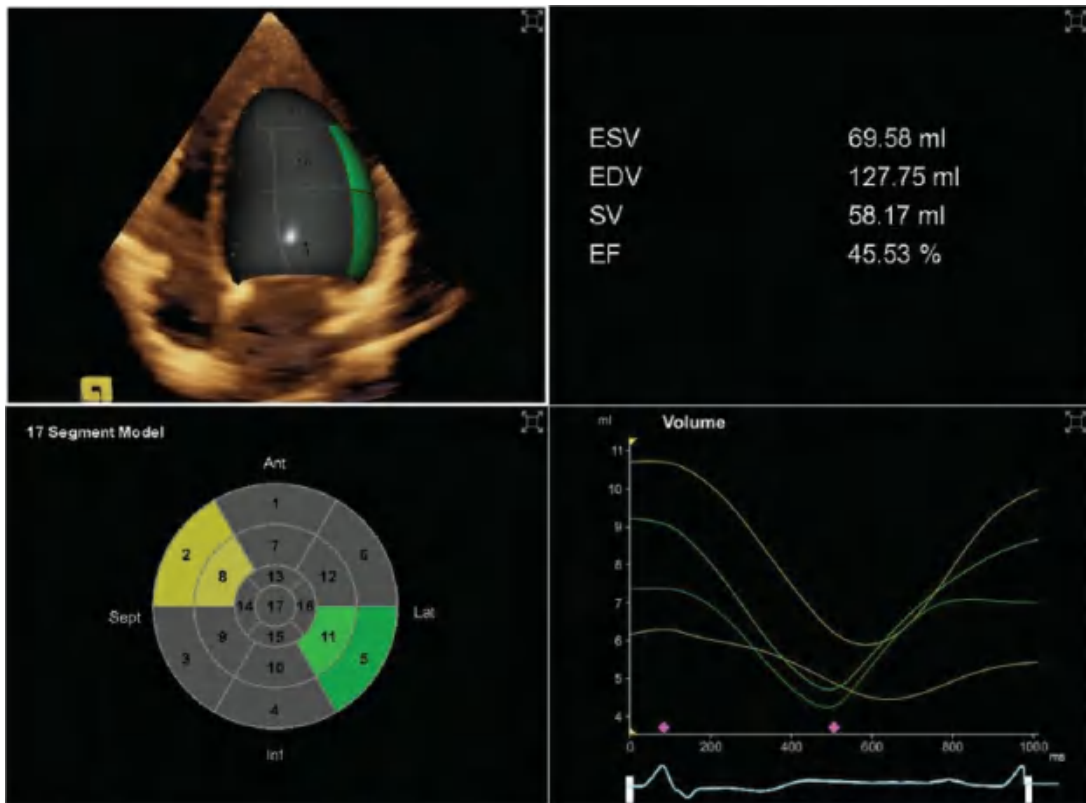
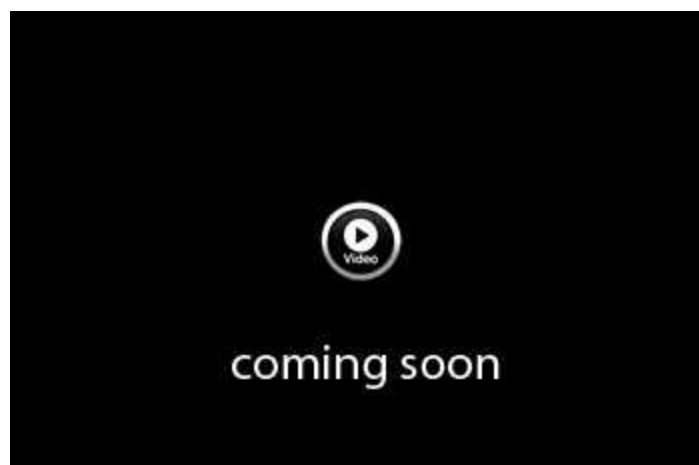


**FIGURE 5.15.** Full-volume three-dimensional imaging recorded in a patient with a left bundle branch block and mildly reduced left ventricular ejection fraction. Volumes, stroke volume, and ejection fraction are at the upper right. At the upper left a stylized shell has been fitted to the anatomically 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.

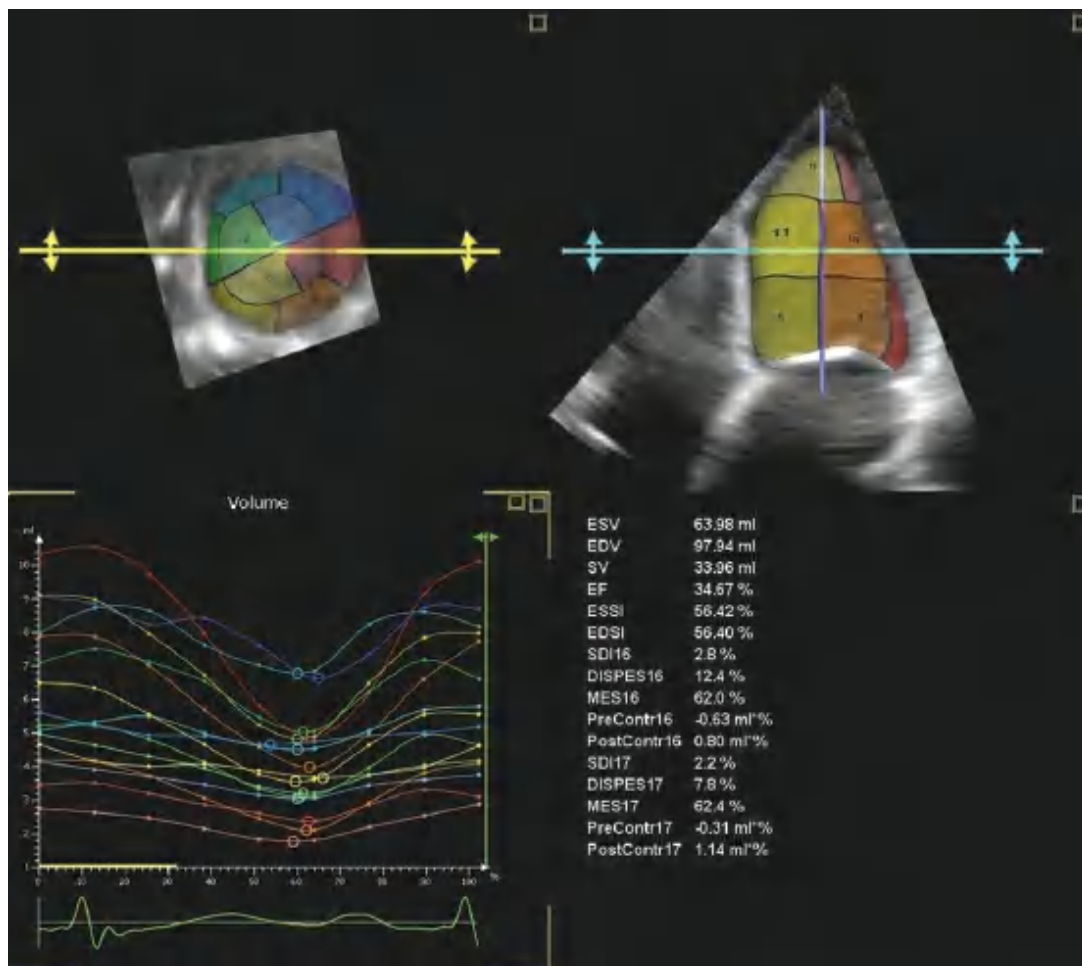


**FIGURE 5.17.** Transthoracic three-dimensional acquisition in a patient with an ischemic cardiomyopathy. The two upper panels are fitted models to the automatically determined endocardial border from the three-dimensional dataset. An apical view as well as a short-axis composite as viewed from the apex are presented. The individual graphs of volume over time in each of the 16 segments demonstrate regional variation in left ventricular function. The table at the right outlines a variety of automatically determined measures including an ejection fraction of approximately 35% as well as parameters of heterogeneity of contraction.

## 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.

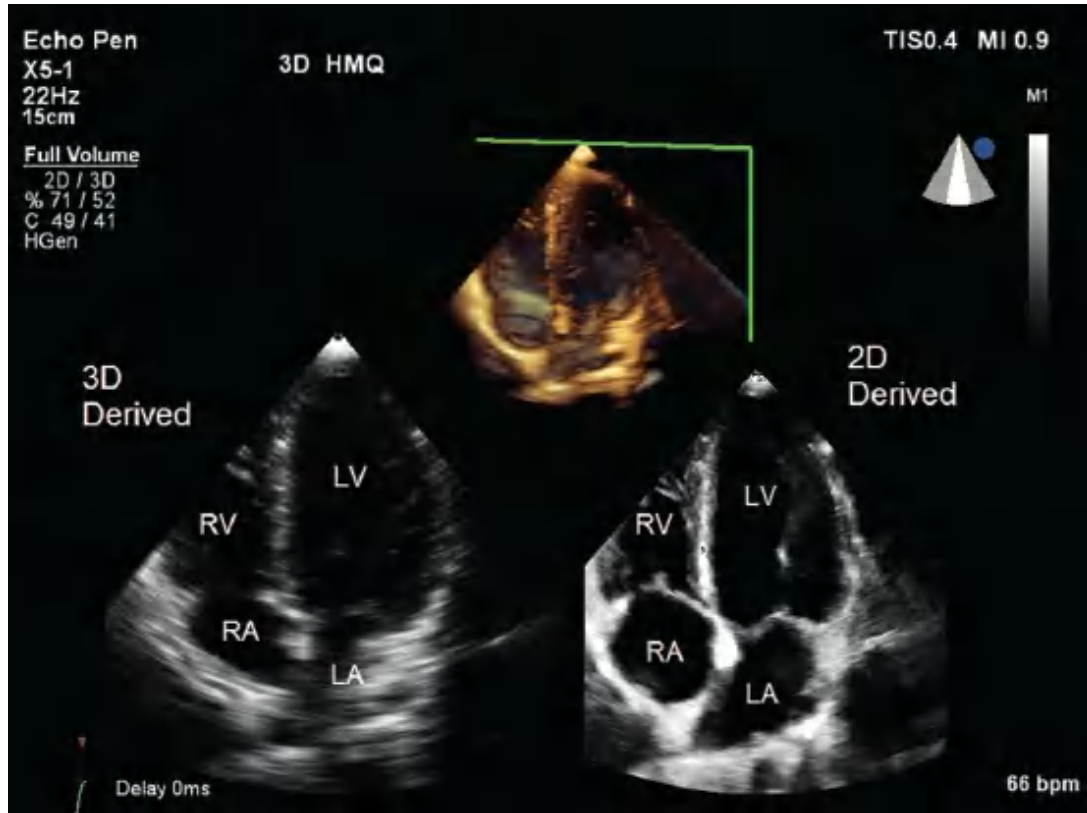
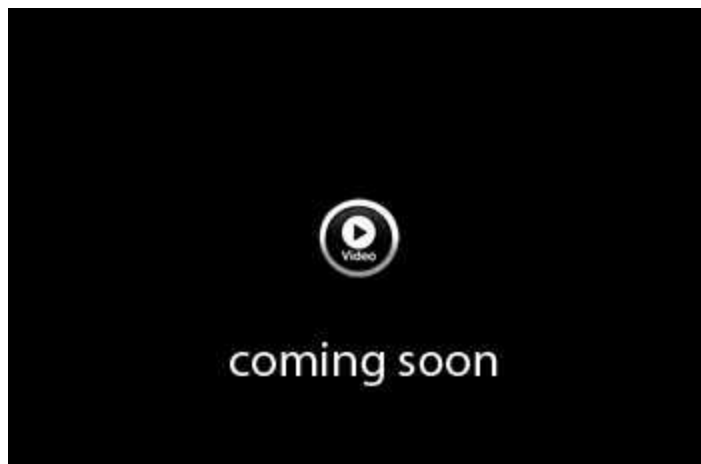


**FIGURE 5.18.** Comparison of the three-dimensionally and two-dimensionally derived apical four-chamber views in a patient with high image quality. The upper figure is the three-dimensional dataset from which apical four- and two-chamber views have been extracted. At the lower left is the three-dimensionally 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. 



**Video 5-18**



**Video 5-18\_2D**



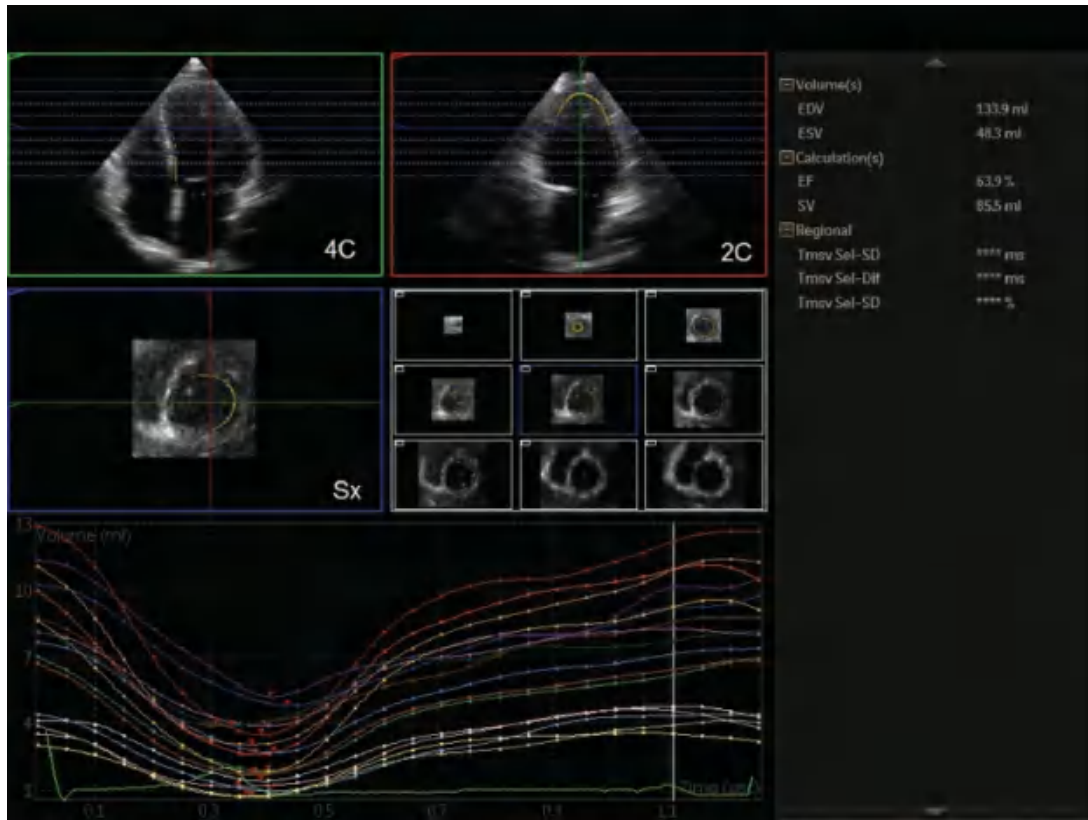
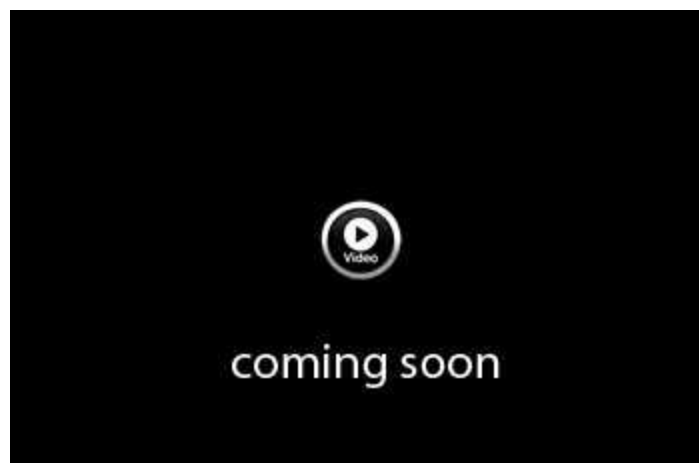


**FIGURE 5.19.** Multiple two-dimensional imaging planes have been extracted from a full-volume three-dimensional dataset. The upper panels show apical four- and two-chamber views and a single short-axis image which have been extracted from the full-volume dataset. At the lower right is a series of nine 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.



**Video 5-19**

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.

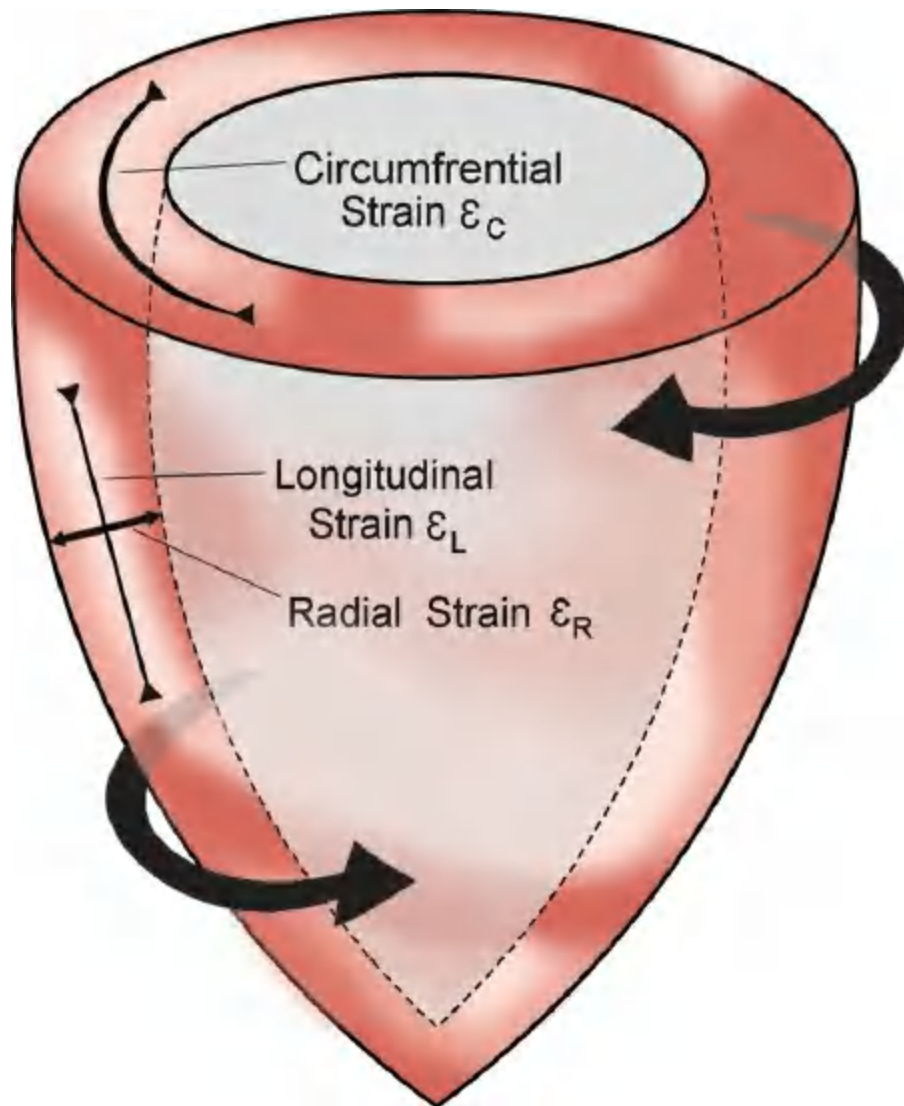


**FIGURE 5.20.** Schematic demonstration of the three orthogonally directed strain calculations. Longitudinal strain ( $\epsilon_L$ ) is defined as along the long axis of the left ventricle. Radial strain ( $\epsilon_R$ ) is orthogonal to the longitudinal strain and oriented perpendicular to the endocardial border. Circumferential strain ( $\epsilon_C$ ), 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.

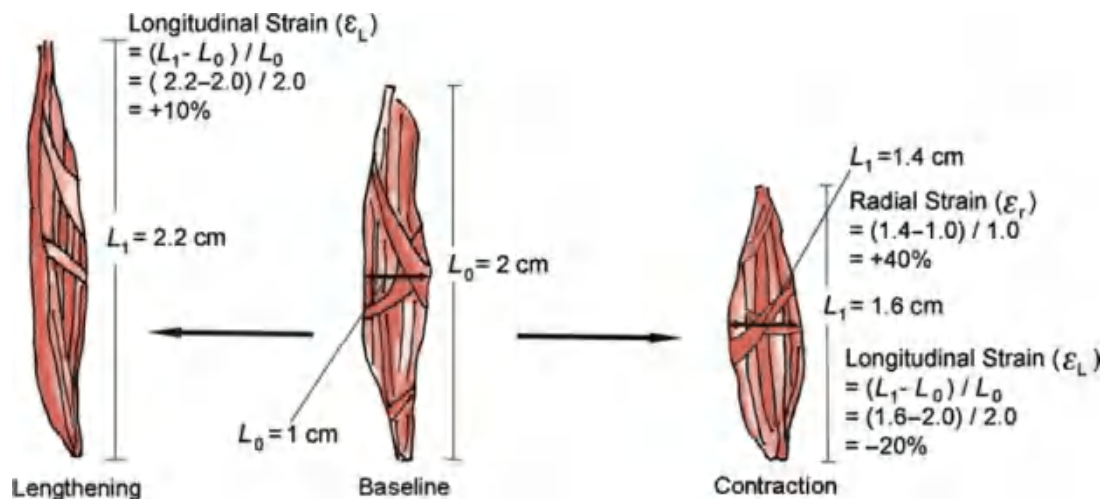


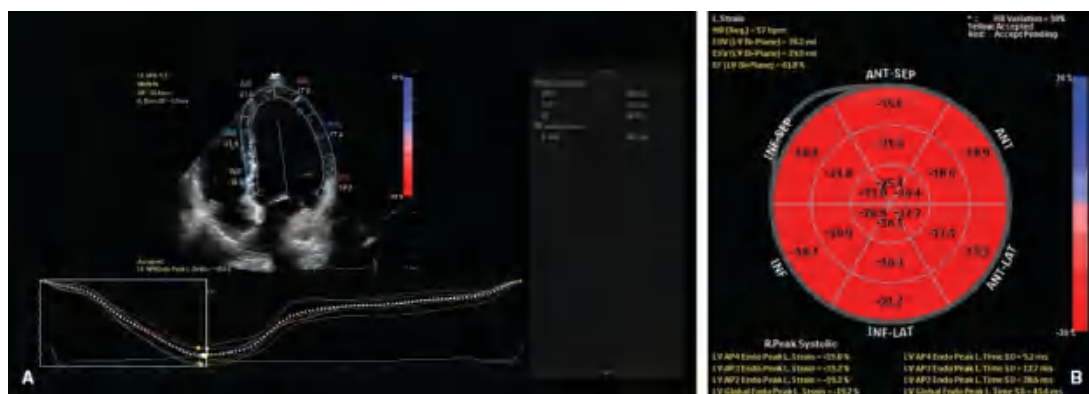
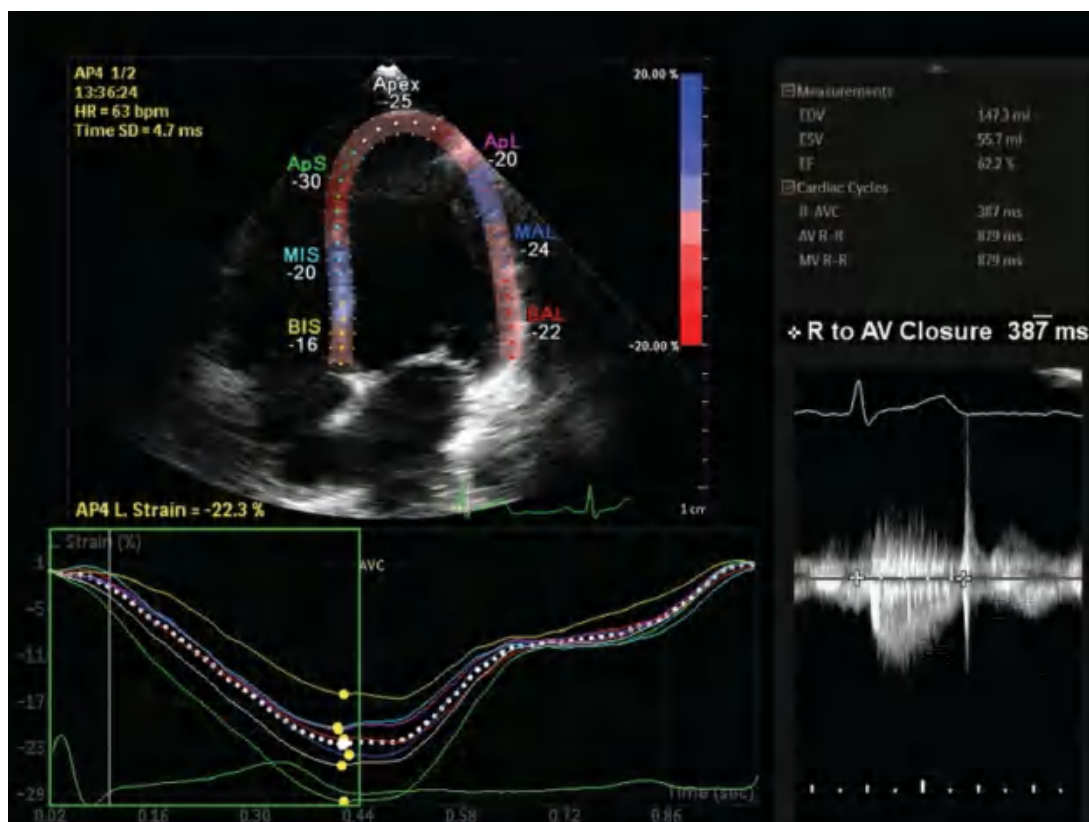
**FIGURE 5.21.** Demonstration of strain in a schematized myocardial segment. Both longitudinal strain ( $\epsilon_L$ ) and radial strain ( $\epsilon_R$ ) are calculated. Assuming a baseline length of 2 cm with contraction the myocardial segment decreases in length to 1.6 cm resulting in a longitudinal strain of  $-20\%$ . If the same fiber has lengthening (as noted on the **left**) to a 2.2 cm, longitudinal strain is calculated to  $+10\%$ . Radial strain is calculated perpendicular to the long axis and, in this instance, thickening of the myocardial segment from 1 to 1.4 cm results in a radial strain of  $+40\%$ . Note that with normal contraction, there is shortening in length but increase in width of the myocardial segment and, as such, normal longitudinal strain is negative and normal radial strain positive.

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.





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.

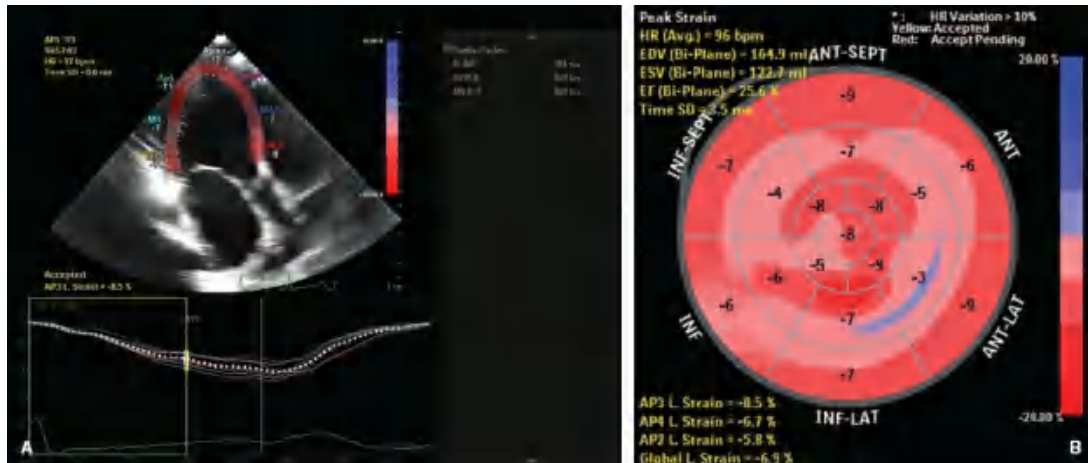
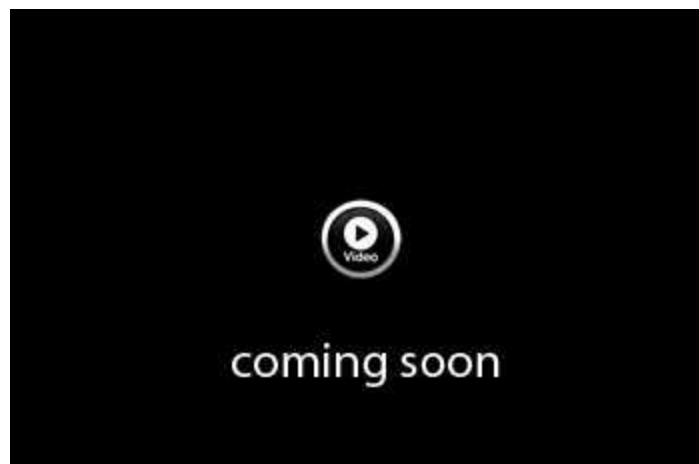


**FIGURE 5.24. A:** Longitudinal strain obtained in a patient with a dilated nonischemic cardiomyopathy. The apical long-axis view is presented. Individual graphs of the visualized seven segments are presented. **B:** Bull's-eye plot of the global longitudinal strain for 17 segments recorded in the same 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**



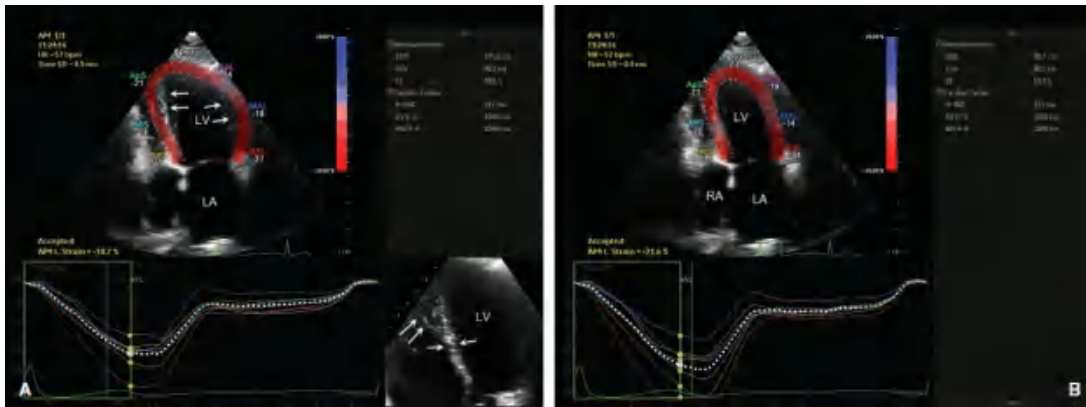
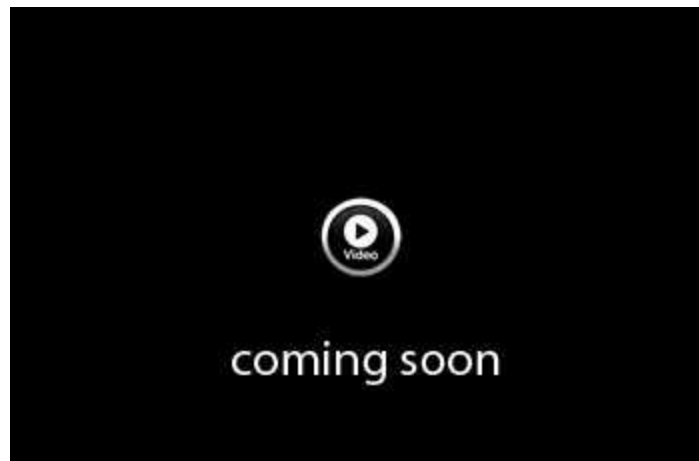
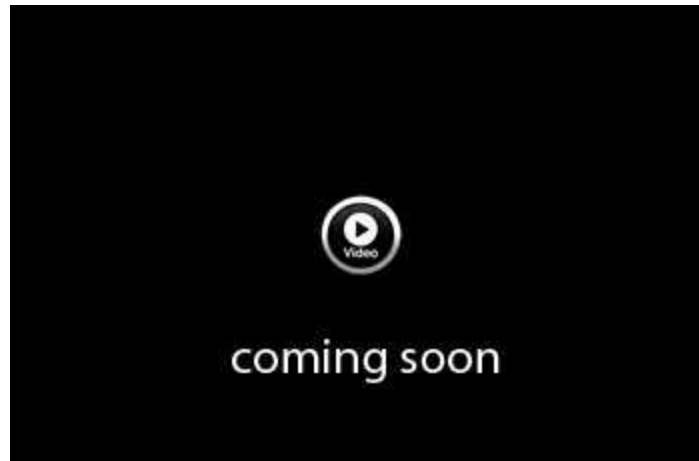


**FIGURE 5.25.** Apical four-chamber view recorded in a patient with less than ideal quality images. **A** represents an apical four-chamber view with superimposed tissue tracking for determination of longitudinal strain. This image is the first approximation created automatically by the ultrasound machine. 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**



**Video 5-25b**

**Table 5.4**

**DISEASES FOR WHICH MYOCARDIAL STRAIN IS ABNORMAL**

**PRIOR TO DETECTION OF TRADITIONAL FINDINGS**

Systemic disease

- Hypertension
- Diabetes mellitus
- Glycogen storage disease
- Cardiac amyloid

Primary myocardial disease

- Hypertrophic cardiomyopathy
- Dilated cardiomyopathy
- Adriamycin toxicity
- Cardiac rejection posttransplant

Coronary artery disease

- Low-grade ischemic
- Hibernation/stunning
- Stress-induced ischemia

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](#)).

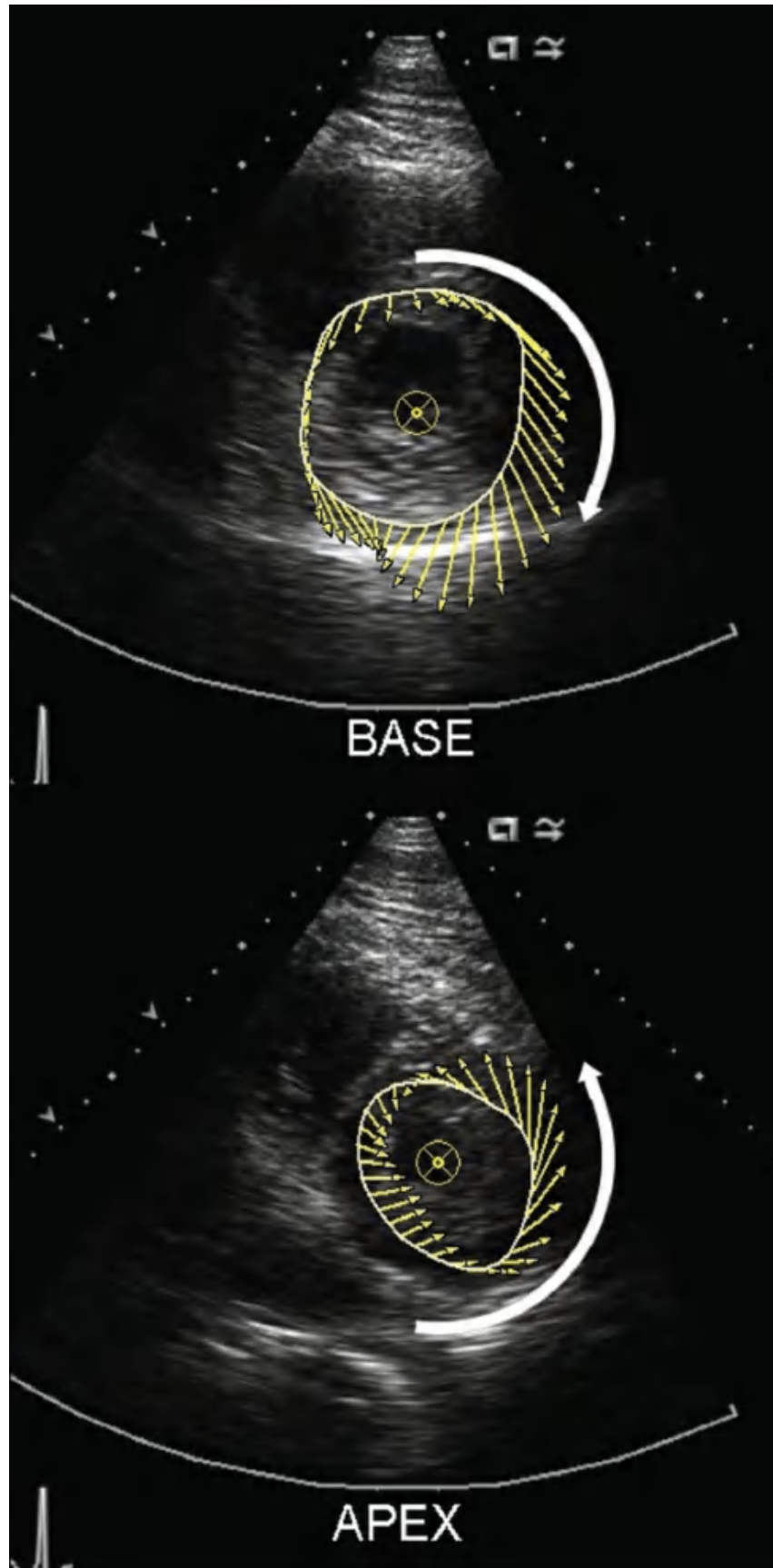


**FIGURE 5.26.** Parasternal short-axis view recorded at the base and apical levels in a patient with normal ventricular function. A modified speckle tracking algorithm has been used to track endocardial targets and displayed as a vector velocity map in which the length of an arrow represents magnitude 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.

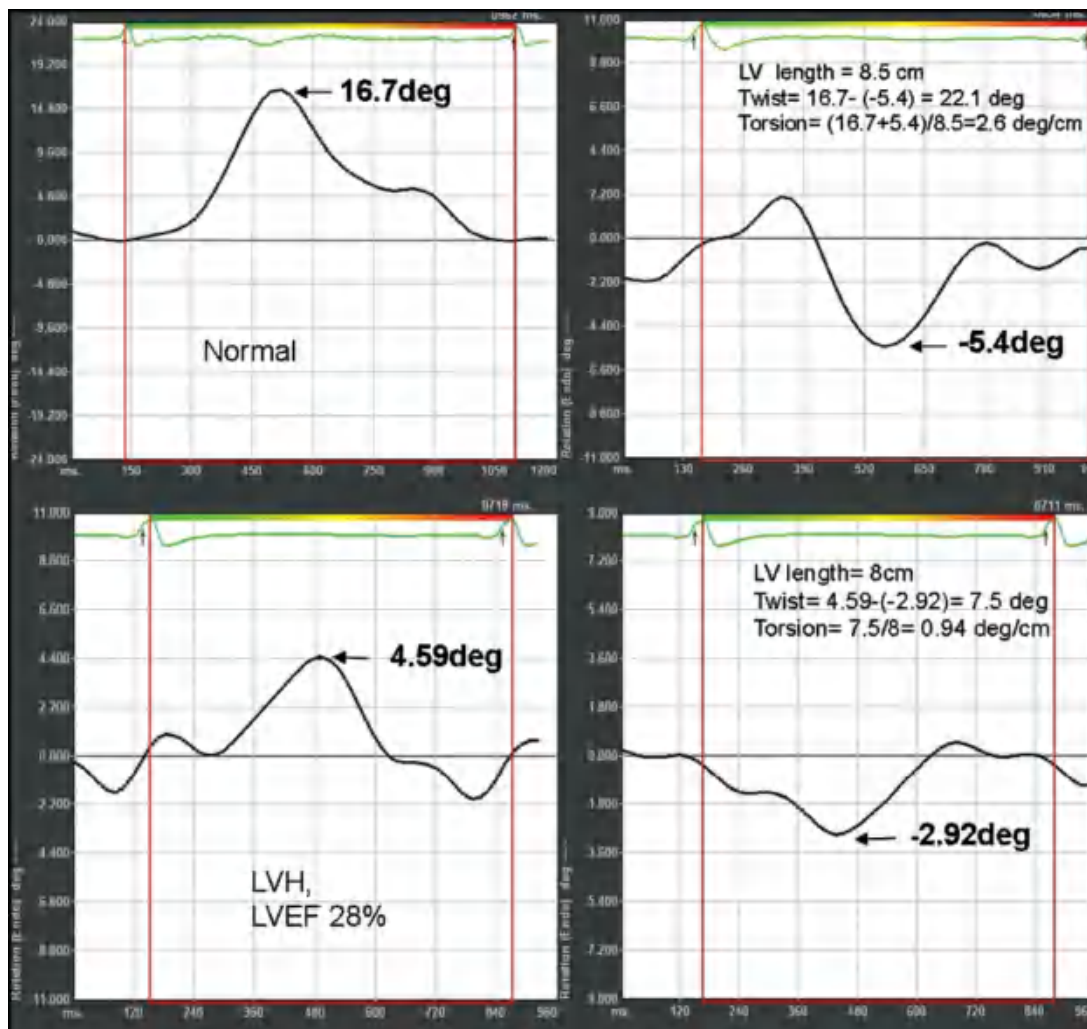


**FIGURE 5.27.** Graphic demonstration of angular rotation extracted from a normal patient in the upper panels and a patient with left ventricular hypertrophy (LVH) and reduced left ventricular ejection fraction (LVEF) in the lower panels. The left-hand panels are recorded at the apex and the right-hand 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.

**Table 5.5**

## METHODS FOR EVALUATION OF REGIONAL WALL MOTION

### ABNORMALITIES

#### Visual/subjective

Descriptive: normal, hypokinetic, akinetic, dyskinetic

Normal myocardial thickness versus scar

Location: anterior, lateral, inferior, posterior, apex, basal, mid, apical segments

#### Semi-quantitative

WMS or WMSI

Normal = 1

Hypokinetic = 2

Akinetic = 3

Dyskinetic = 4

Scored for each segment

$$WMSI = \sum_{n=1}^{n=N} WMS = N$$

#### Quantitative

Anatomy based

Radian change

Regional area change

Center-line chordal shortening

Doppler tissue imaging or speckle tracking

Local velocity

Velocity gradient (endocardial – epicardial)

Myocardial displacement

Myocardial strain



## Strain rate imaging

WMS, wall motion score; WMSI, wall motion score index.

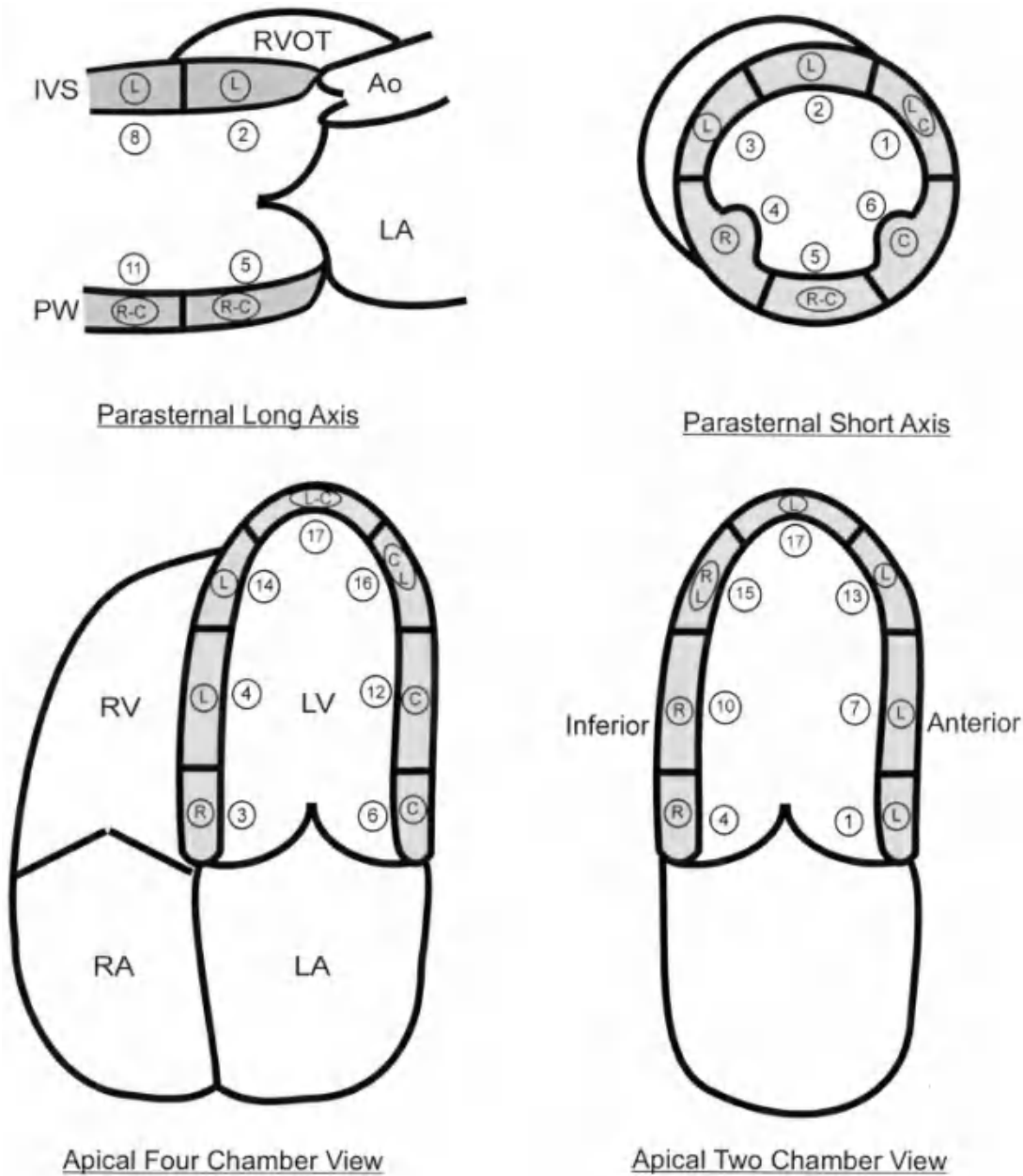


**FIGURE 5.28.** Schematic representation of the 17-segment model of the left ventricle. Parasternal and apical views are depicted. For the 16-segment model, each of the distal segments (13 to 16) incorporates its adjacent portion of the apical segment. For each segment, the coronary distribution most 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

coronary artery, it may also be perfused by that artery. PW, posterior wall.

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.

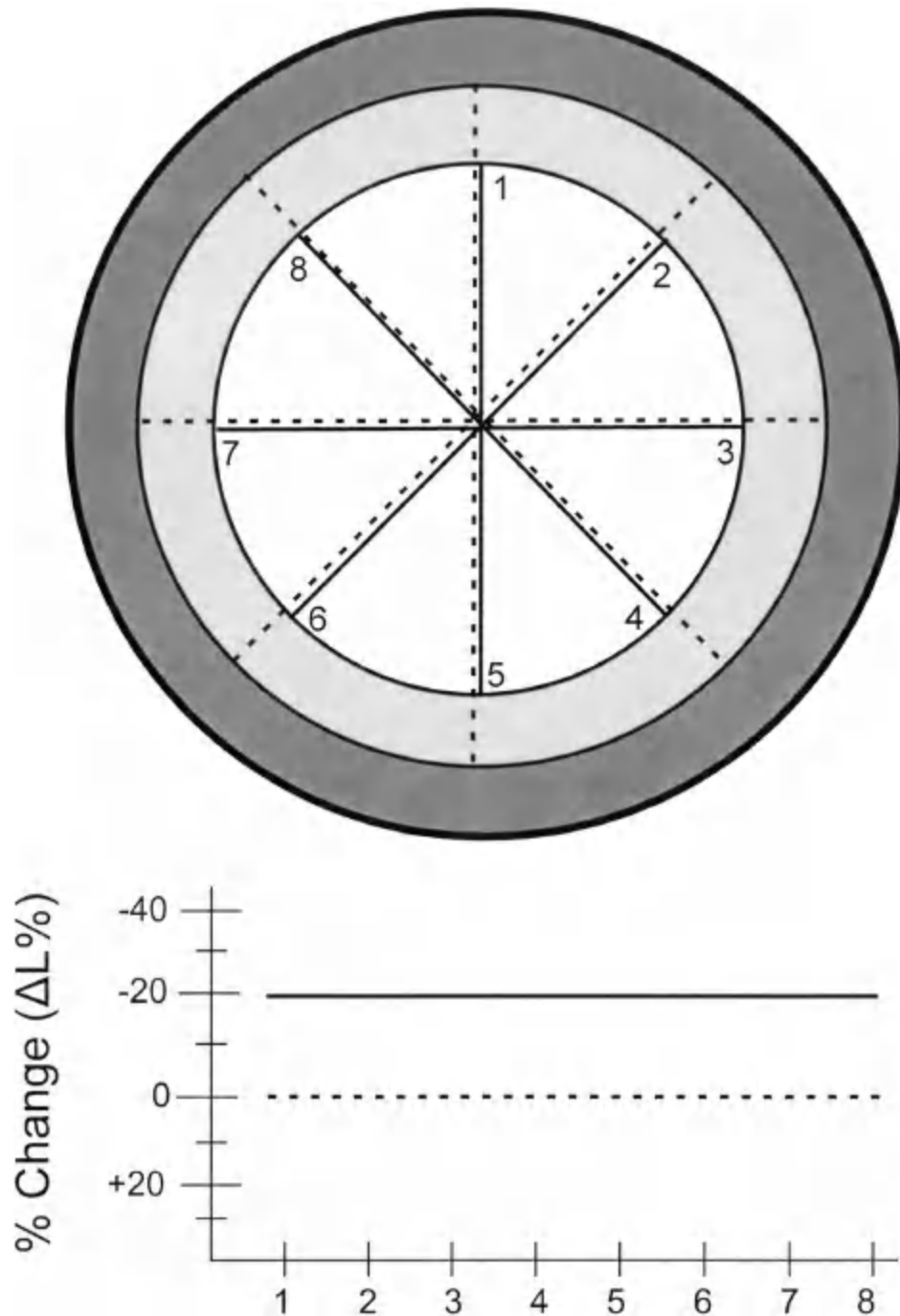


**FIGURE 5.29.** Schematic diagram of normal endocardial wall motion without translational motion. **Top:** The *outer dark circle* represents the diastolic thickness of the left ventricle and the *inner lighter shaded circles* represent the extent of systolic contraction. Eight radii from the center 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.

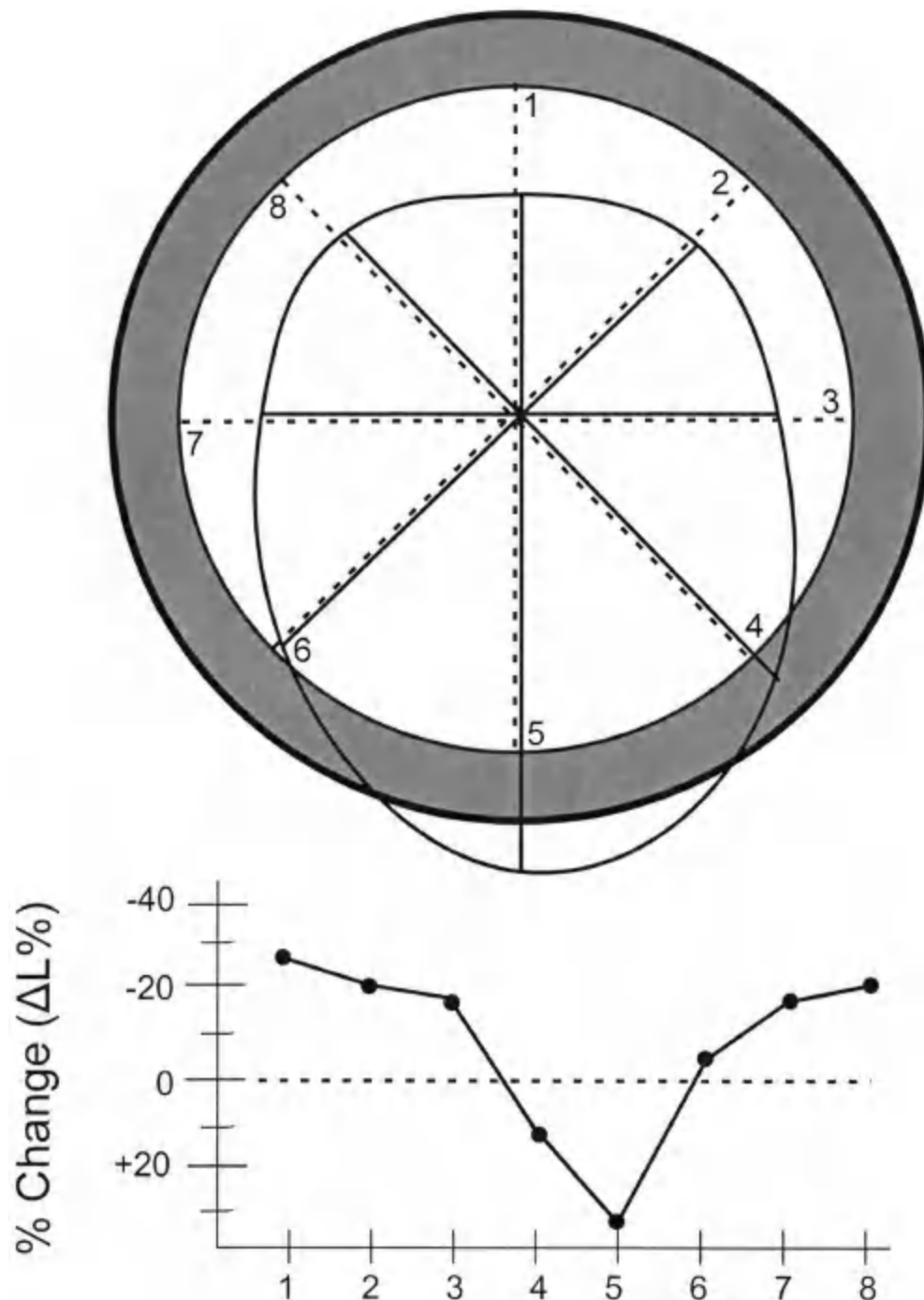


**FIGURE 5.30.** Schematic demonstration of posterior dyskinesia with no translational or rotational motion using the diastolic center of mass for both systole and diastole. **Top:** The *darker outer ring* represents the contour of the ventricle in diastole and the *inner circle* represents the endocardial contour in systole. Note the maximal area of dyskinesia at segment five with less dyskinesia

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](#).

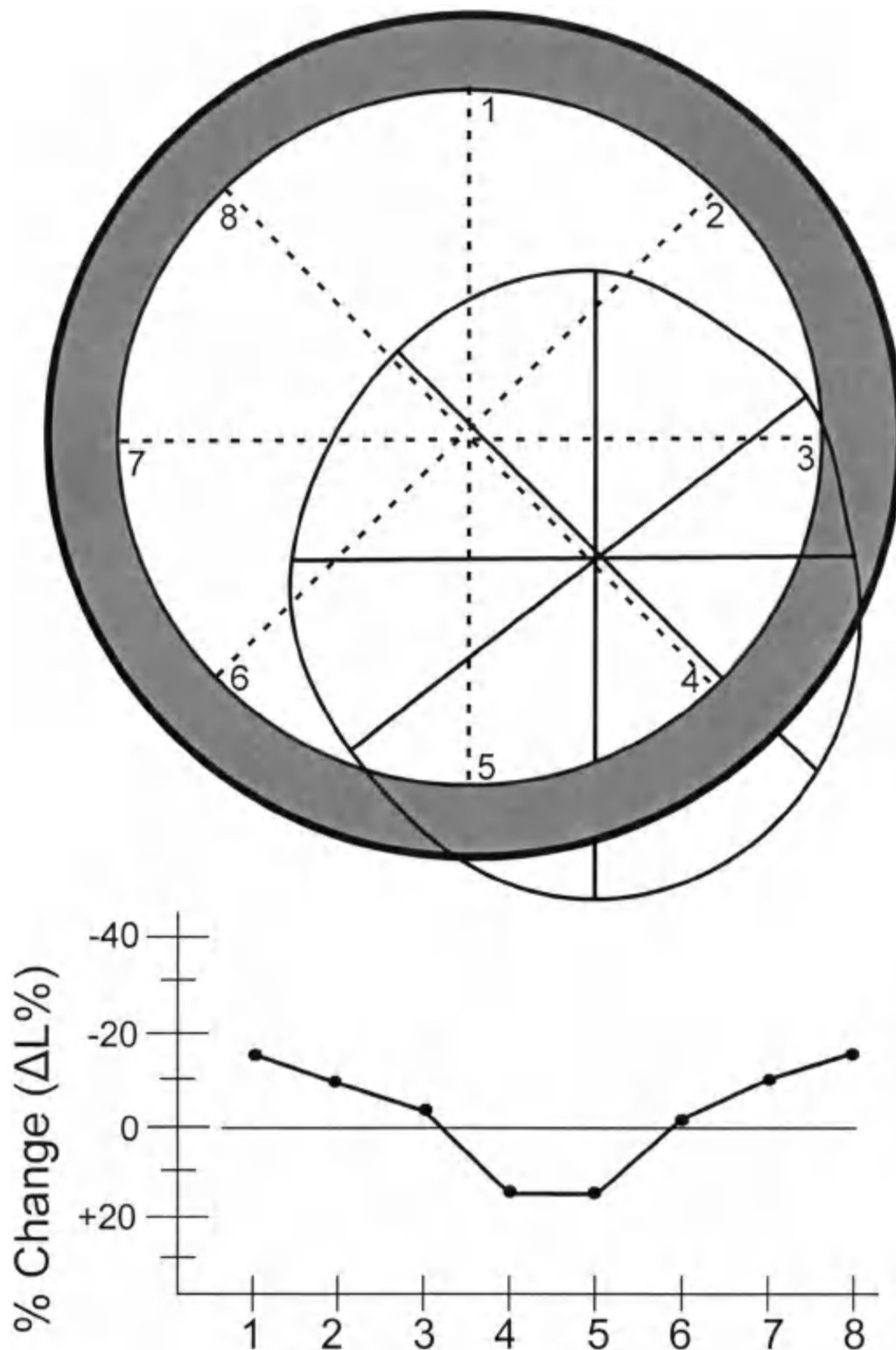


**FIGURE 5.31.** Schematic representation of posterior dyskinesis with posterolateral translation, using separate diastolic and systolic centers of mass for determining radian length. Note that because there is posterior dyskinesis, the systolic center of mass moves toward the dyskinetic wall, resulting in an apparent reduction in the degree of dyskinesia 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  $\text{left ventricular mass} = (\text{interventricular septum} + \text{left ventricular interior dimension} + \text{posterior wall})^3 - \text{left ventricular interior dimension}^3$  (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 ( $1.05 \text{ g/cm}^3$ ), 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.

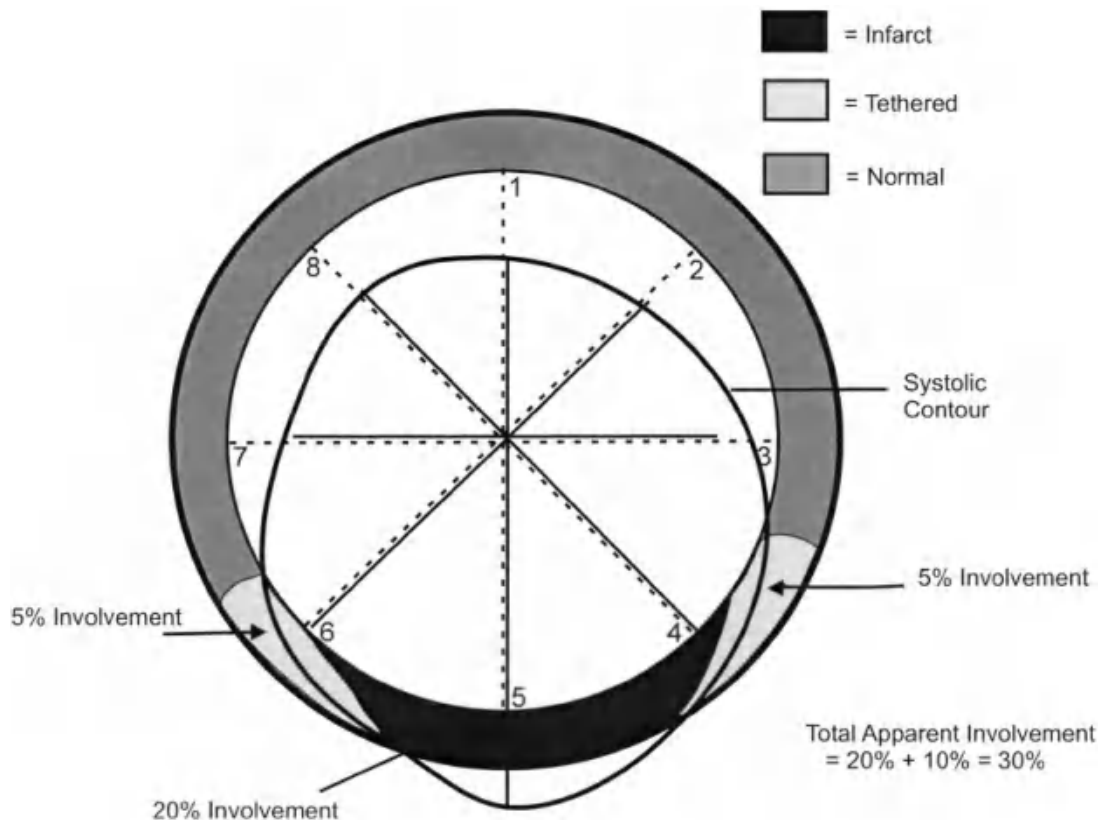


**FIGURE 5.32.** Schematic representation of horizontal tethering. This diagram represents posterior dyskinesis without translational motion. Note that the true extent of the infarct is as noted in the *darkly shaded area*, encompassing radian five and parts of radians six and four. Note that there is 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%.

#### Chapter 5 Evaluation of Systolic Function of the Left Ventricle

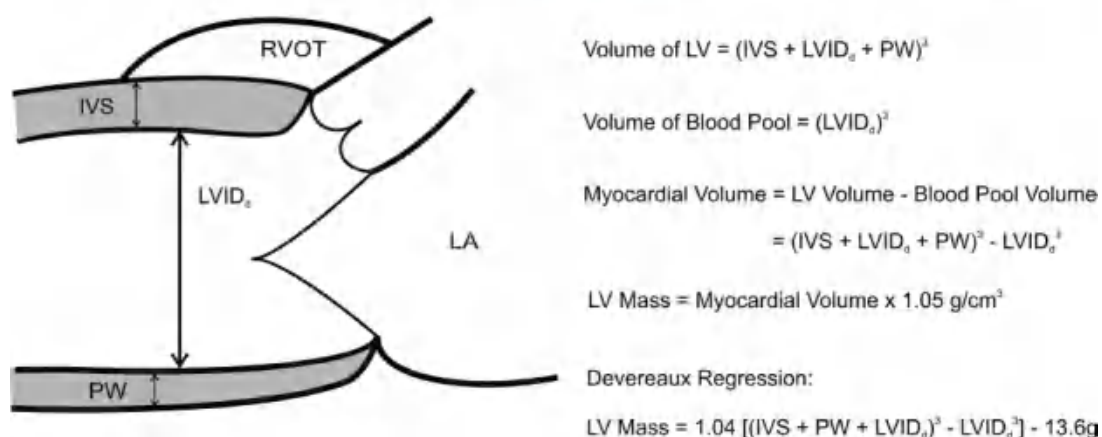


**FIGURE 5.33.** Schematic representation of the cubed formula for determining left ventricular mass. All measurements can be taken from either a two-dimensional

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. LVID<sub>d</sub>, left ventricular internal dimension in diastole; PW, posterior wall.

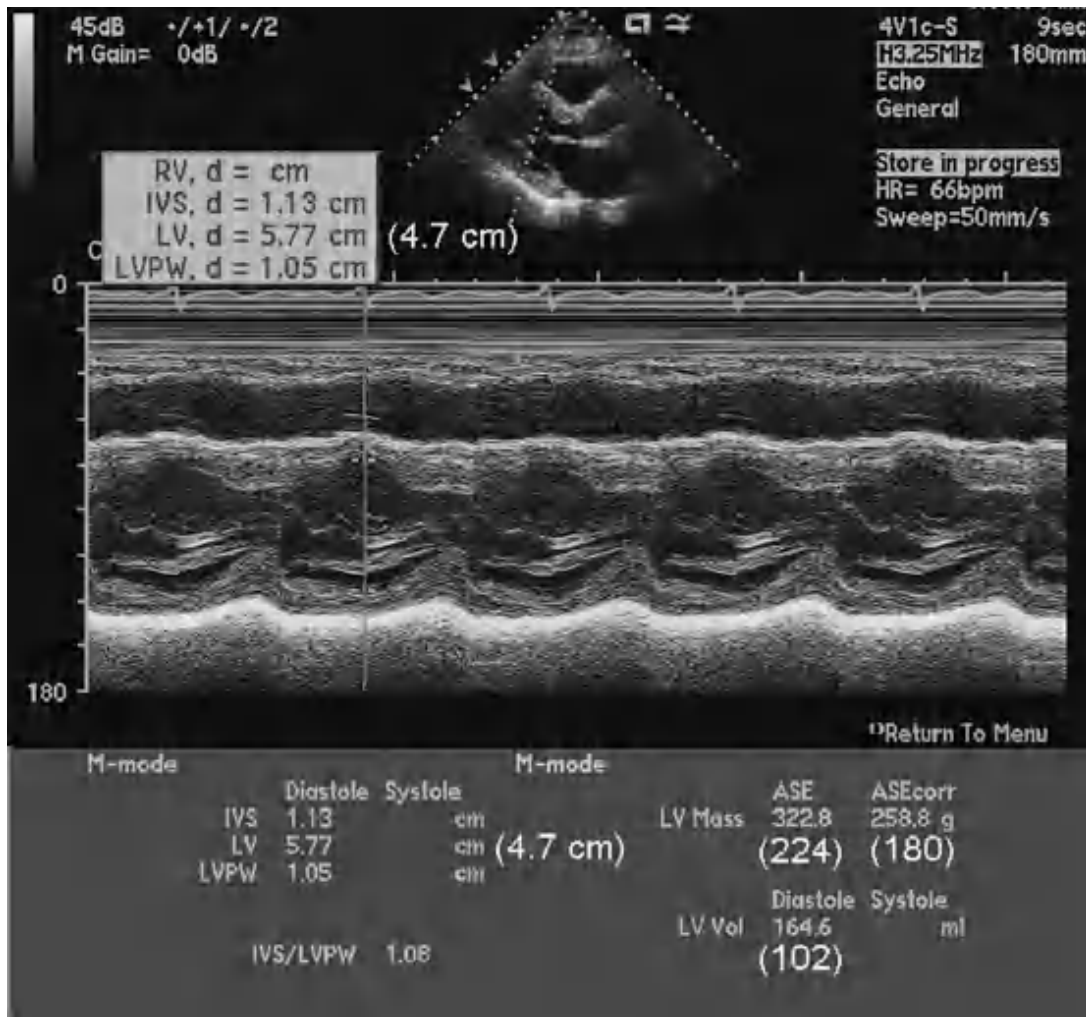


**FIGURE 5.34.** Two-dimensionally guided M-mode echocardiogram recorded in a patient with mild hypertension. Note in the small inset, the tangential M-mode interrogation beam which is a result of beam orientation and slight angulation of the heart. The M-mode is as displayed from which a left ventricular 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  $\geq 0.42$  has been used as a threshold of pathologic left ventricular hypertrophy.

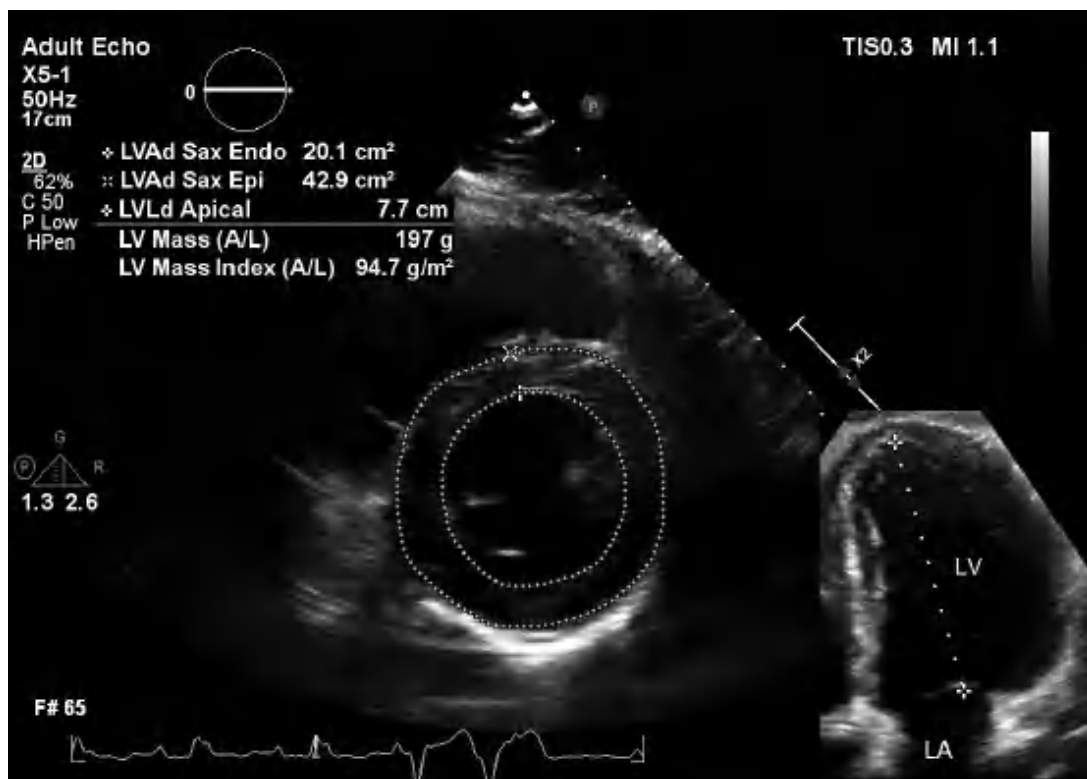


**FIGURE 5.35.** Illustration of methodology for calculating left ventricular mass using two-dimensional imaging. In the central figure note that the epicardium and endocardium have been outlined in the short-axis view of the left ventricle from which mean wall thickness is calculated (*dotted lines*). 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.

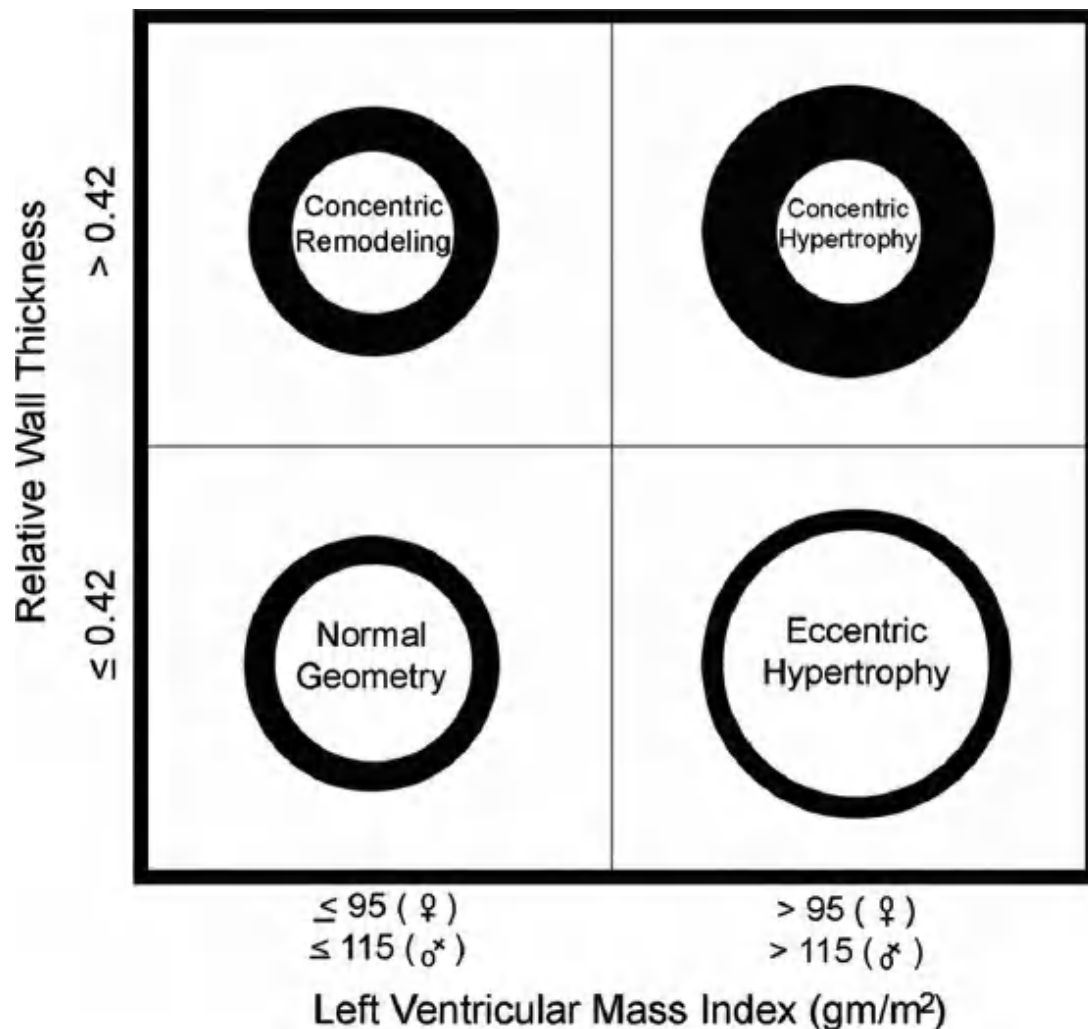


**FIGURE 5.36.** Graphic demonstration of normal geometry, concentric remodeling, concentric hypertrophy, and eccentric hypertrophy. American Society of Echocardiography recommended thresholds for defining hypertrophy are as noted.

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)  $\pm$  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.



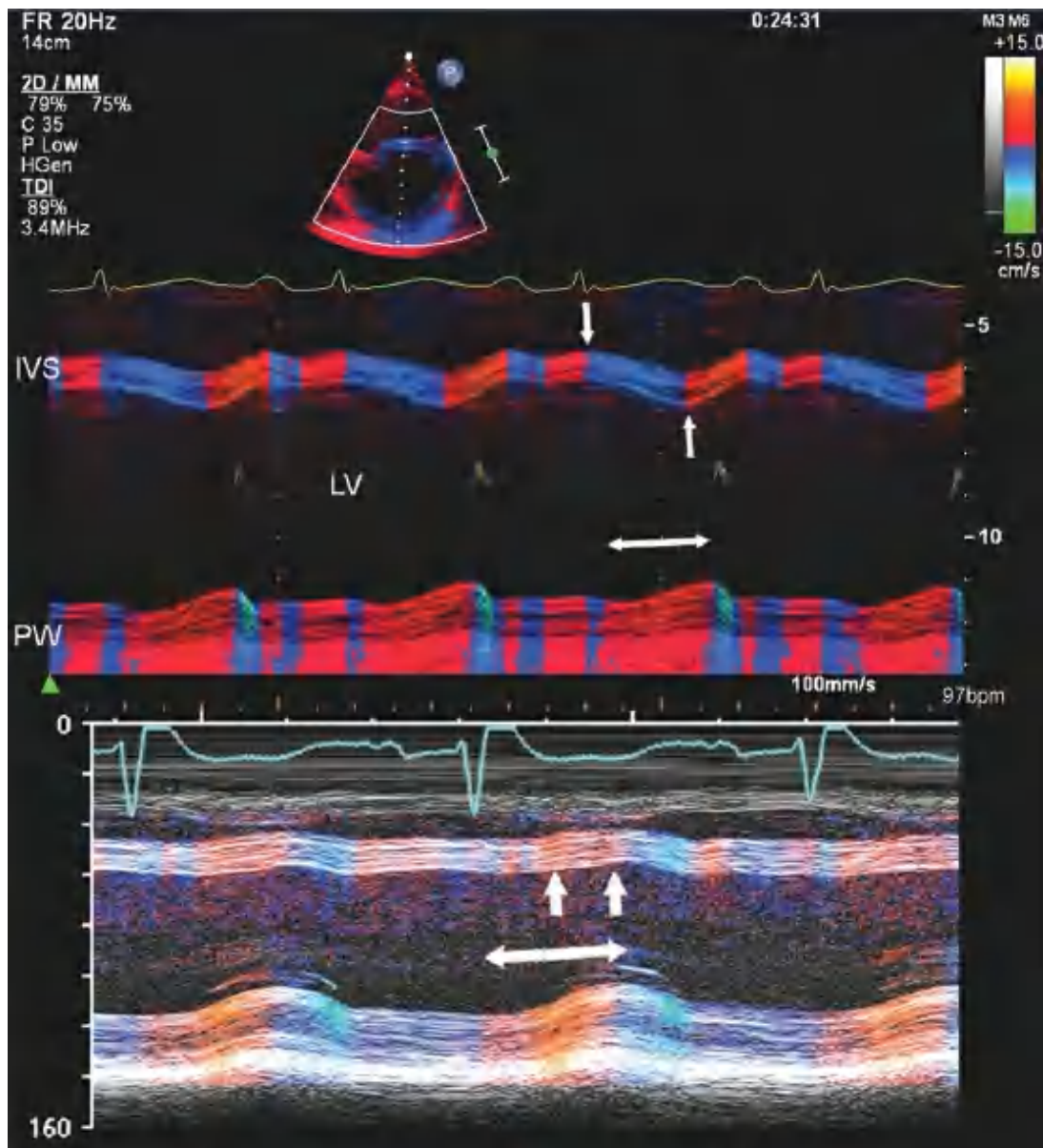


**FIGURE 5.37.** Color tissue Doppler M-mode echocardiograms recorded in a normal patient in the upper panel and in a patient with anteroseptal dyskinesia in the lower panel. Notice in the upper panel, the abrupt blue colorization timed with the QRS (*downward-pointing arrow*) of the ventricular septum 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 dyskinesia. 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 dyskinesia (*small arrows*). PW, posterior wall.

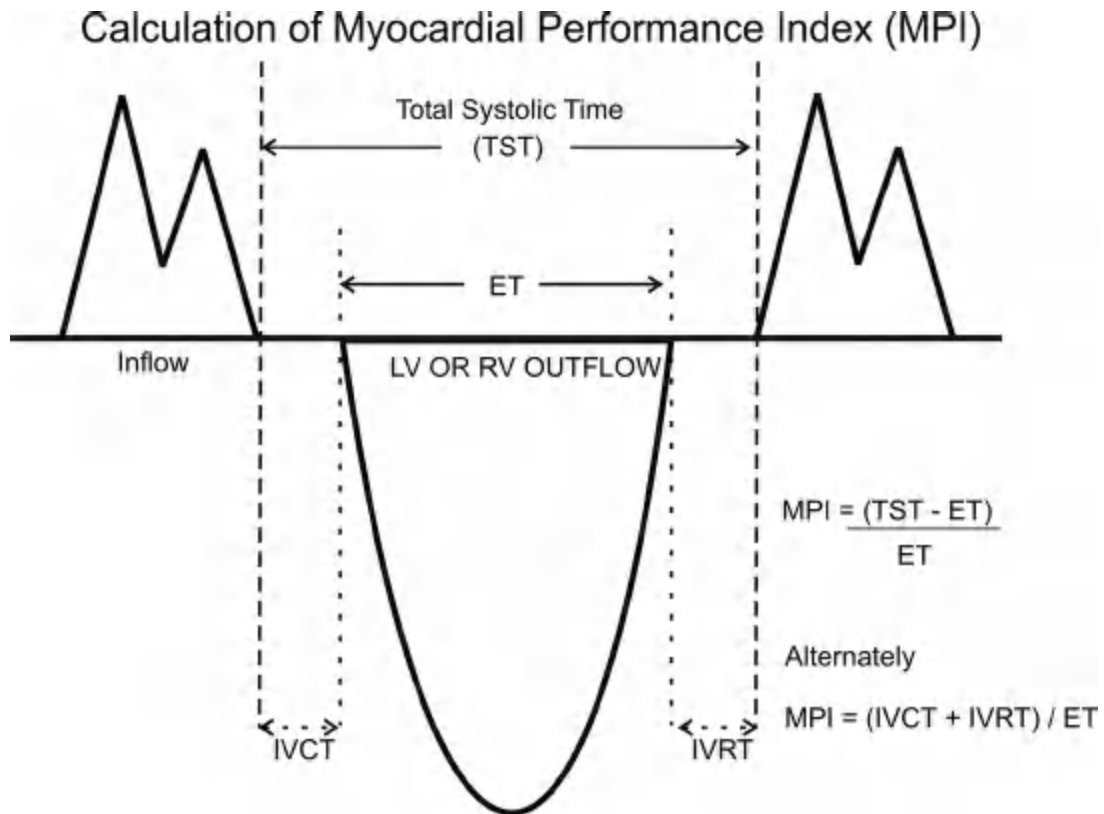


**FIGURE 5.38.** Schematic outlining calculation of the myocardial performance index (MPI). The myocardial performance index is the ratio of the sum of the isovolumic contraction and relaxation times (IVCT, IVRT) to ejection time (ET). It can be calculated by subtracting ET from total systolic time (TST) as noted in the two alternate formulas. Normal MPI is  $\leq 0.40$ .



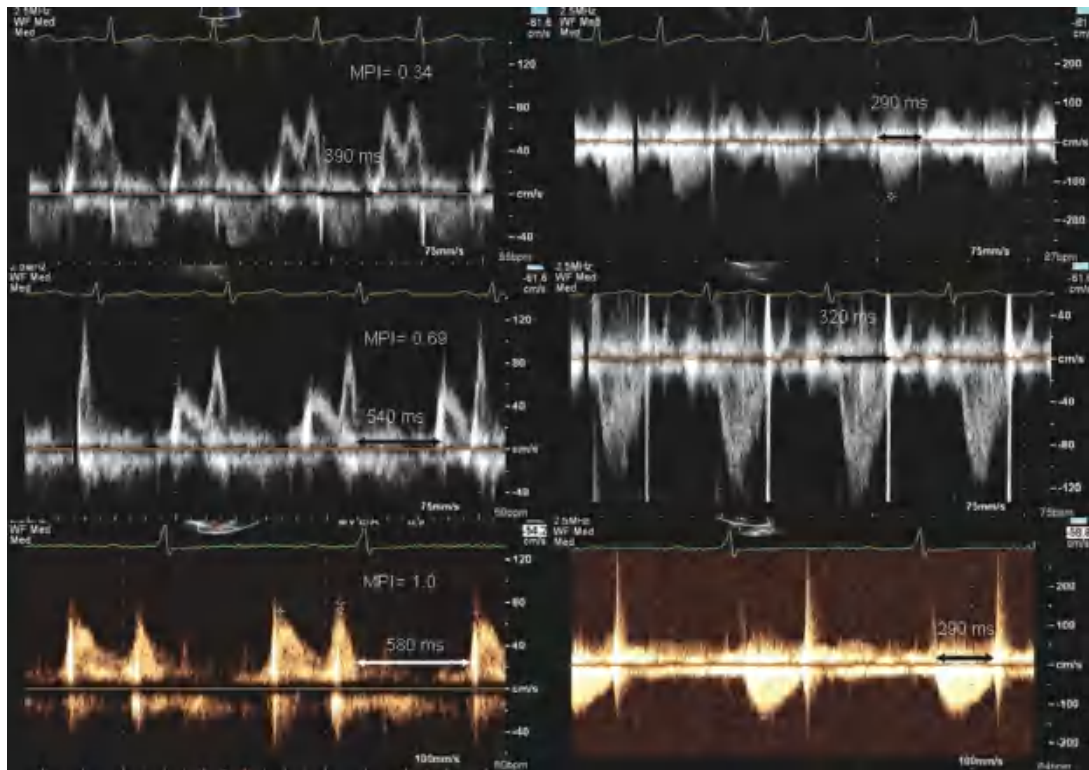


**FIGURE 5.39.** Composite illustration of myocardial performance index (MPI) calculated in three different patients. For each patient, the mitral inflow and left ventricular outflow track velocities are provided from which the time from mitral closure to mitral opening and ejection time are calculated. 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  $dP/dt = 32 \text{ mm Hg} / \text{time (ms)}$ . 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.

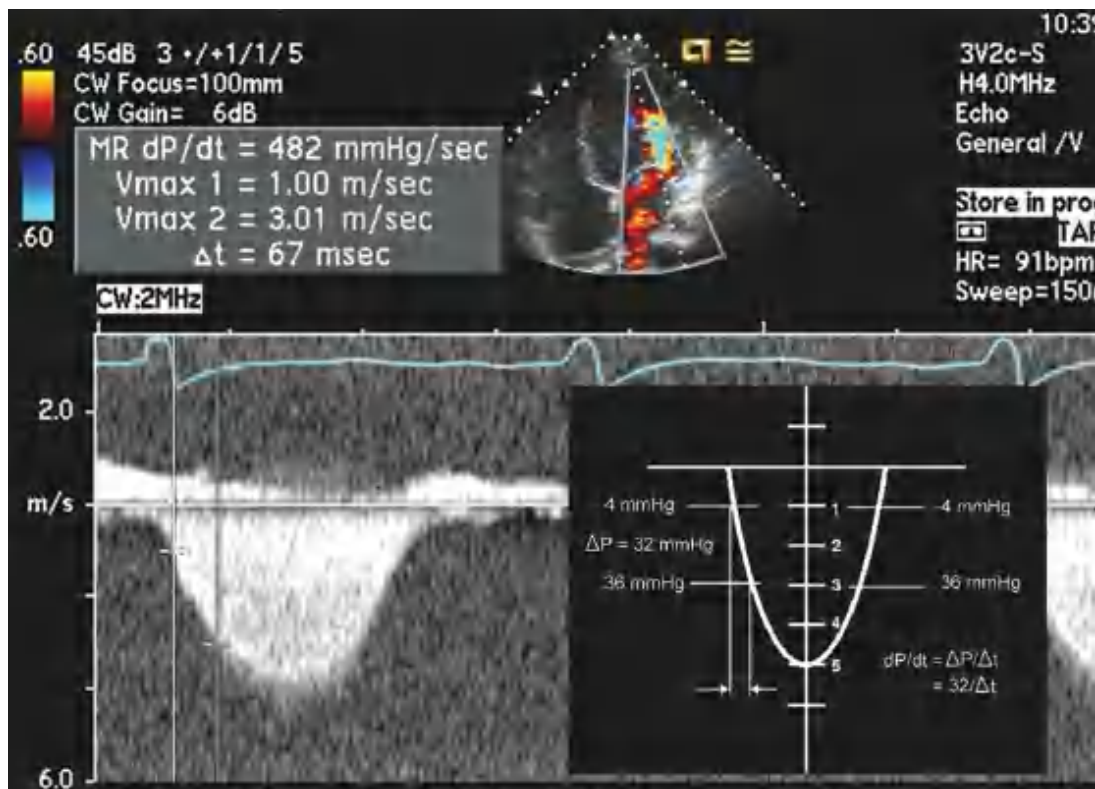


**FIGURE 5.40.** Schematic representation and example of calculating the left ventricular  $dP/dt$  from the continuous-wave Doppler mitral regurgitation spectral signal. **Left:** A continuous-wave spectral Doppler image recorded in a patient with severe left ventricular systolic dysfunction in which 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 ( $\Delta t$ ) is then divided into the pressure difference (32 mm Hg) for calculation of  $dP/dt$ .

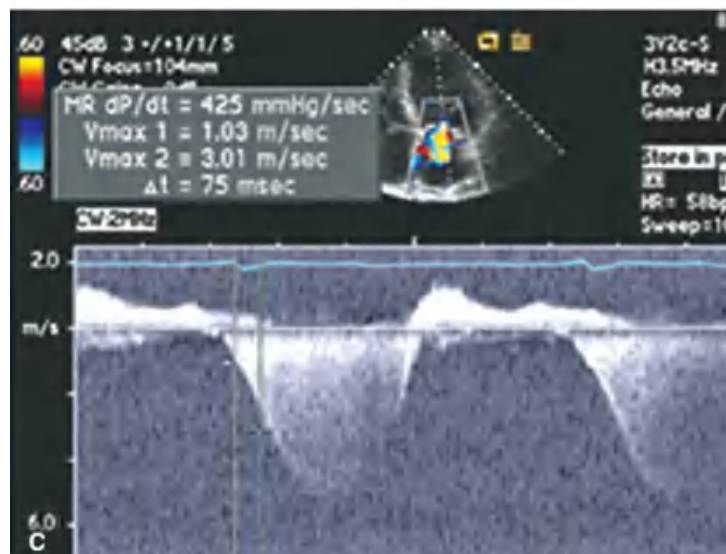
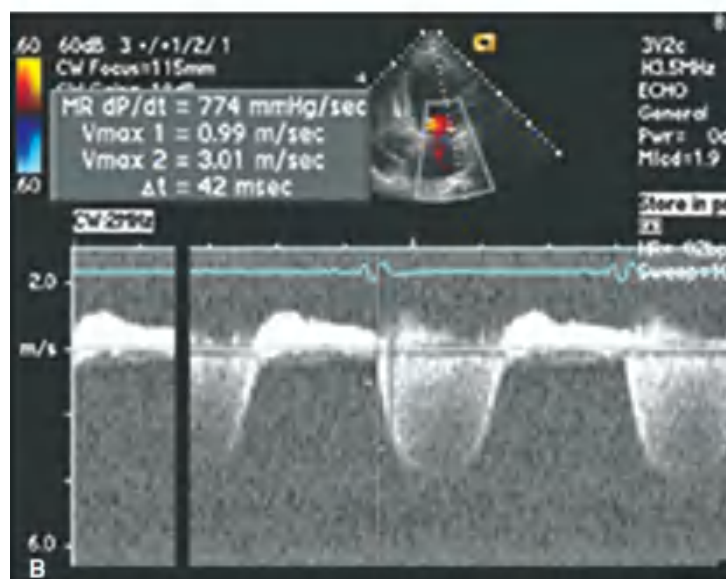
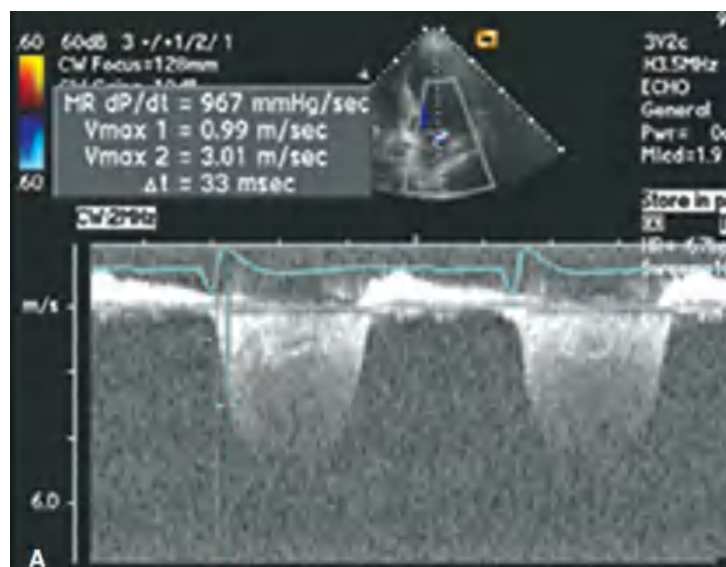


**FIGURE 5.41.** Continuous-wave Doppler imaging–derived left ventricular  $dP/dt$  in three patients with varying degrees of left ventricular systolic dysfunction. **A:** Recorded in a patient with relatively mild systolic dysfunction and a  $dP/dt$  of 967 mm Hg/s. **B:** Recorded in a patient with intermediate dysfunction. **C:** Recorded in a patient with severe systolic dysfunction and a  $dP/dt$  of 425 mm Hg/s.

## 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:  $\text{stress} = (\text{pressure} \times \text{radius}) \div h$  (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 combining 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.



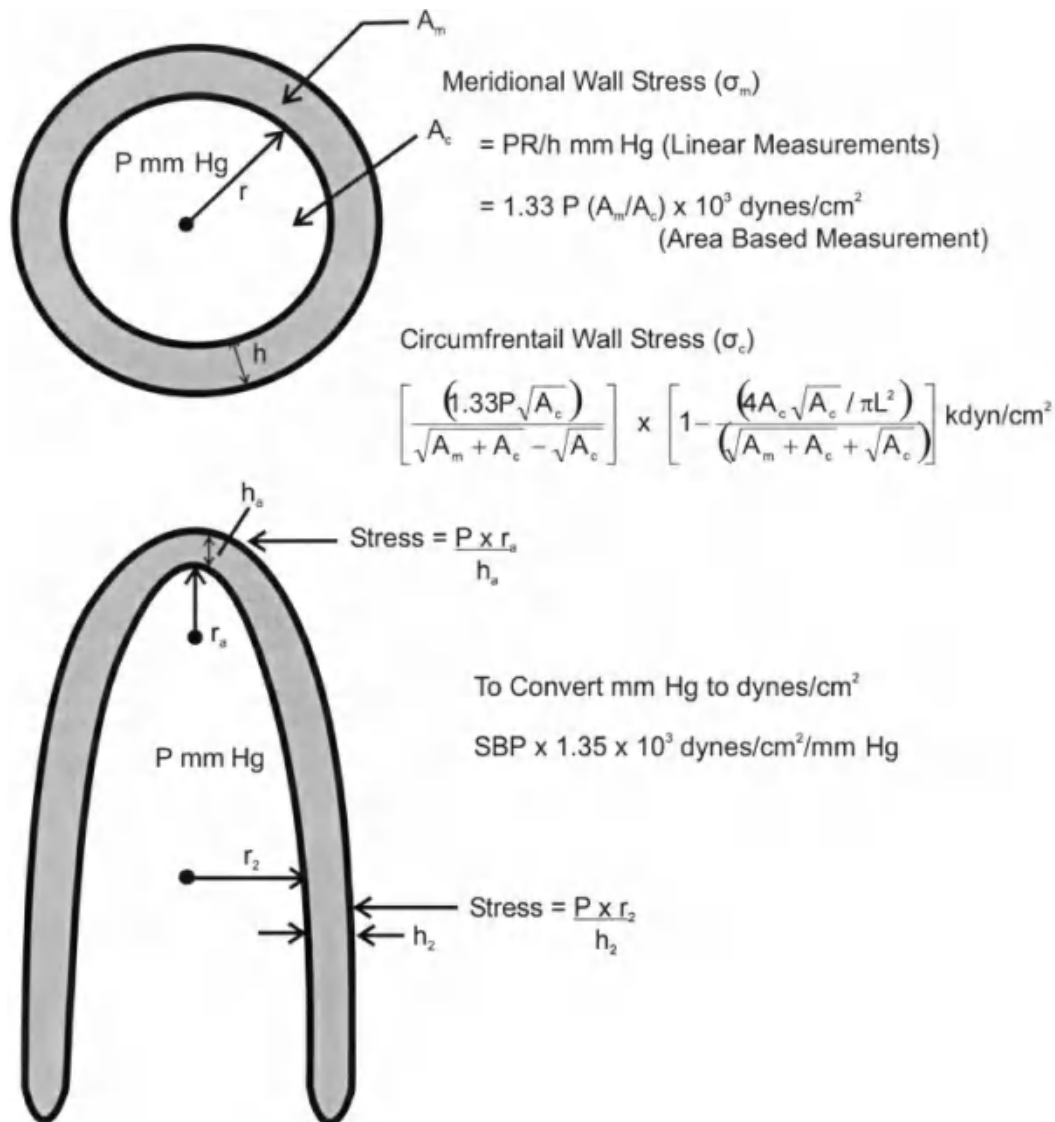


**FIGURE 5.42.** Schematic representation of the simplified methods for determining left ventricular wall stress. Wall stress can be defined as radial, circumferential, or meridional, all of which are mutually orthogonal. Meridional wall stress is the simplest to calculate. Circumferential wall stress 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.

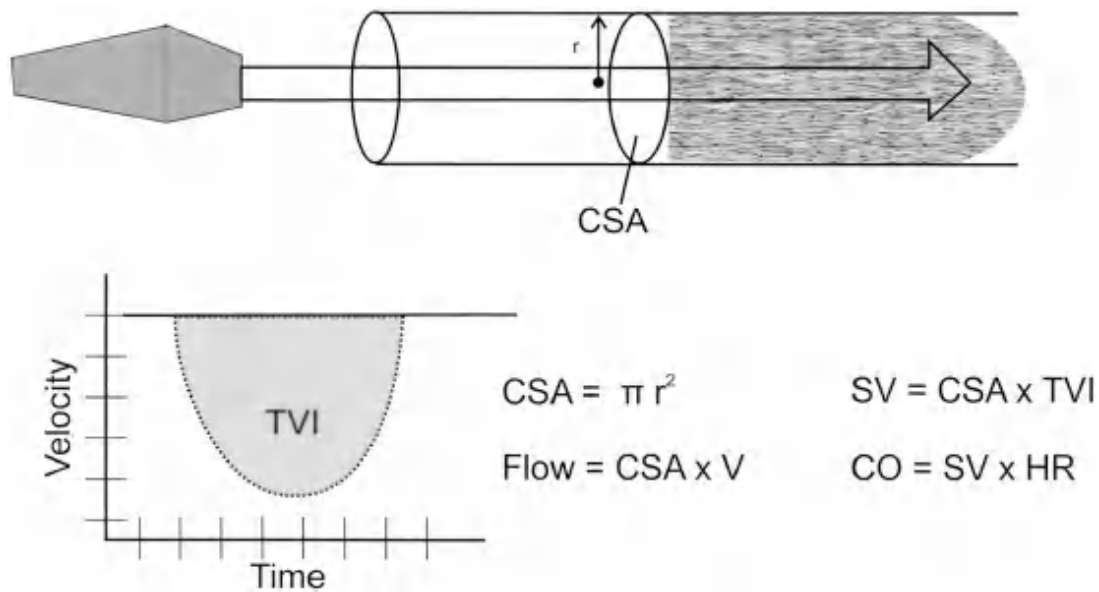


**FIGURE 5.43.** Schematic representation of the method for determining volumetric flow. This method is applicable for any laminar flow for which the cross-sectional area (CSA) of the flow chamber can be determined. The product of cross-sectional area and the time velocity integral (TVI) is stroke volume (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.

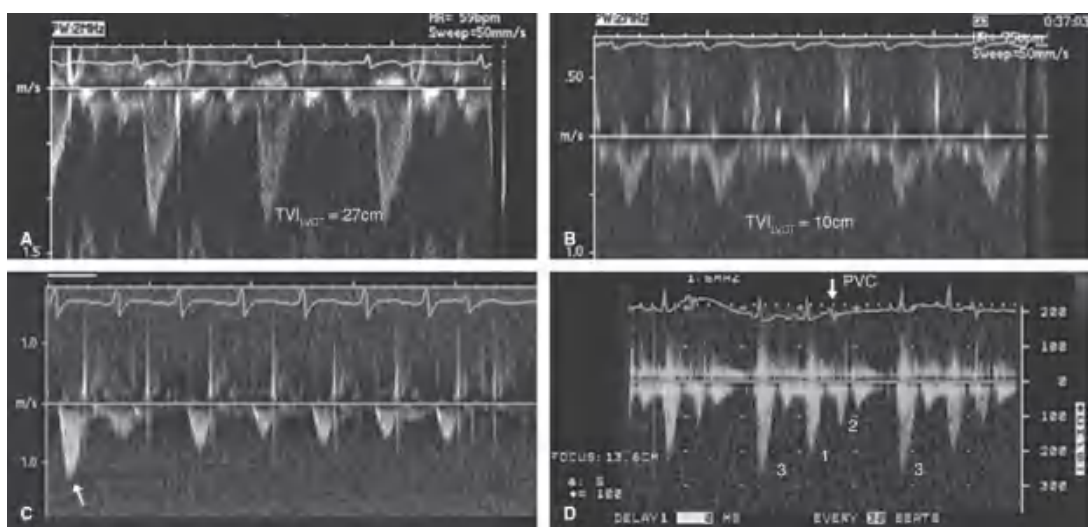


**FIGURE 5.44.** Time velocity integral (TVI) in the left ventricular outflow tract recorded in four different patients. **A:** Note the TVI of 27 cm recorded in a patient

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.

**Table 5.6**

#### NONISCHEMIC REGIONAL WALL MOTION ABNORMALITY

Conduction system based  
 Left bundle branch block  
 Ventricular pacing  
 Premature ventricular contractions

Ventricular pre-excitation (Wolf–Parkinson–White syndrome)

Abnormal ventricular interaction

Right ventricular volume overload

Right ventricular pressure overload

Pericardial constriction

Miscellaneous

Tardokinesis

Early relaxation

After cardiac surgery

Congenital absence of the pericardium

Posterior compression

Ascites

Hiatal hernia

Pregnancy

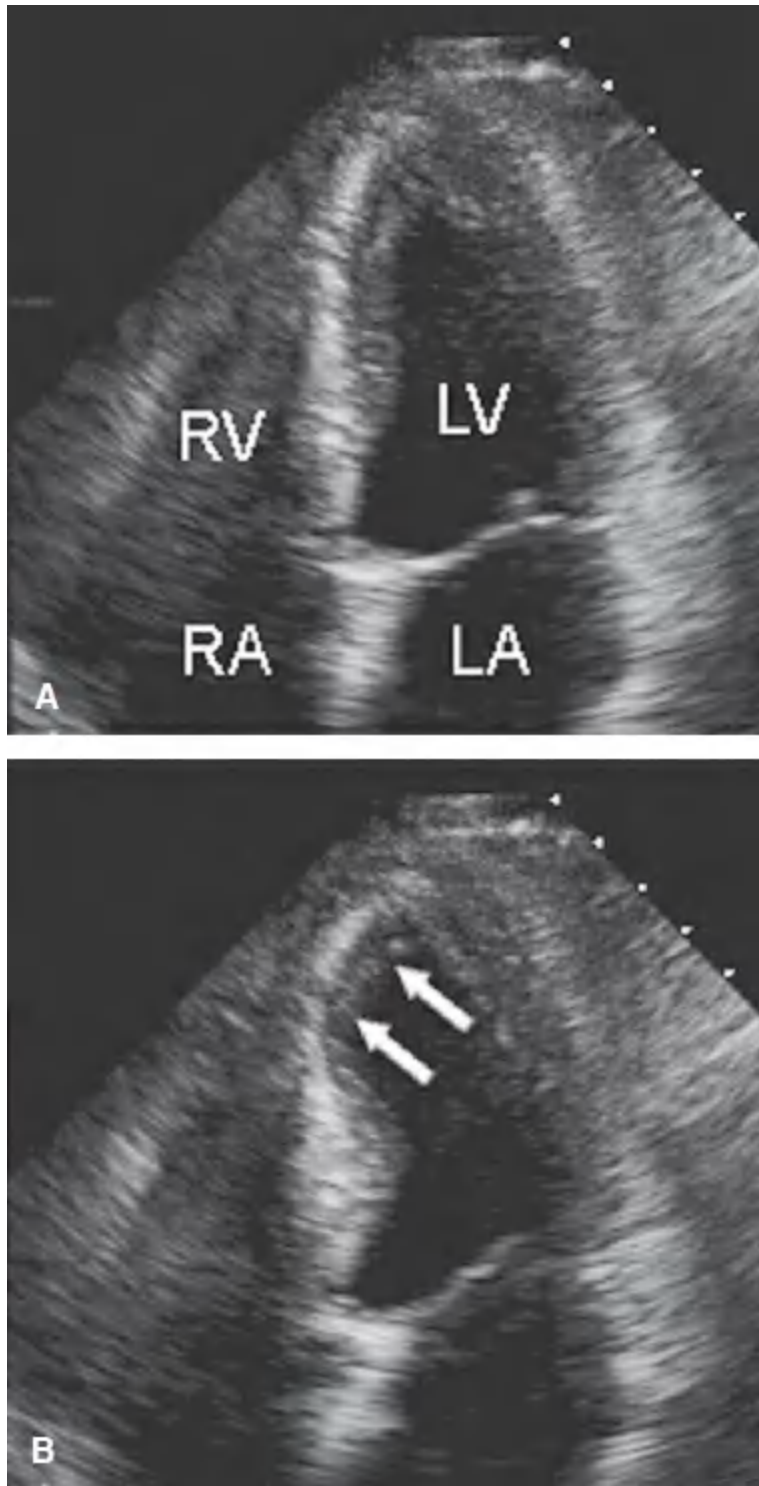


**FIGURE 5.45.** Apical four-chamber view recorded in a young, healthy individual immediately postexercise, demonstrating early relaxation of the apical septum. The upper panel (**A**) was recorded at end-systole and shows normal hyperdynamic motion of all visualized segments. The lower panel (**B**) 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 5.7** ISCHEMIC VERSUS NONISCHEMIC WALL MOTION ABNORMALITIES

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

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.

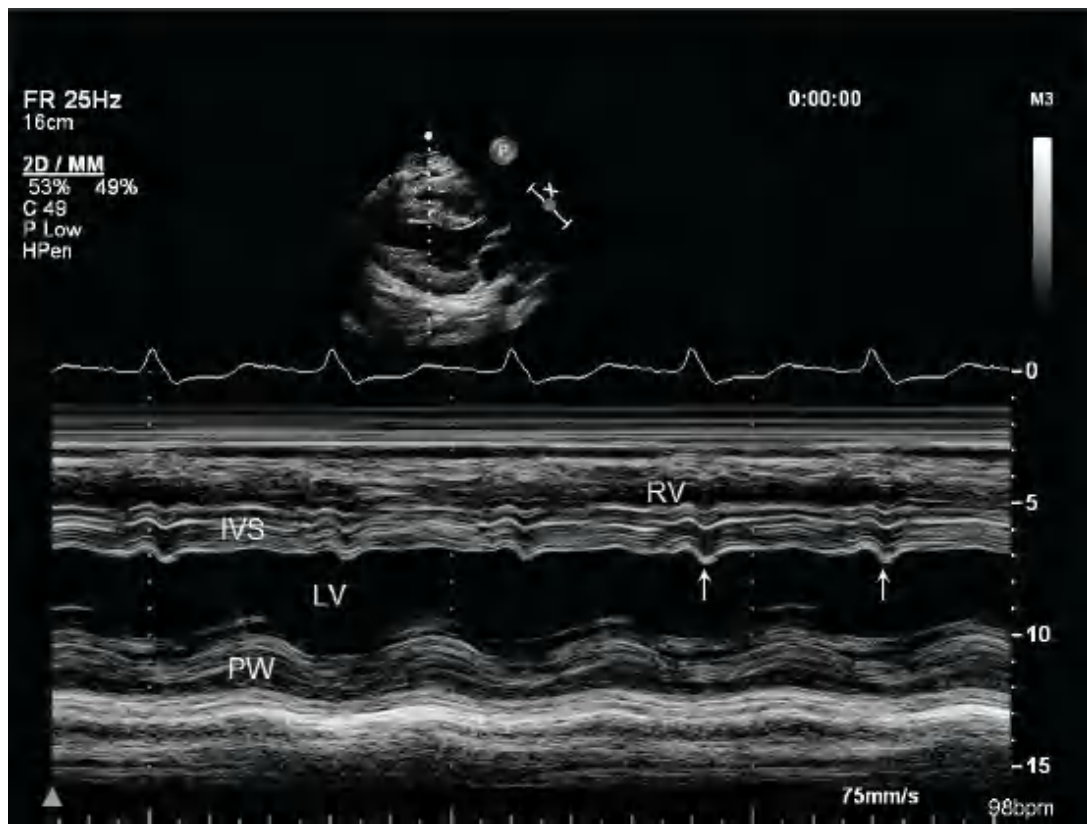
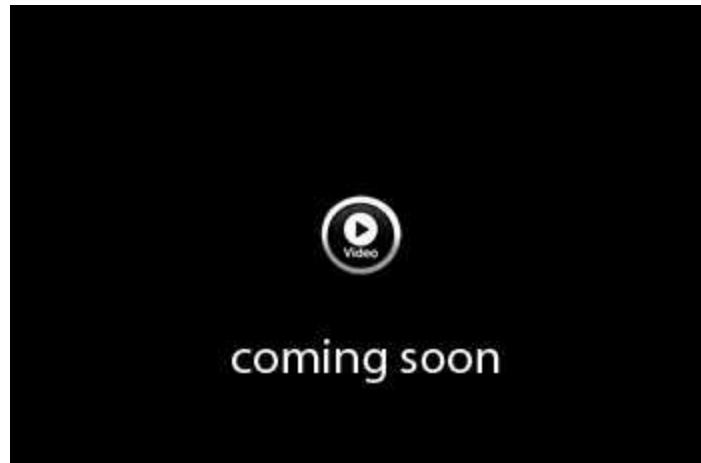


**FIGURE 5.46.** M-mode echocardiogram recorded in a patient with a left bundle branch block. In the M-mode note the abrupt downward motion of the ventricular septum shortly after the onset of the QRS (*arrows*) and the relatively neutral position of the ventricular septum throughout the remainder of 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.





**Video 5-46**

## **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).

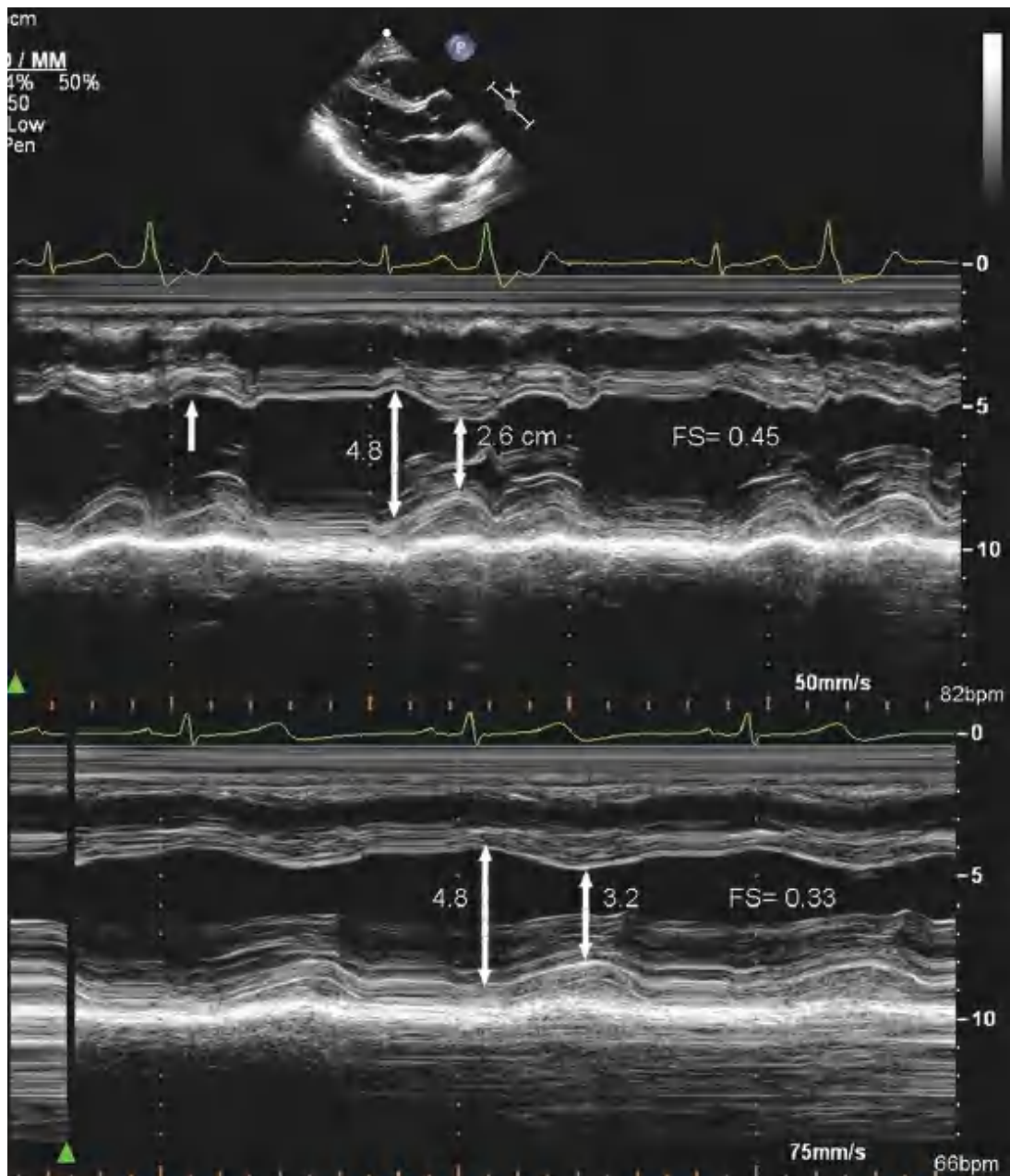
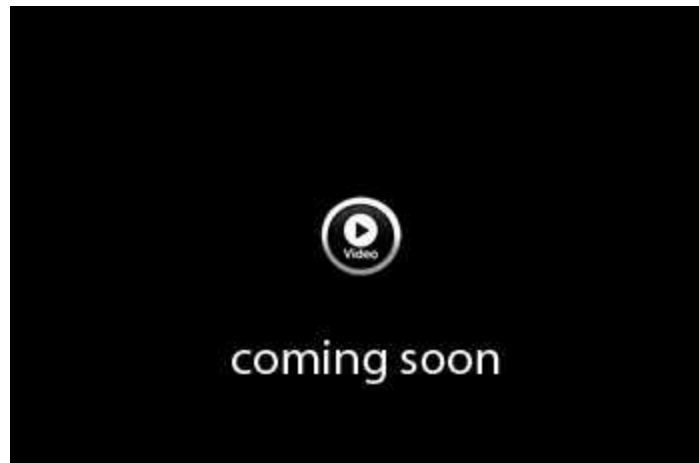




**FIGURE 5.47.** M-mode echocardiogram recorded in a patient with ventricular bigeminy. The upper panel was recorded during bigeminy and reveals an abnormal contraction pattern of the ventricular septum (*arrow*) coincident with the PVC. The internal dimension (*double-headed arrows*) in diastole and 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.

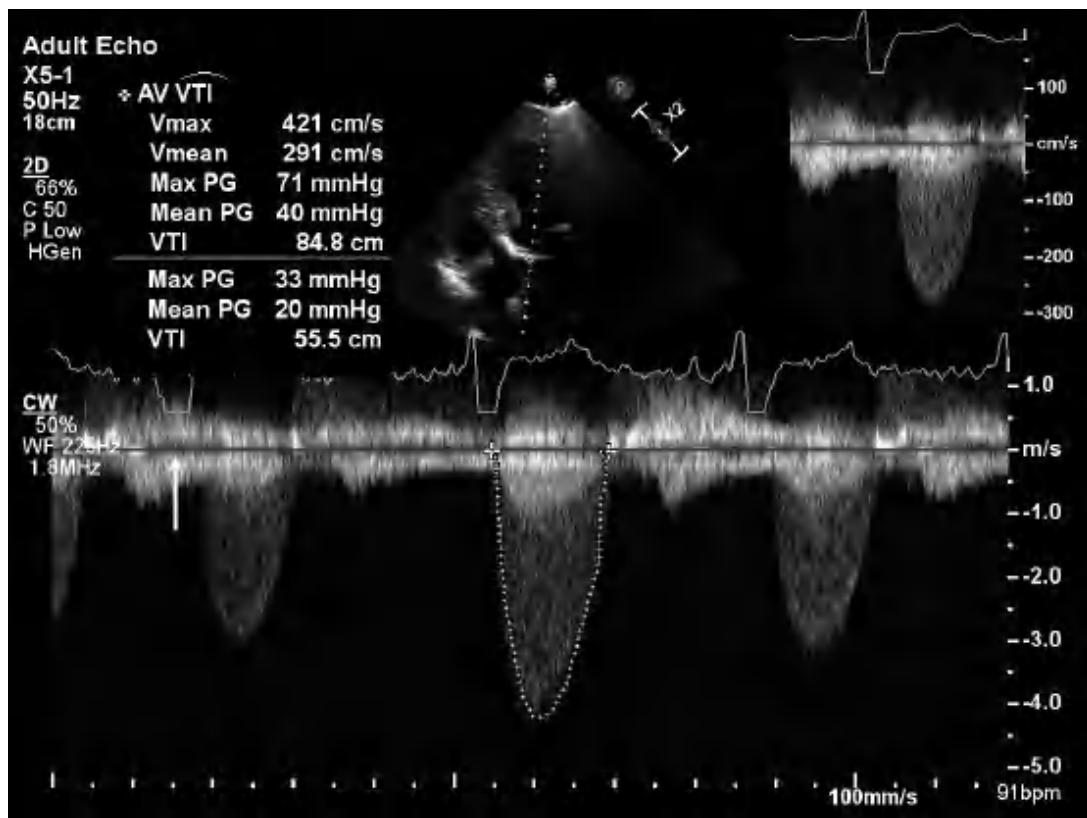


**FIGURE 5.48.** Continuous-wave Doppler in a patient with aortic stenosis and reduced left ventricular systolic function, demonstrating the impact of a PVC on left ventricular contractility. The central continuous-wave Doppler is a continuous recording. Note the PVC (*upward-pointing arrow*), after 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.

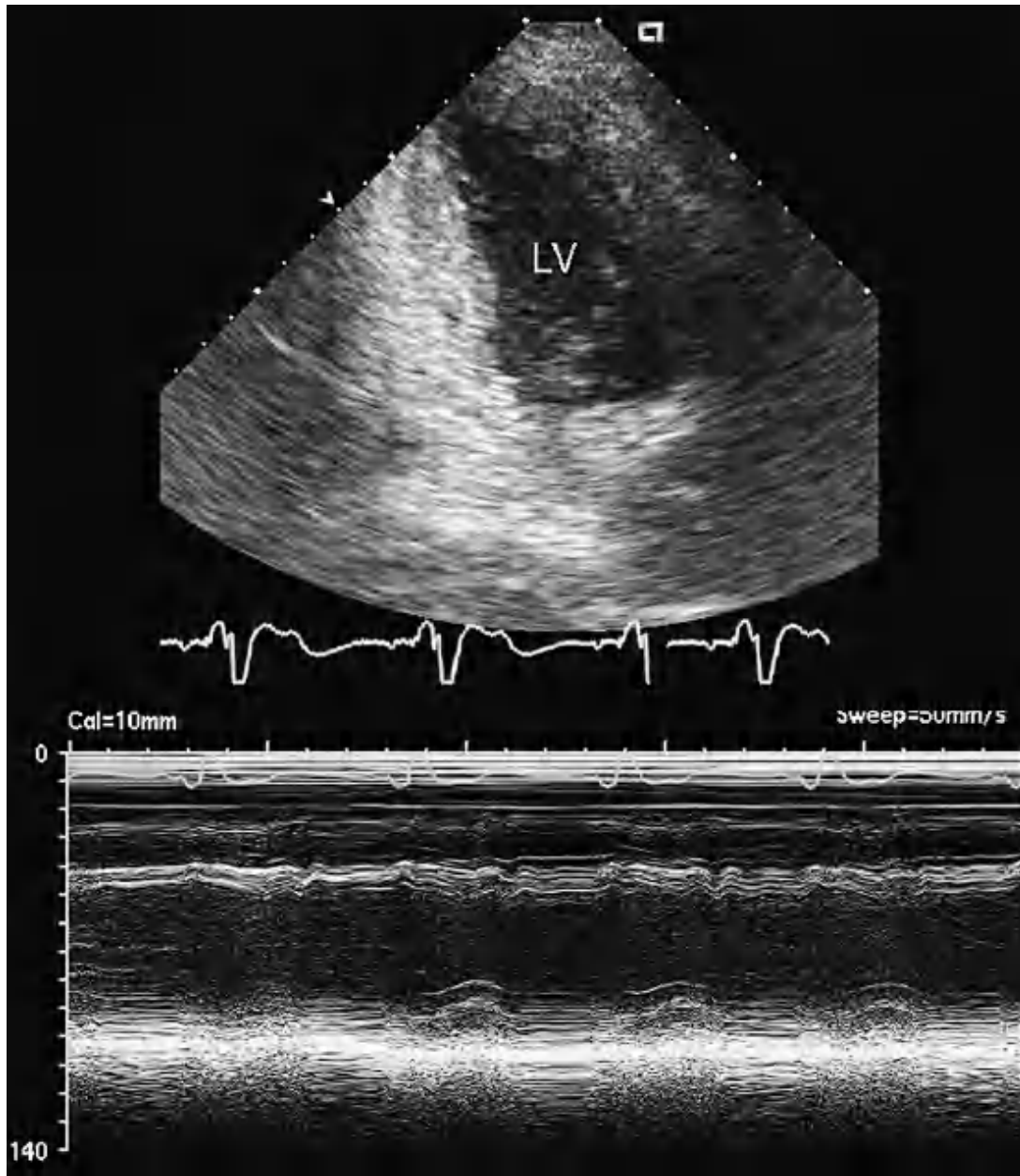
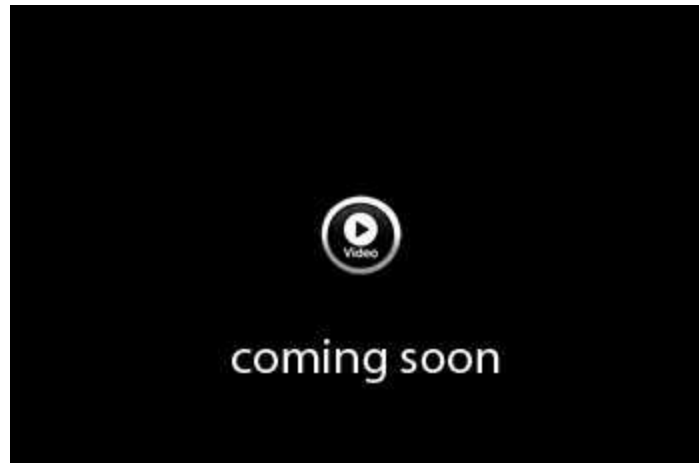


**FIGURE 5.49.** Apical two-chamber view recorded in a patient with a right ventricular transvenous pacemaker. In the M-mode echocardiogram, notice the atypical pattern of septal motion consistent with a bundle branch block. In the apical two-chamber view, note the marked inferoapical wall motion abnormality in this patient, known to be free of coronary artery disease, related to pacing at the inferoapical aspect of the right ventricle. 



**Video 5-49**

## **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](#).

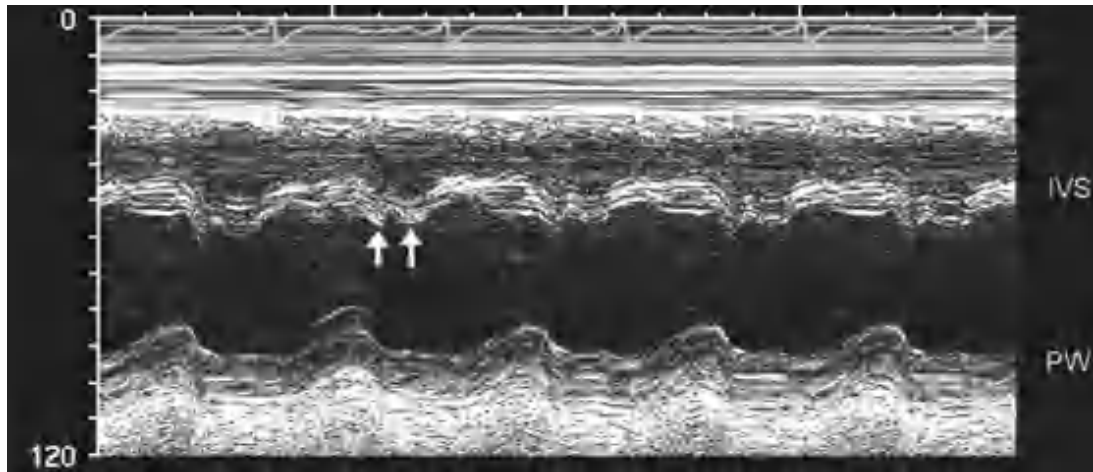


**FIGURE 5.50.** M-mode echocardiogram recorded in a patient with constrictive pericarditis. Note the relatively flat motion of the posterior wall endocardium and the abnormal multiphasic diastolic motion of the ventricular septum (*arrows*) related to an increase in ventricular interdependence. PW, posterior wall.

## 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.



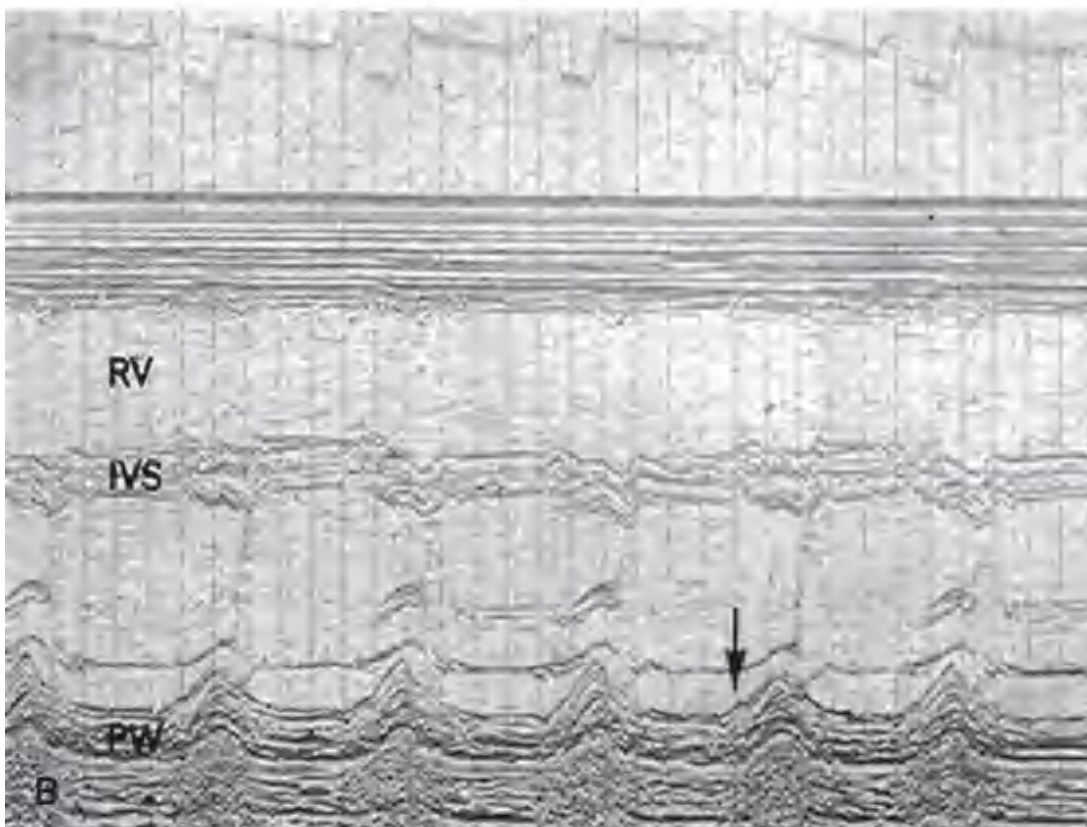
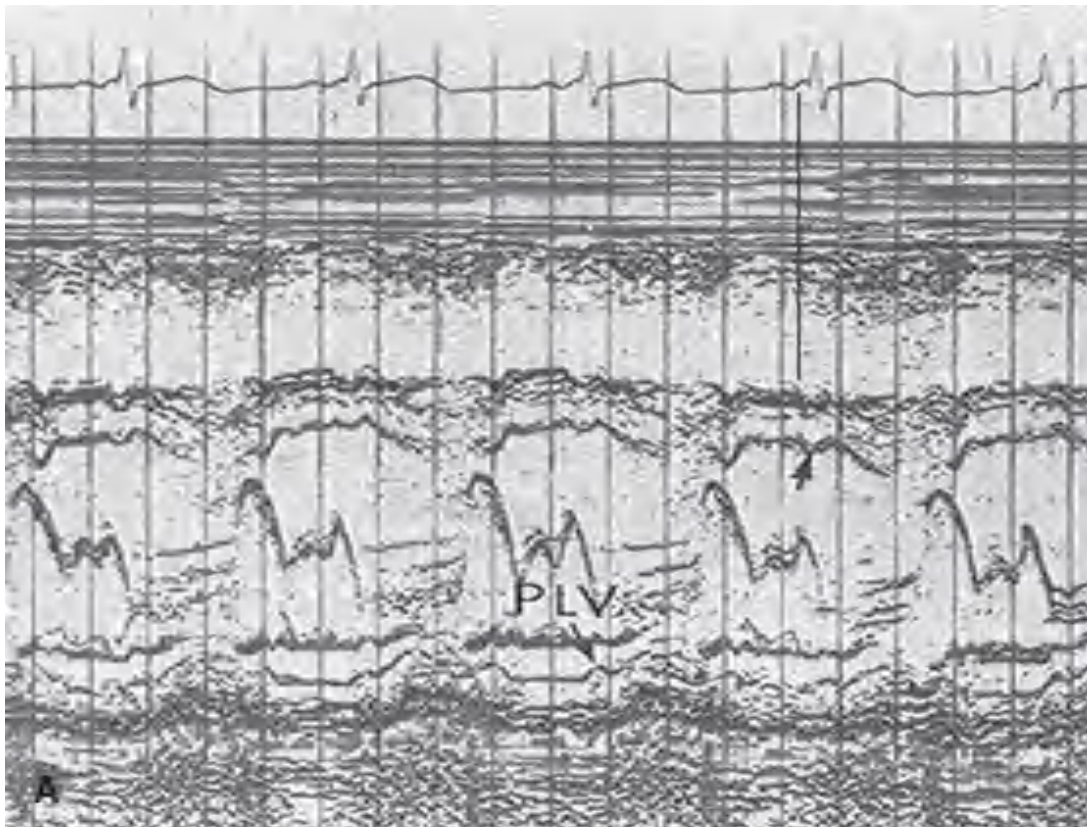


**FIGURE 5.51.** M-mode echocardiograms recorded in two patients with ventricular

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.



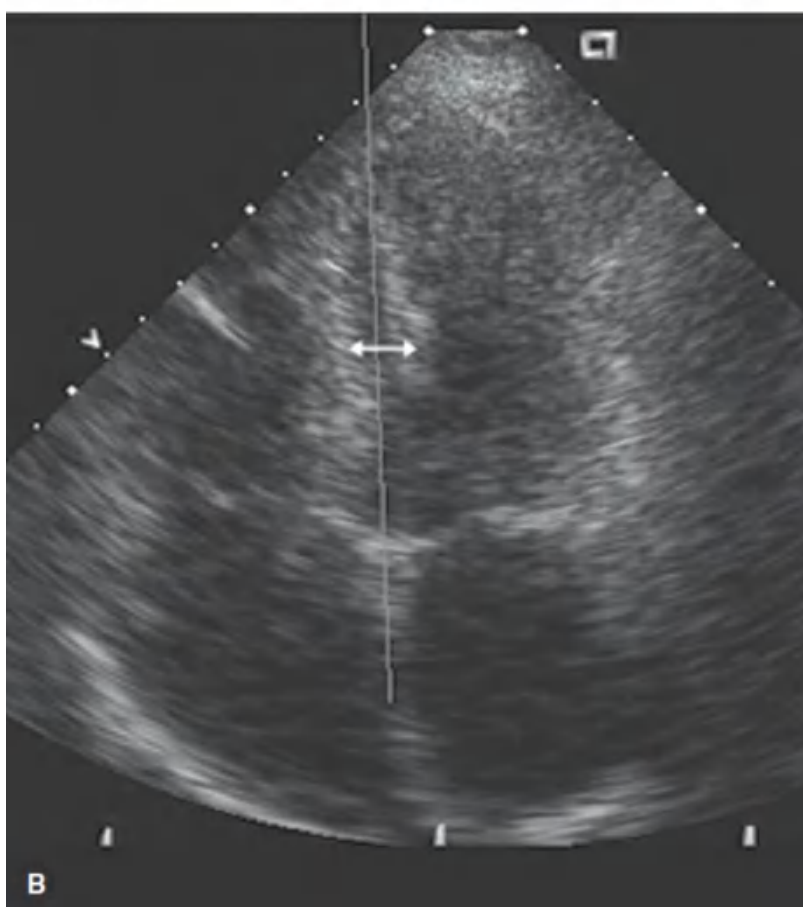
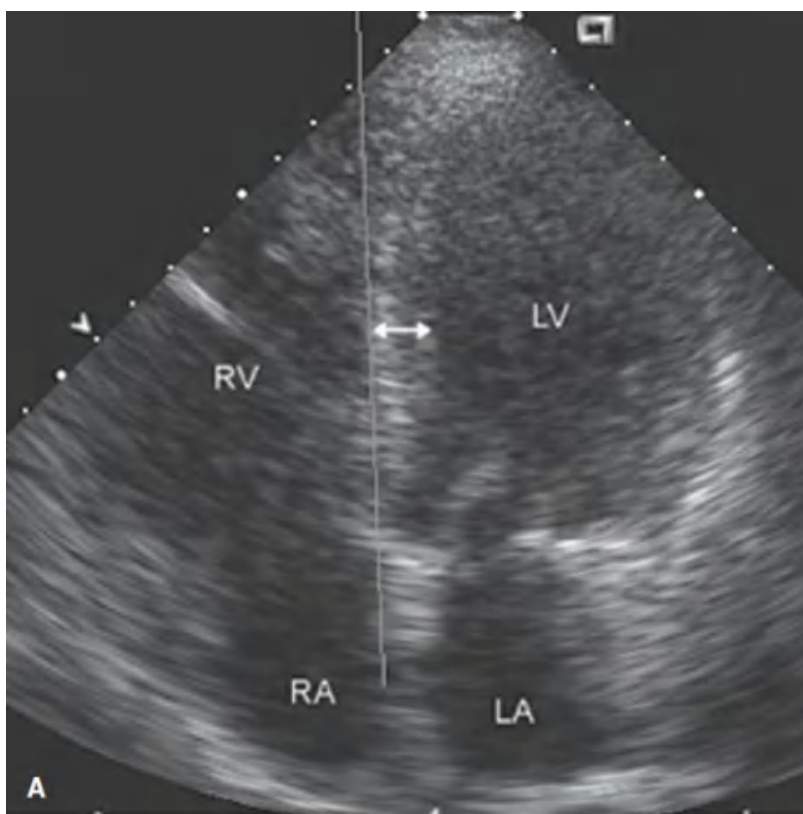
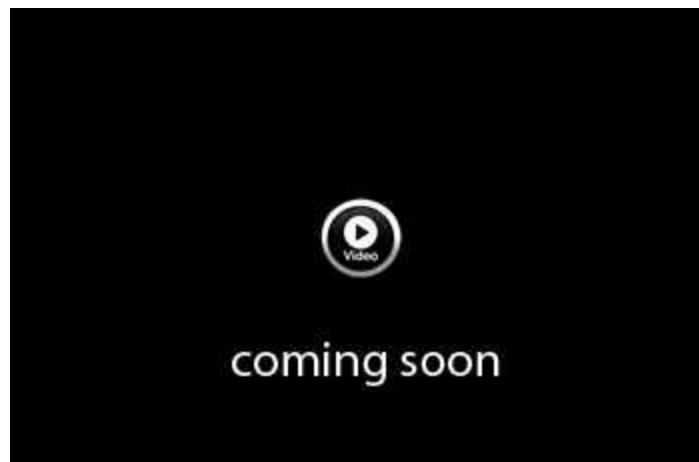


**FIGURE 5.52.** Apical four-chamber view recorded in a patient after cardiac surgery demonstrates postoperative motion of the entire heart. **A:** Image was recorded in end-diastole. The *vertical line* marks the position of the right side of the ventricular septum. **B:** Image was recorded in end-systole. 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*). 



**Video 5-52**

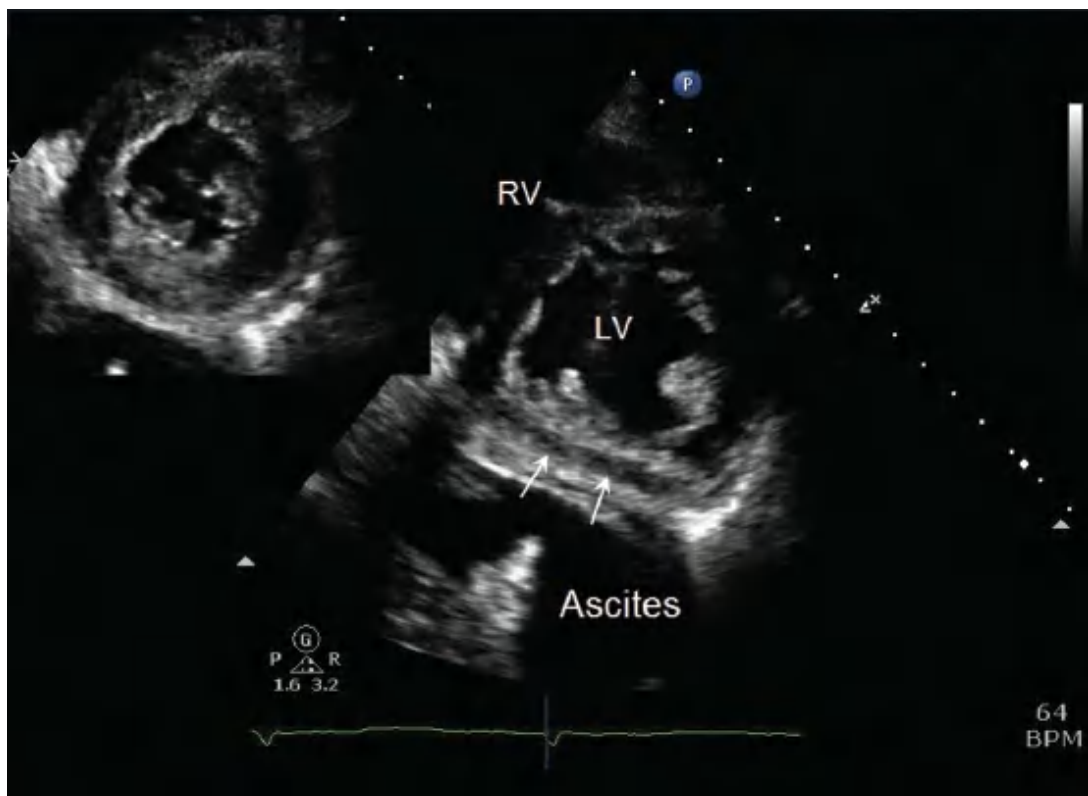
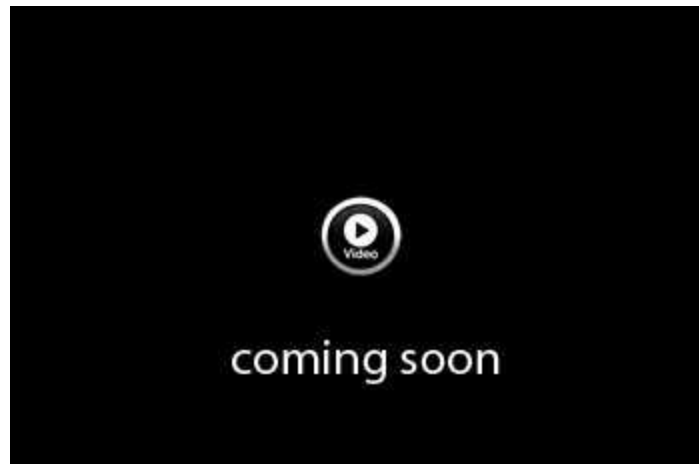


**FIGURE 5.53.** Example of posterior wall pseudodyskinesis related to ascites. In the central image note that the ascites has compressed the posterior wall of the left ventricle (*arrows*) and that in diastole it has a “D-shaped” configuration. In early systole (upper left inset, note the ventricle 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. 📺



**Video 5-53**

## 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.

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### **STRAIN AND STRAIN RATE IMAGING**

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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.



## 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.

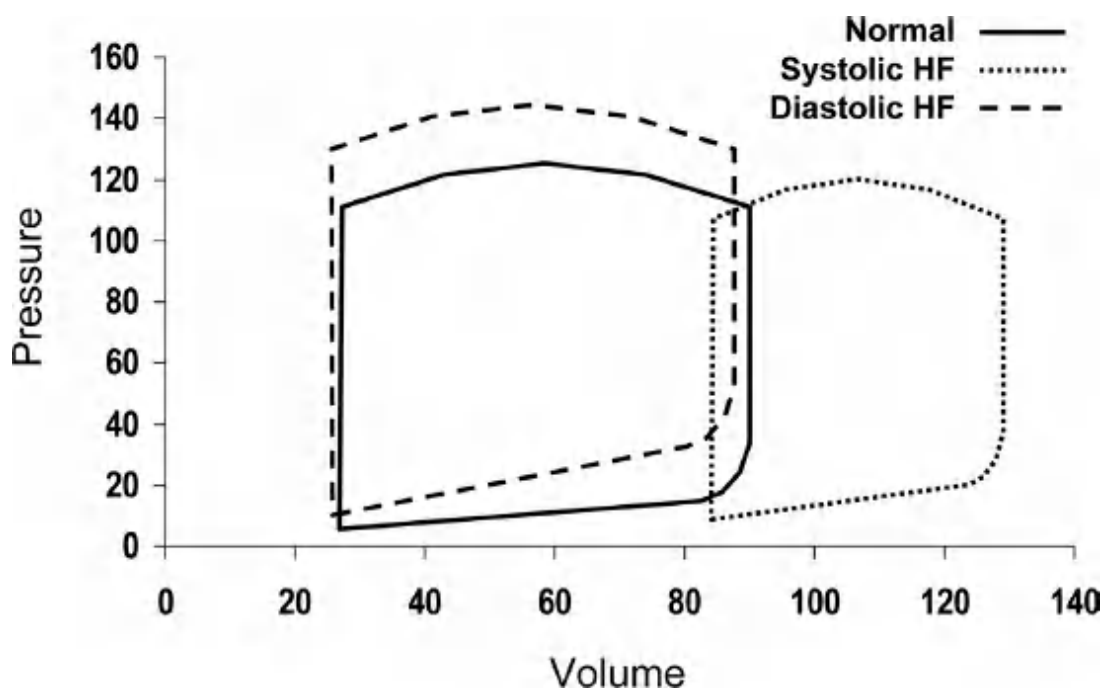


**FIGURE 6.1.** Diastolic filling and systolic ejection can be demonstrated using pressure–volume loops. By tracing a loop counterclockwise, the entire cardiac cycle depicting the interplay between pressure and volume, is illustrated. In this example, the changes that occur with systolic versus diastolic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

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.

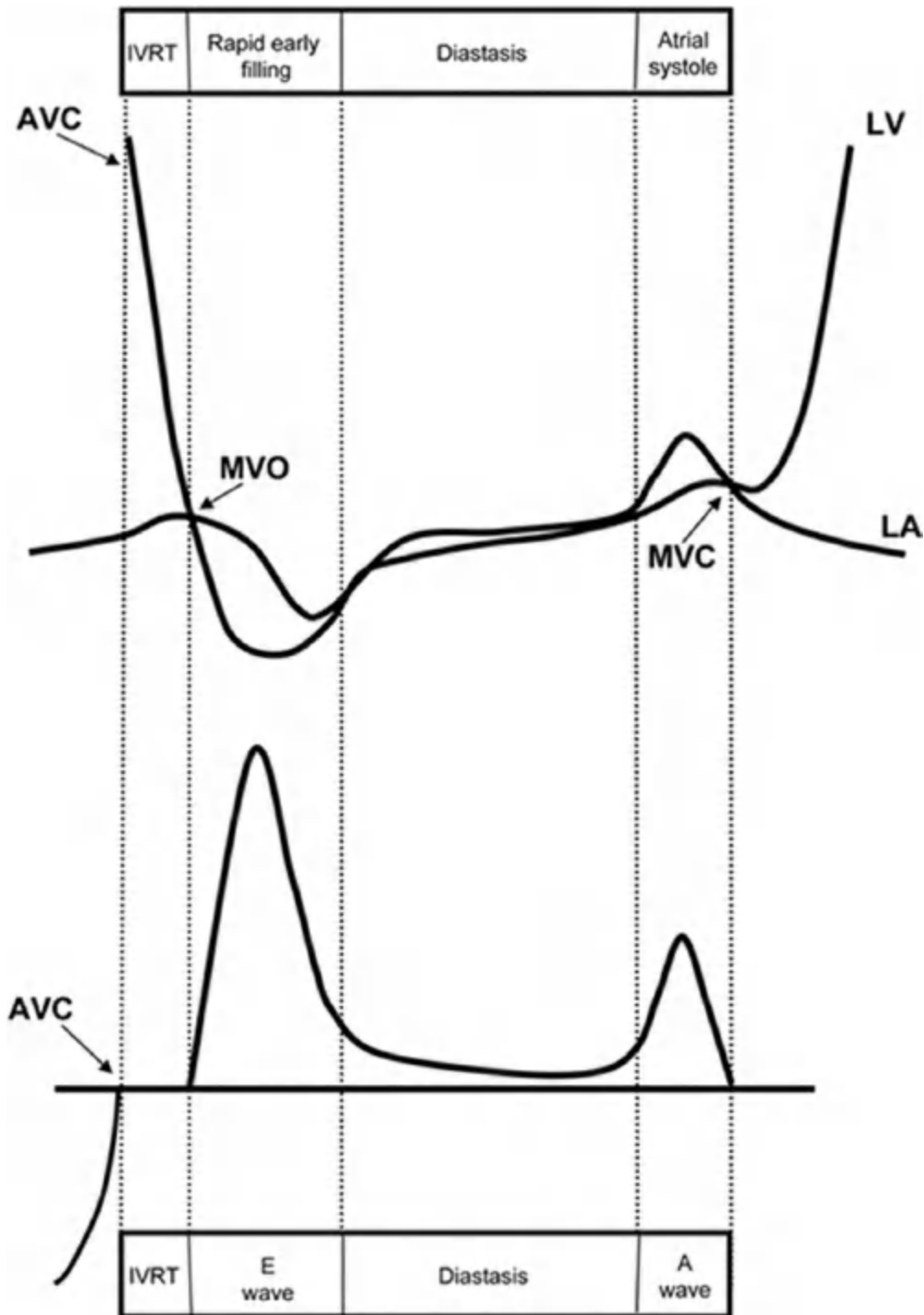


**FIGURE 6.2.** The four stages of diastole are illustrated in this schematic. The upper tracing illustrates the LV and LA pressure curves, while the lower tracing demonstrates the associated transmitral filling pattern, recorded with Doppler. Isovolumic relaxation begins with aortic valve closure (AVC) 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.

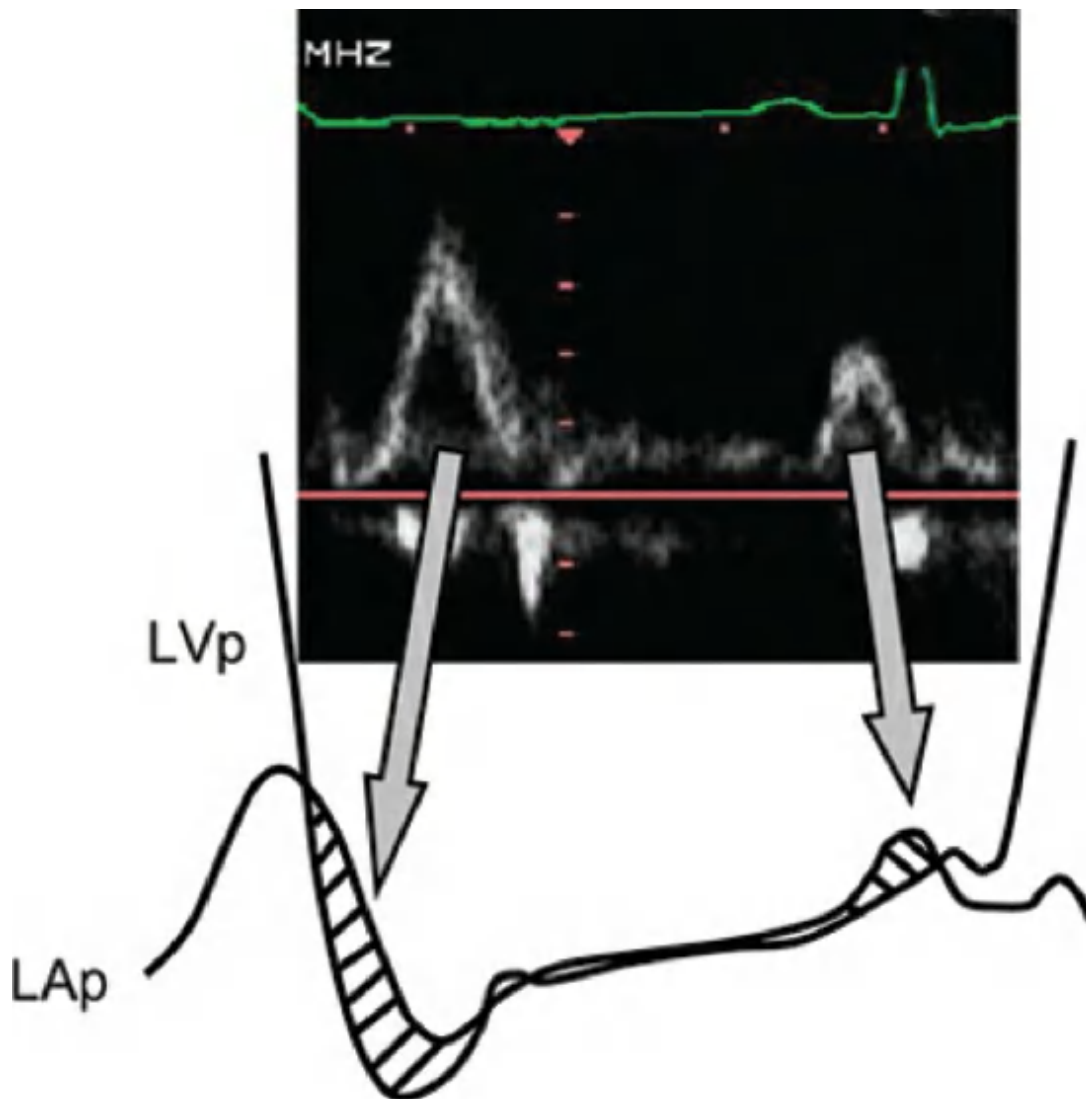


**FIGURE 6.3.** The instantaneous pressure gradient across the mitral valve, between the left atrium and left ventricle creates flow which can be recorded using the Doppler technique. In early diastole, the rapid fall in left ventricular pressure (LVp) produces the E wave, while in late diastole, left 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  $V_p$ . 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 6.1**

**GRADES OF DIASTOLIC DYSFUNCTION**

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

|  |  |                                                                                                                                                                                                     |                                                                                           |
|--|--|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------|
|  |  | Diminished LV suction forces<br>High opening LA-LV pressure gradient<br>Elevated LA pressure (inflow by “pushing” blood)<br>Failing LA contractility<br>May or may not respond to preload reduction | High LV filling pressure, may or may not respond to diuresis (reversible or irreversible) |
|--|--|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------|

HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction.

## 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.

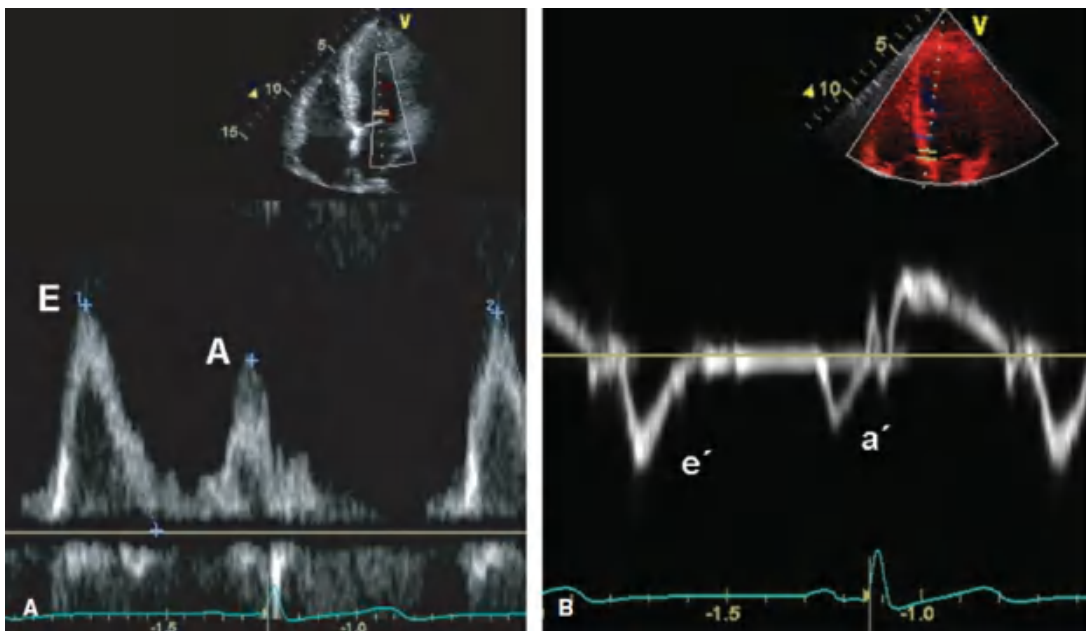


**FIGURE 6.4.** In **A**, a normal mitral inflow velocity pattern is illustrated, demonstrating the E wave velocity greater than the A wave velocity. In **B**, the corresponding tissue Doppler recording of mitral annular velocity shows the e' velocity greater than a' velocity.

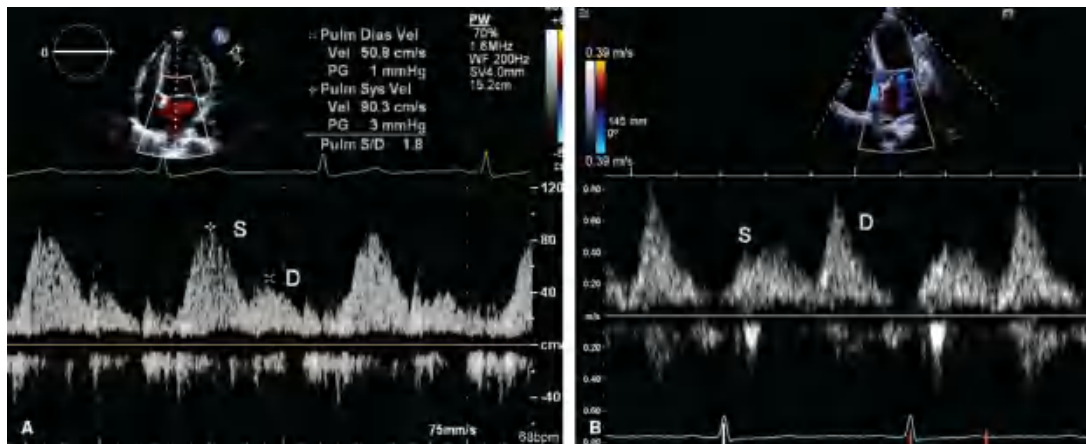


**FIGURE 6.5.** Two examples of pulmonary vein flow patterns are shown. In **A**, from a patient with well-controlled hypertension, a dominant systolic (S) wave is present, with a smaller diastolic component (D). In **B**, from a patient with advanced diastolic dysfunction, the S wave is much smaller than D, suggesting a decrease in left atrial compliance and increased left atrial pressure.

## 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 ( $Ar - A > 30$  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.

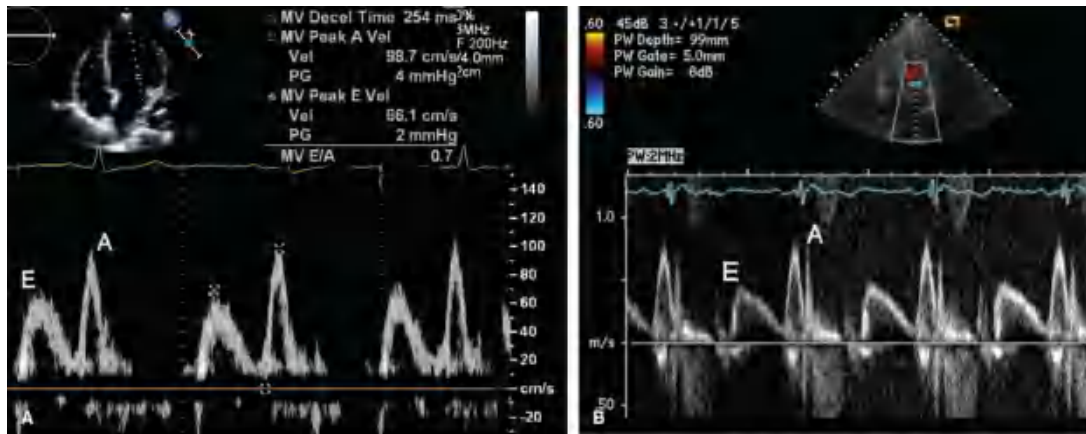


**FIGURE 6.6.** Two examples of impaired relaxation are shown. In **A**, the mitral inflow pattern shows E/A reversal, with a ratio of 0.7. In **B**, the patient demonstrates low E wave velocity, prolonged E wave deceleration, and a large A wave. See text for details.

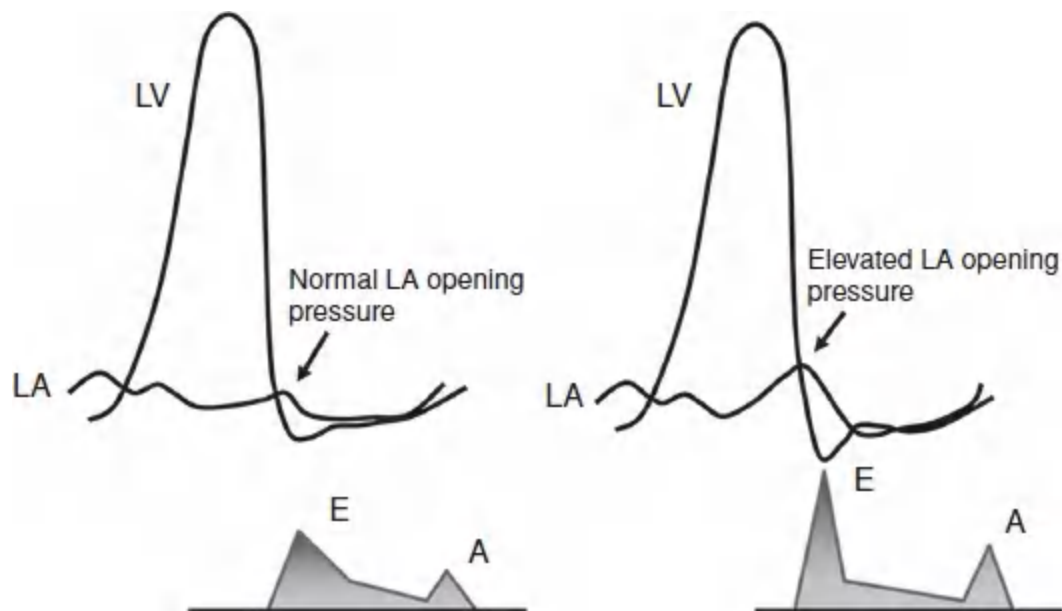


**FIGURE 6.7.** The effect of an increase in mean left atrial pressure on Doppler inflow velocity is demonstrated. On the left, in the setting of normal left atrial pressure, a typical mitral inflow velocity pattern is shown. On the right, when left atrial pressure is elevated, isovolumic relaxation 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 become 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.

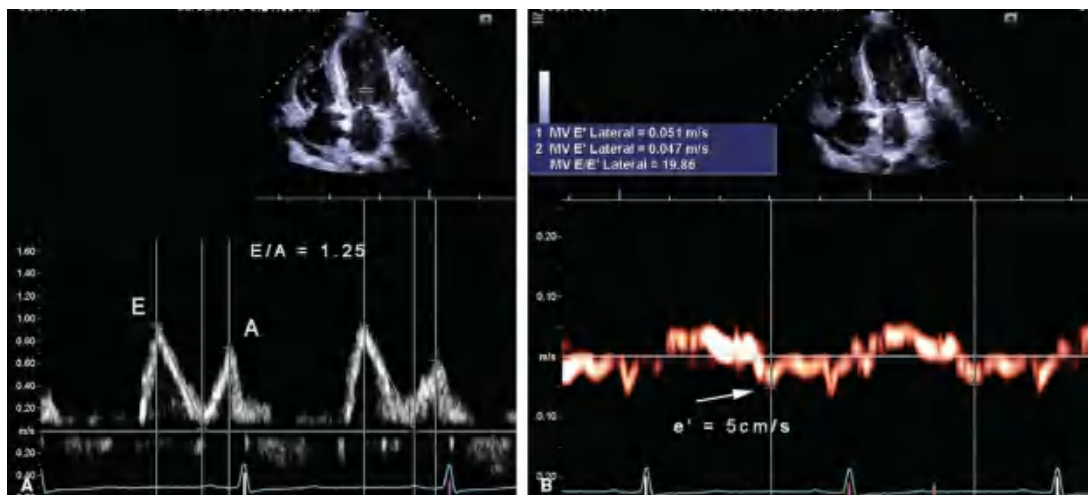


**FIGURE 6.8.** An example of pseudonormal diastolic dysfunction is provided. In **A**, a “normal” E/A ratio is noted (1.25). In **B**, however, the annular velocity ( $e'$ ) is markedly reduced at 5 cm/s, indicating delayed relaxation.

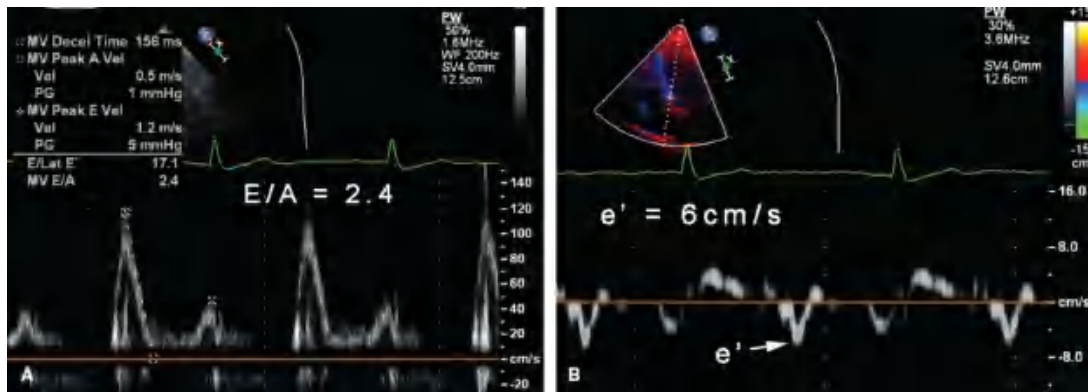


**FIGURE 6.9.** Restrictive physiology is demonstrated in this elderly patient with ischemic heart disease. In **A**, mitral inflow shows a high E wave velocity, short deceleration time, and an abnormal E/A ratio of 2.4. In **B**, a very low e' velocity is noted.

**Table 6.2**

## ECHO DOPPLER MODALITIES FOR EVALUATING DIASTOLIC FUNCTION

| Parameter                 | Modality       | Significance                                                                                                               |
|---------------------------|----------------|----------------------------------------------------------------------------------------------------------------------------|
| IVRT                      | Pulsed Doppler | Information on LA pressure, rate of early active LV relaxation                                                             |
| Mitral inflow:            | Pulsed Doppler |                                                                                                                            |
| E/A ratio                 |                | Reflects LA-LV gradient in early and late diastole; helps define stages                                                    |
| Deceleration time         |                | Information on LV chamber compliance                                                                                       |
| Response to Valsalva      |                | Helps differentiate normal from pseudonormal stages                                                                        |
| A wave duration           |                | Combined with PVa wave, reflects LV filling pressure                                                                       |
| Flow propagation velocity | Color M-mode   | Reflects elastic recoil, rate of early diastolic LV relaxation; can be used to estimate pulmonary capillary wedge pressure |
| Annular velocity:         | Tissue Doppler |                                                                                                                            |
| e'                        |                | Correlates with early diastolic relaxation; distinguishes RCM from constrictive pericarditis                               |
| E/e' ratio                |                | Predicts LV filling pressure                                                                                               |
| Pulmonary                 | Pulsed         |                                                                                                                            |

|           |         |                                                                                   |
|-----------|---------|-----------------------------------------------------------------------------------|
| flow:     | Doppler |                                                                                   |
| S/D ratio |         | Changes correlate with stages of diastolic dysfunction                            |
| A – Ar    |         | Difference in duration of the two waves reflects LV filling pressure              |
| LA volume | 2D echo | Information on presence and chronicity of diastolic dysfunction; prognostic value |

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.

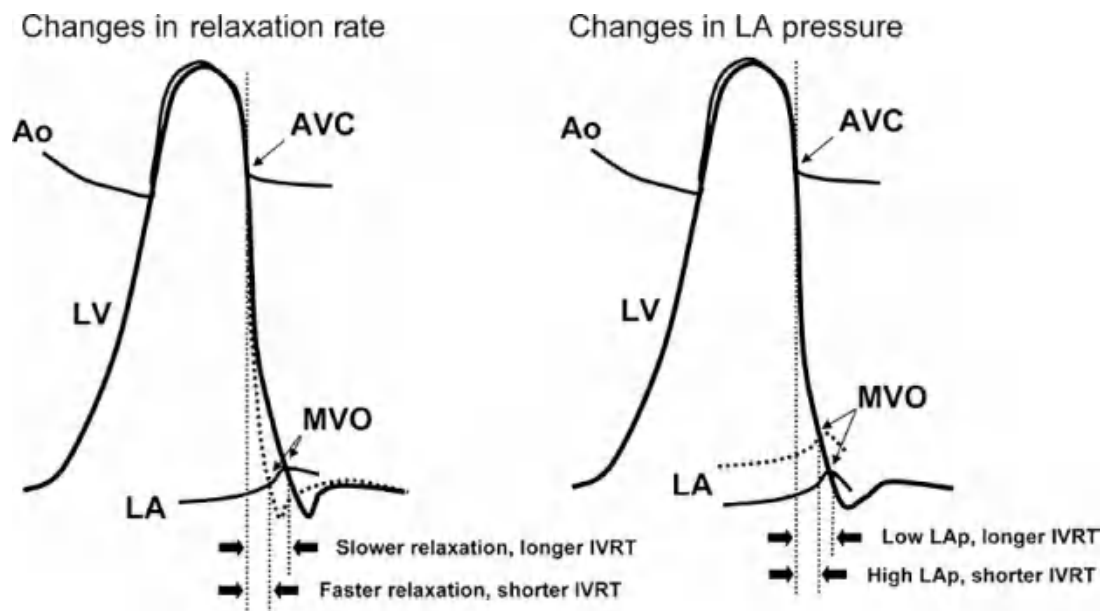


**FIGURE 6.10.** This schematic demonstrates how left ventricular relaxation rate and changes in left atrial pressure affect isovolumic relaxation time (IVRT). See text for details. AVC, aortic valve closure; MVO, mitral valve opening.

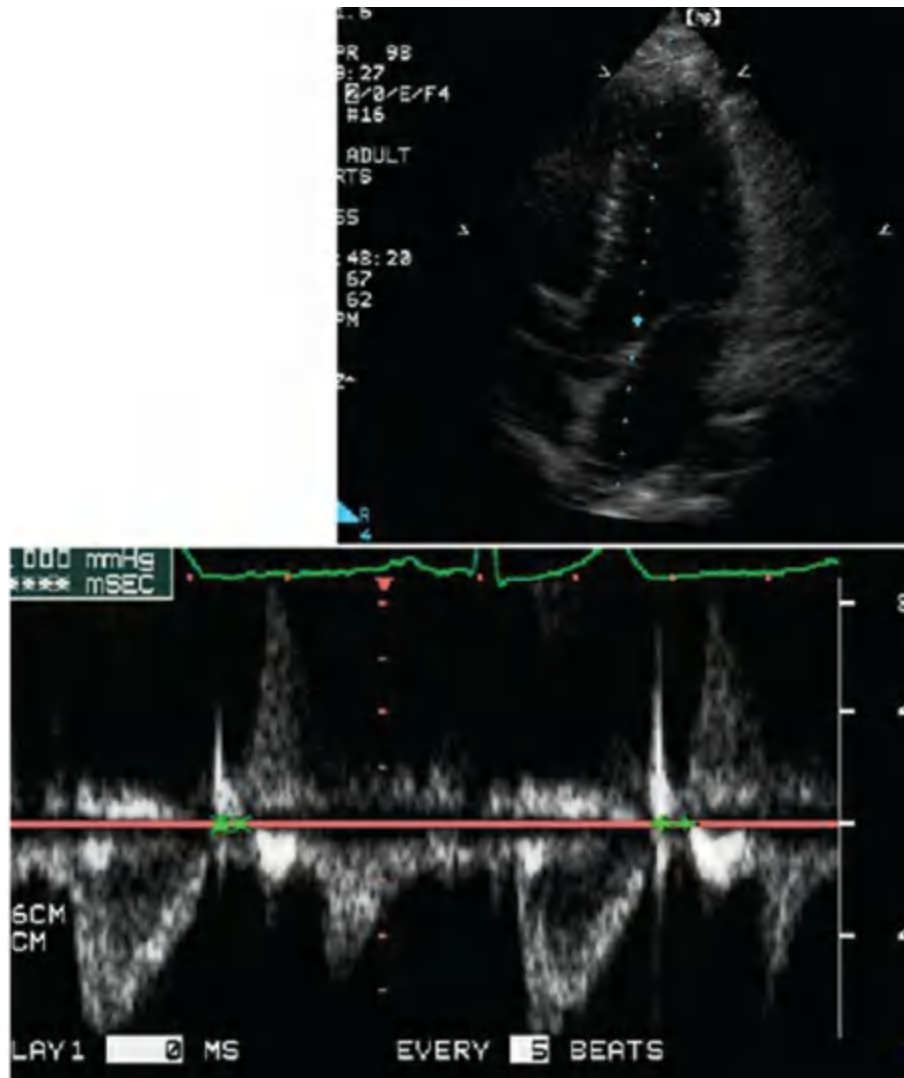


**FIGURE 6.11.** The method used to record the isovolumic relaxation time (IVRT) is demonstrated. Using a modified apical four-chamber view, the Doppler sample volume (SV) is placed between the inflow and outflow regions so that mitral and aortic flows are recorded simultaneously. Using a rapid sweep speed, the interval between aortic valve closure and mitral valve opening can be determined. See text for details.

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.



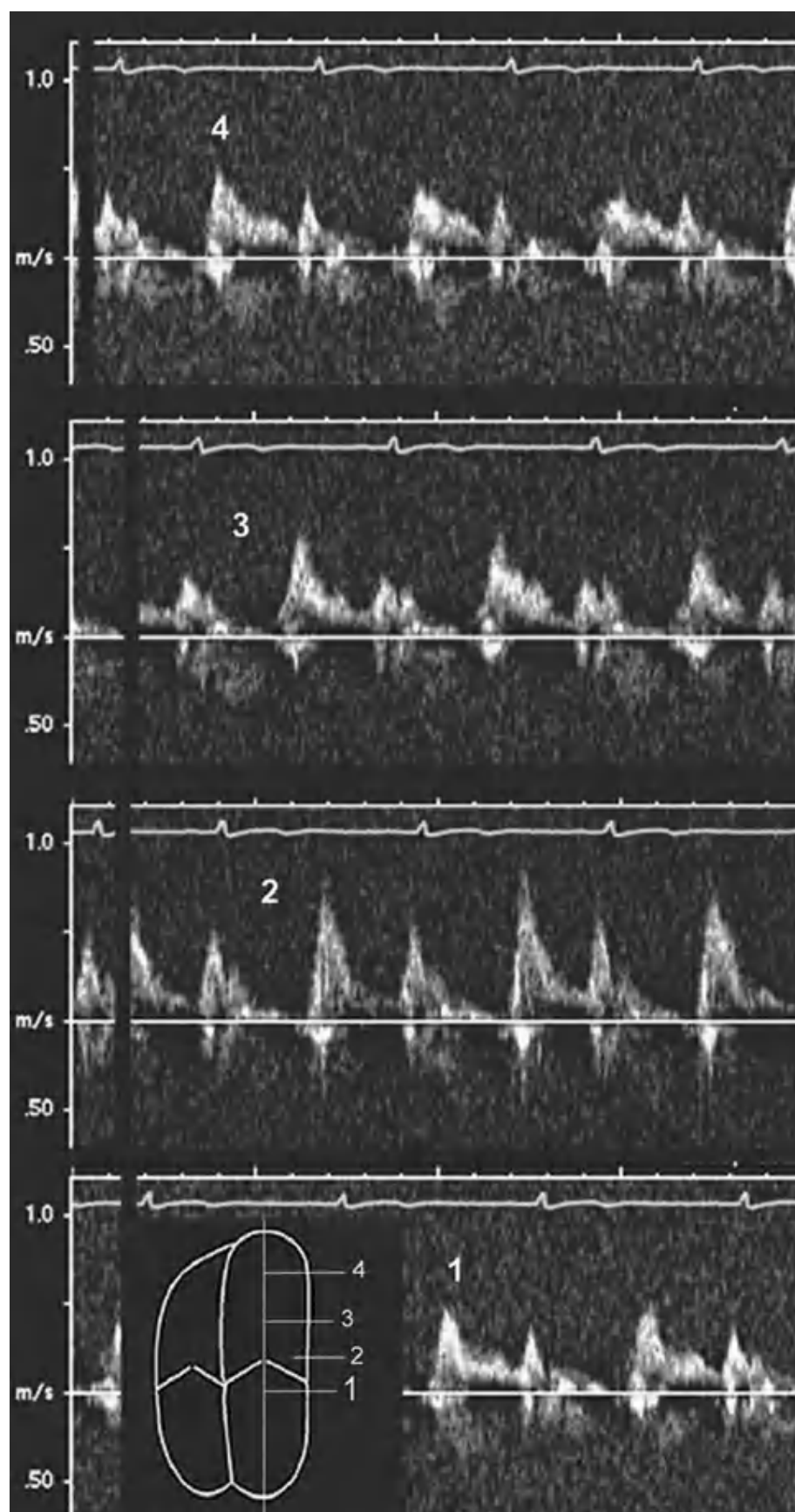




**FIGURE 6.12.** The effect of sample volume location on the mitral inflow velocity pattern is demonstrated. The schematic at the lower left shows four sample volume locations. Each location results in a different pattern of mitral inflow. The proper location, used to record peak velocity in early and late diastole, usually requires placement of the sample volume at the tips of the mitral leaflets.

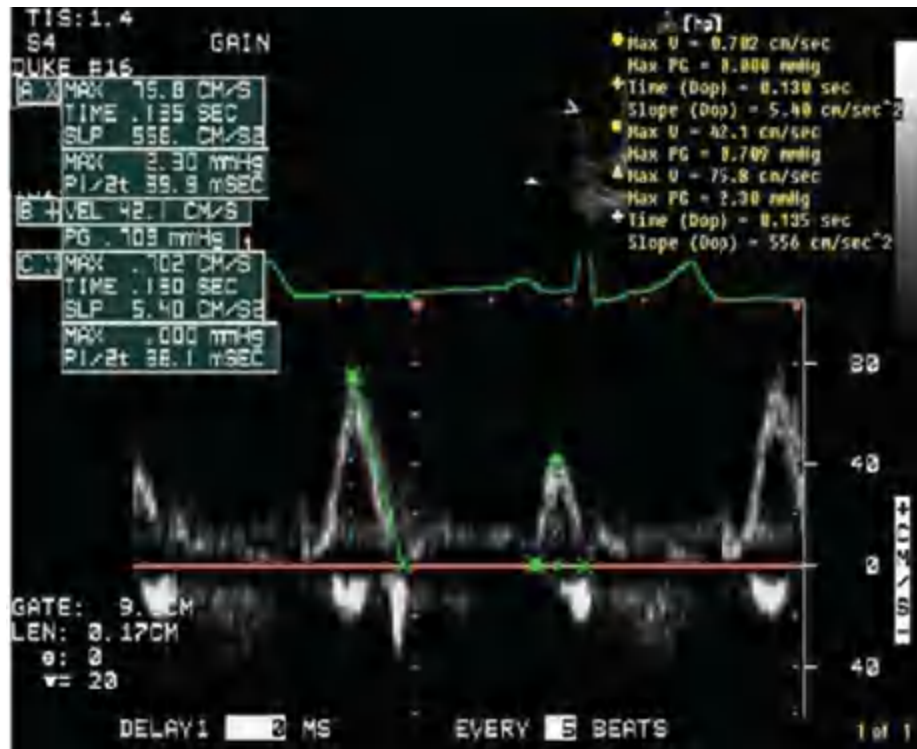


**FIGURE 6.13.** Once an optimal mitral inflow velocity recording has been obtained, a variety of measurements can be made. These are illustrated in the figure. See text for details.

## Color M-Mode Flow Propagation Velocity ( $V_p$ )

When the mitral valve opens, flow accelerates from the valve orifice toward the apex of the left ventricle. Propagation velocity ( $V_p$ ) 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.

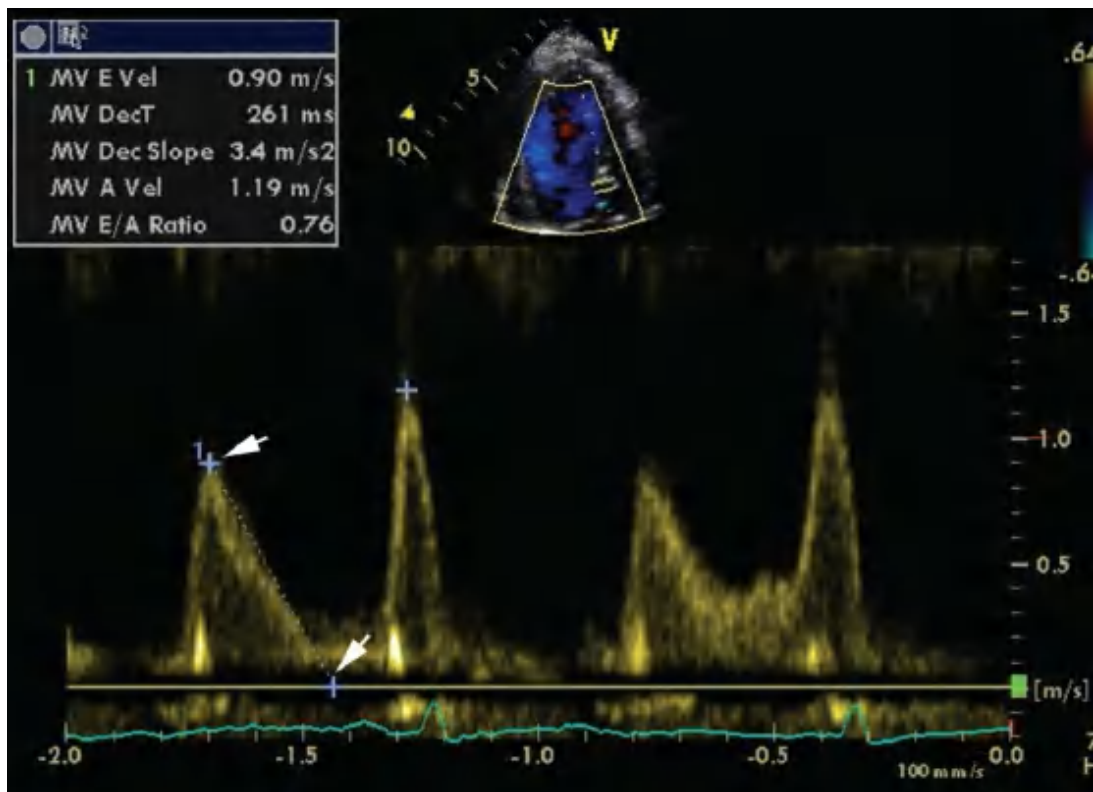


**FIGURE 6.14.** The method used to measure deceleration (decel) time is shown. It is defined as the time required for the peak E wave velocity to the cessation of early rapid filling as indicated by the arrows. See text for details.

## 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 septal and lateral  $E/e'$  are available.

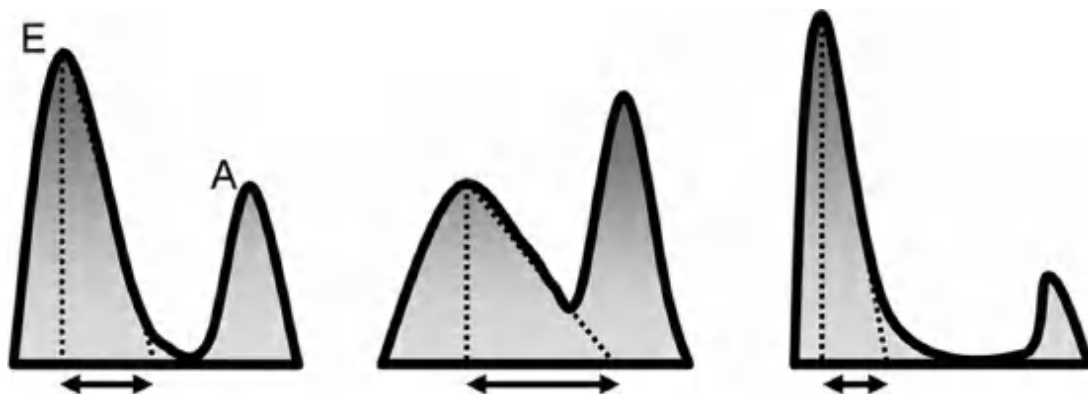


**FIGURE 6.15.** The schematic demonstrates three types of mitral inflow velocity curves and shows how deceleration time should be determined in each case. Note in the middle panel that the velocity curve does not reach the baseline and the deceleration line must be extrapolated in order to determine the deceleration time.

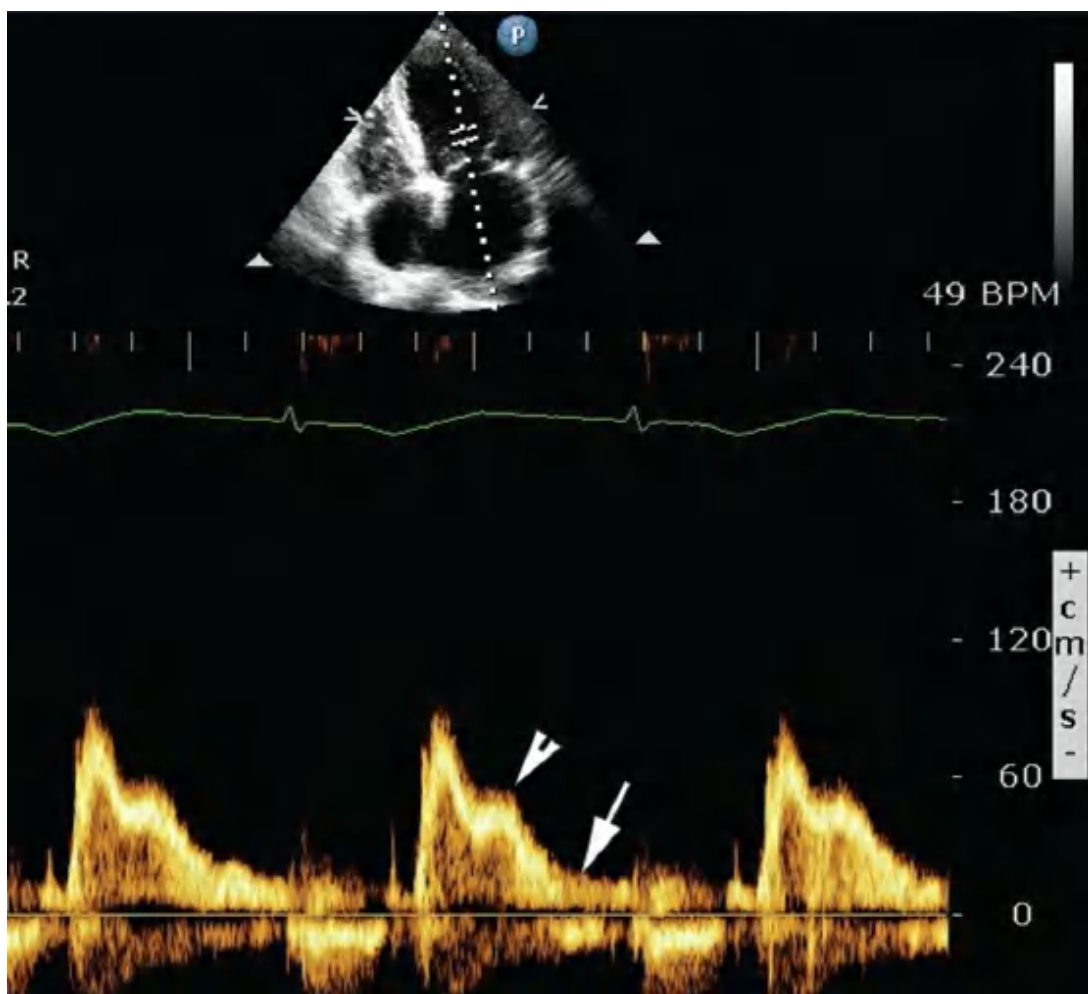


**FIGURE 6.16.** An example of very abnormal mitral inflow are shown. The E wave is followed immediately by a wave of mid-diastolic filling (sometimes called an L wave, see *arrowhead*). Little or no filling occurs in late diastole (*larger arrow*).

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'.

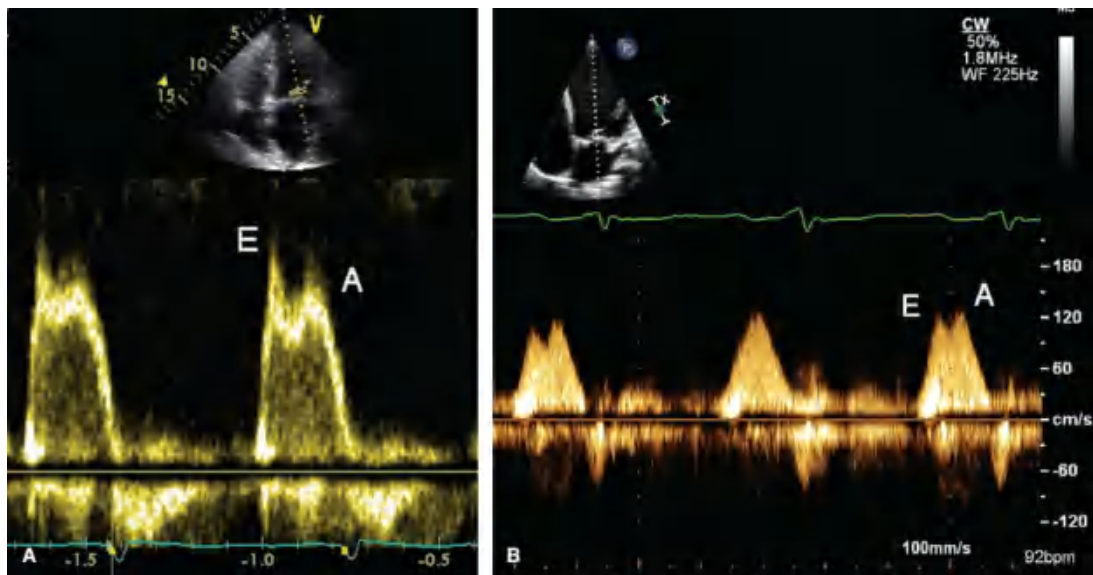


**FIGURE 6.17.** Two examples of E-A fusion are shown. In both, a combination of heart rate, very delayed left ventricular relaxation, and high left atrial pressure contribute to this pattern.



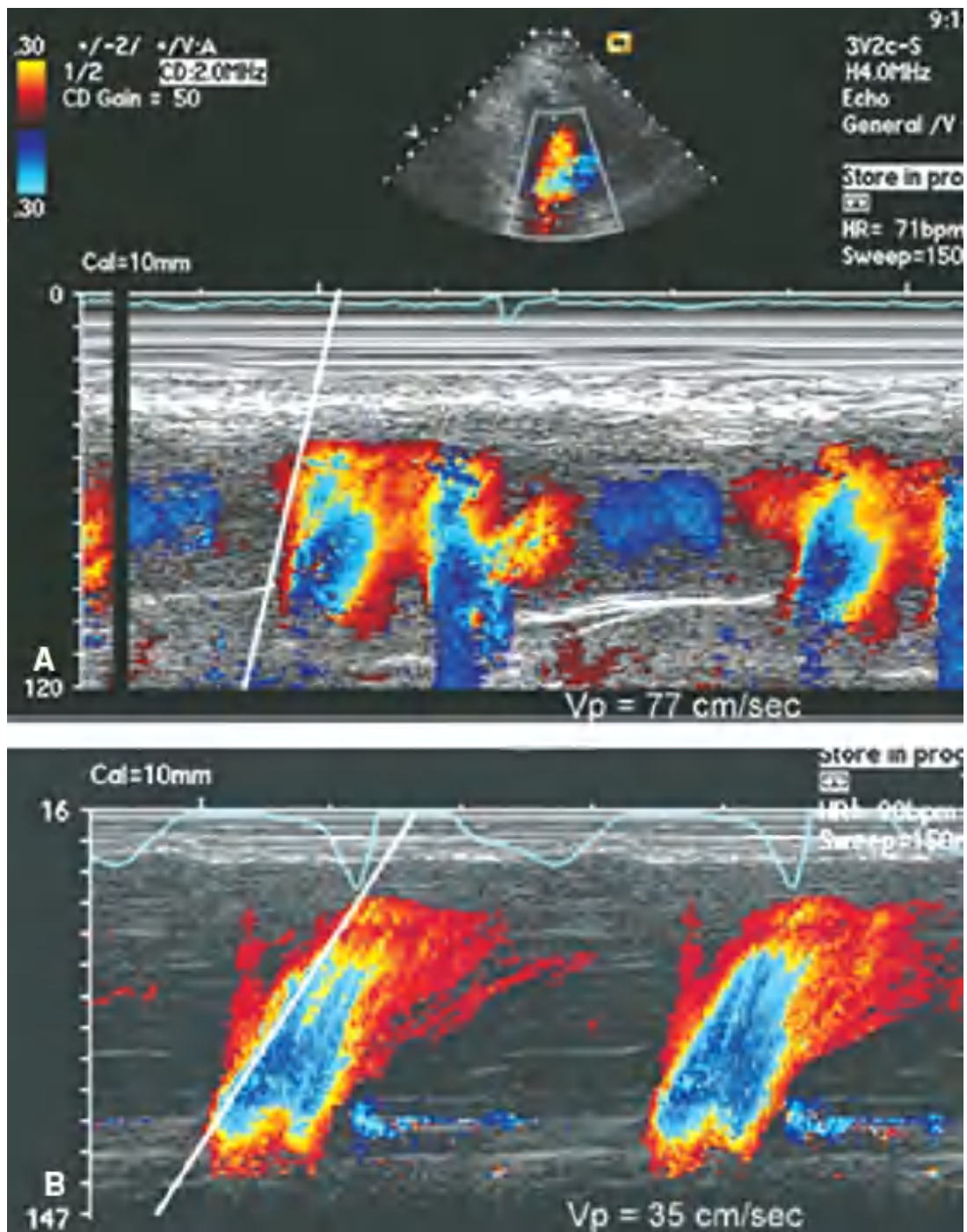


**FIGURE 6.18.** A color Doppler M-mode image recorded from the apical four-chamber view. In **A**, normal flow propagation velocity ( $V_p = 77$  cm/s) is demonstrated as evidenced by the steep slope of the early diastolic valve-to-apex contour. In **B**, the reduced slope and lower velocity ( $V_p = 35$  cm/s) is consistent with decreased chamber compliance.

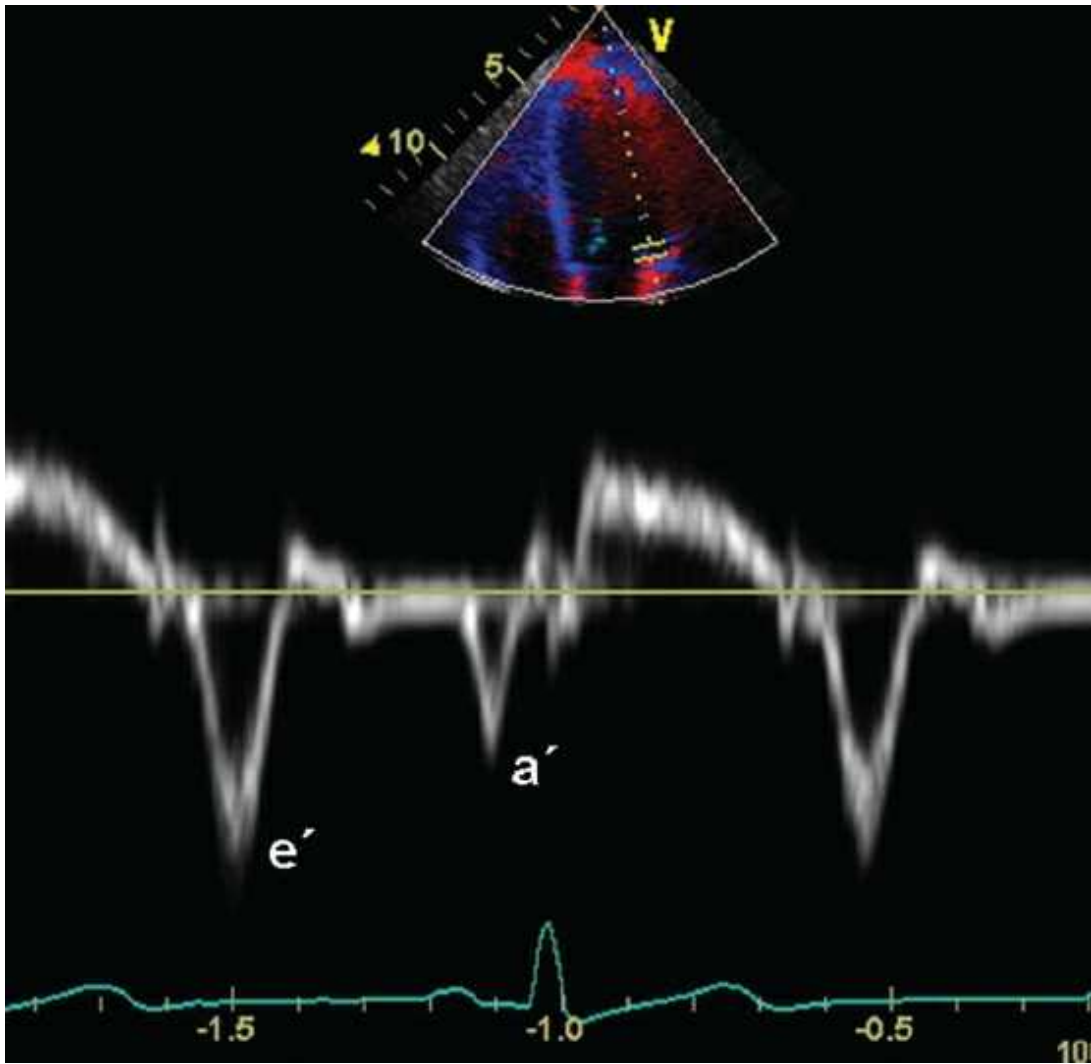


**FIGURE 6.19.** Tissue Doppler recording of the lateral mitral annular velocity from a normal subject is shown. The early (e') and late (a') diastolic annular velocities are labeled.

## 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.

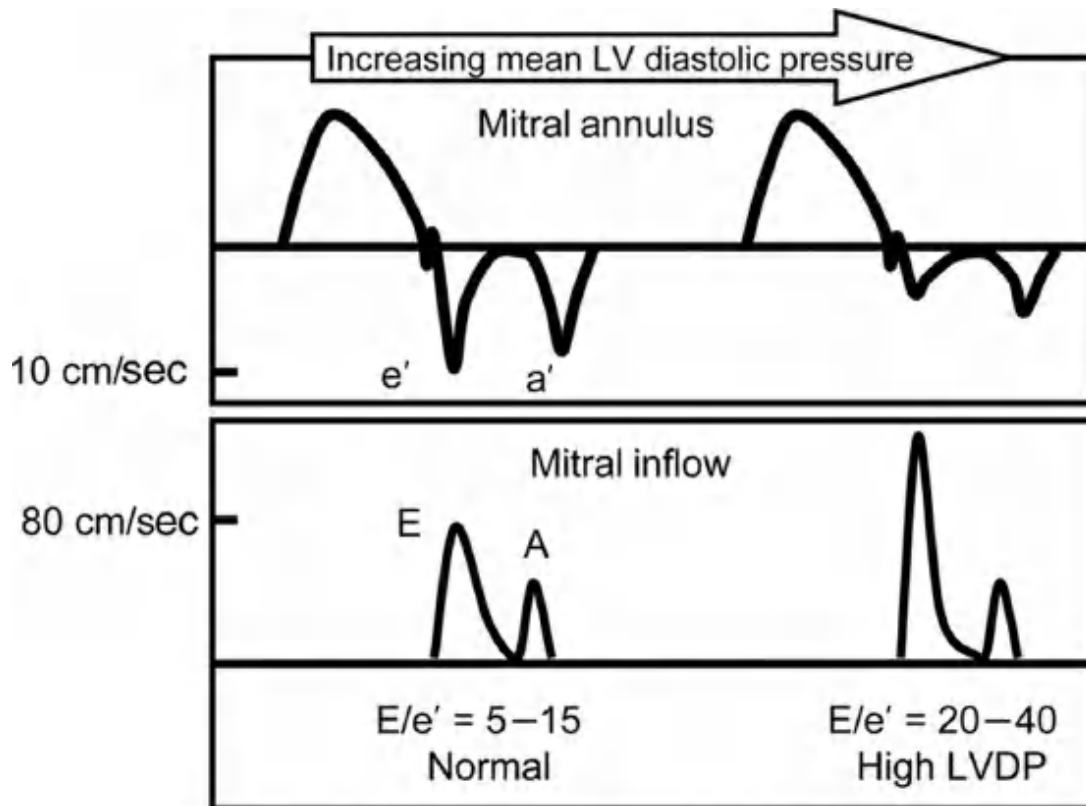


**FIGURE 6.20.** This schematic demonstrates the relationship between mitral annular velocity (**top**) and mitral inflow velocity (**bottom**). As filling pressure increases, the annular  $e'$  velocity decreases while the mitral inflow  $E$  velocity increases, as shown in the right panel. This results in an increase in the  $E/e'$  ratio. LVDP, left ventricular diastolic pressure.



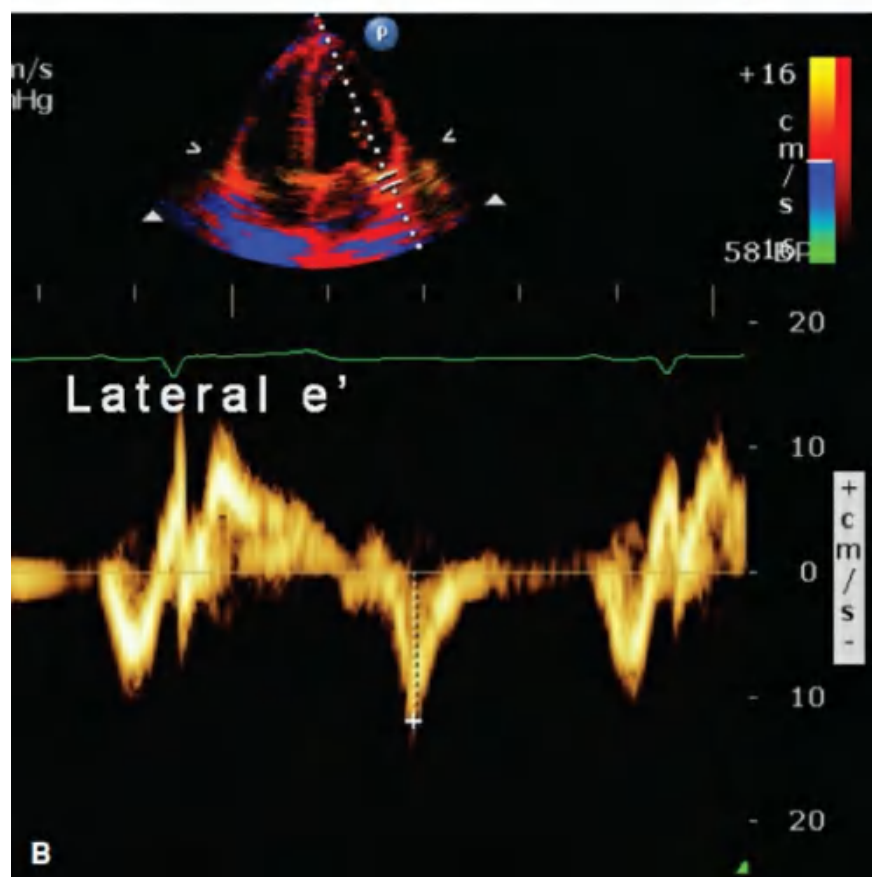
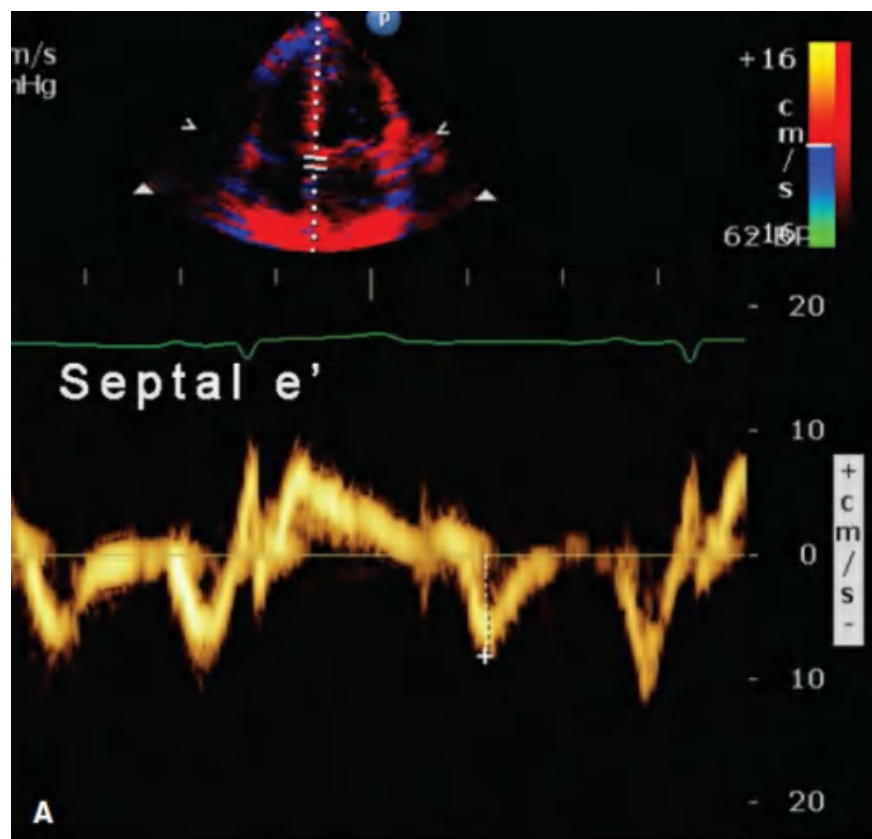


**FIGURE 6.21.** The  $e'$  velocity should be measured at the septal (9 cm/s, **A**) and lateral (12 cm/s, **B**) annulus sites. In most patients, the lateral value will be slightly higher, as shown here.

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%.

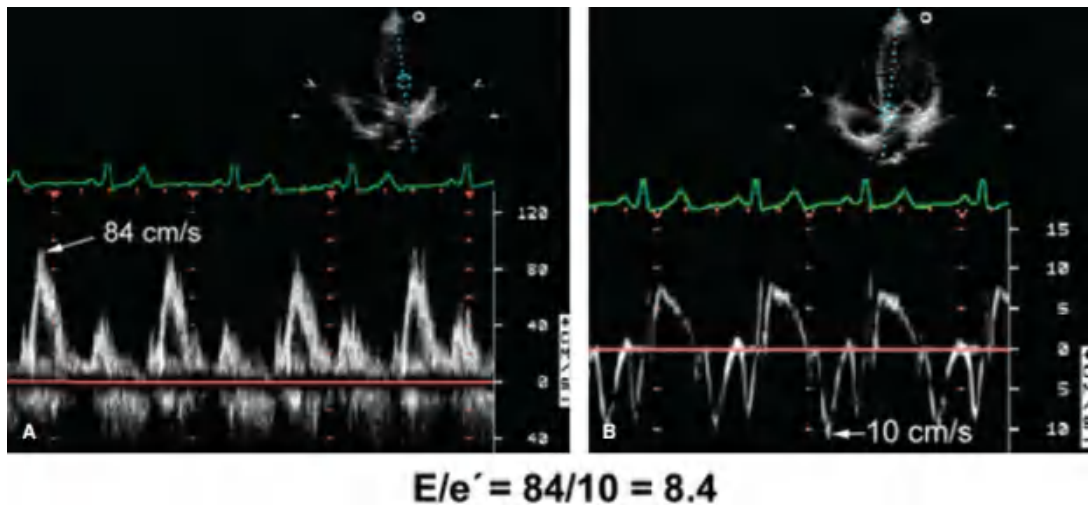


**FIGURE 6.22.** An example of the derivation of the  $E/e'$  ratio is provided in a patient with normal filling pressure. In **A**, mitral inflow is shown and in **B**, tissue Doppler imaging of the mitral annulus is demonstrated. The calculated ratio is 8.4.

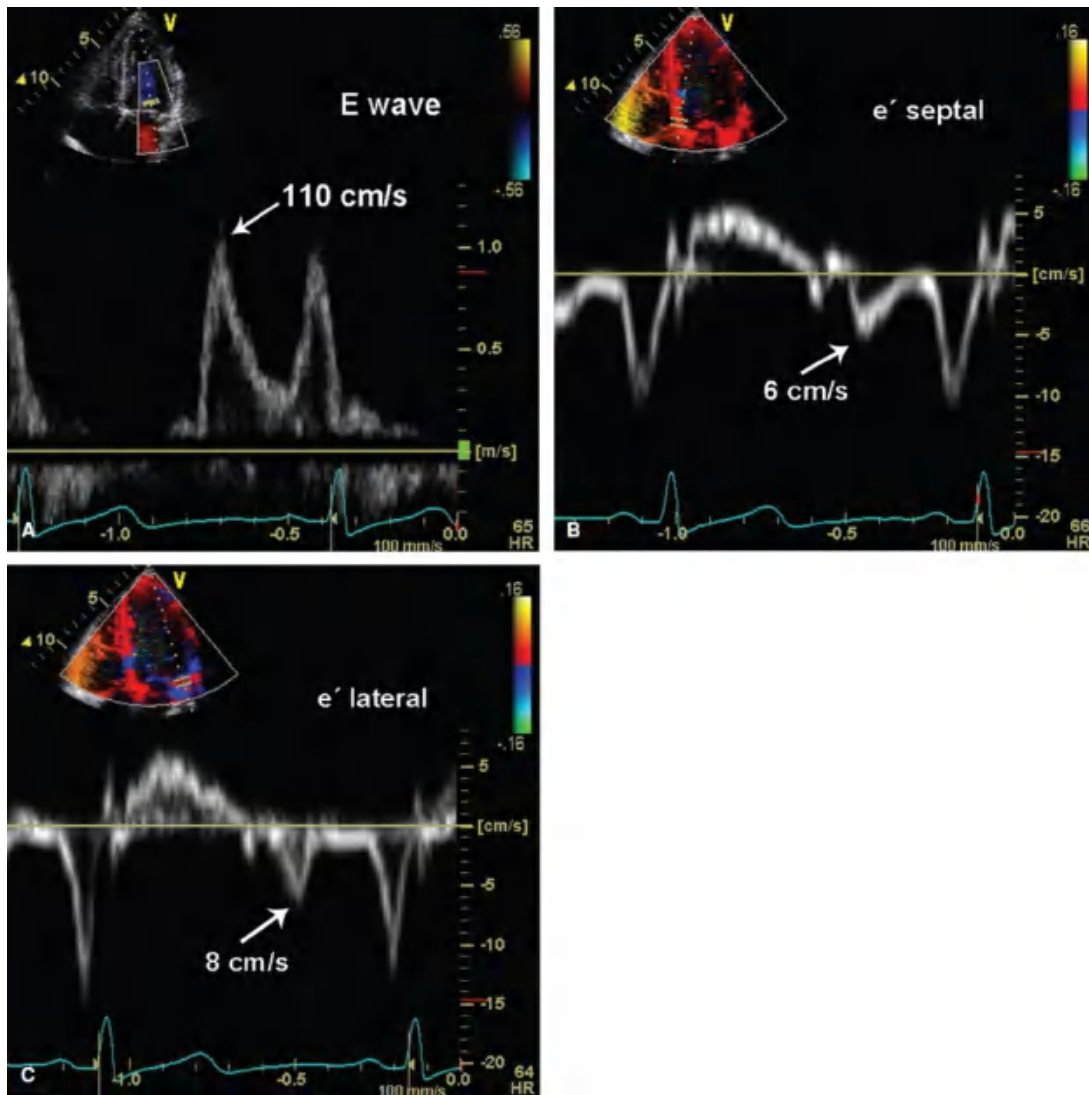


**FIGURE 6.23.** This example, taken from a patient with elevated left ventricular filling pressure, demonstrates an abnormally high E/e' ratio of approximately 18. In (A), mitral inflow demonstrates an E wave velocity of 110 cm/s. Annular e' is recorded in B and C. Note that a different E/e' ratio is obtained depending on whether the septal (B) or lateral (C) e' value is utilized.

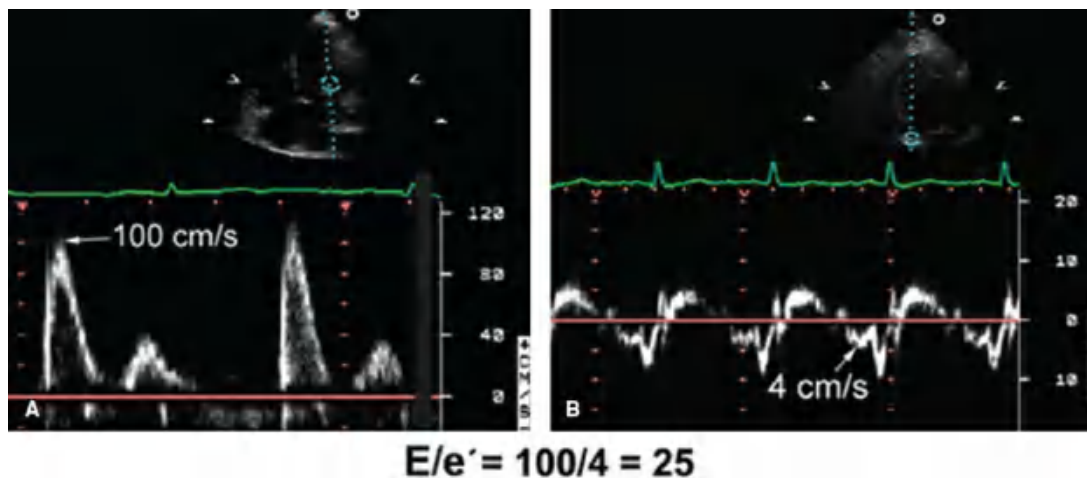


**FIGURE 6.24.** From a patient with restrictive cardiomyopathy, mitral inflow (**A**) and annular velocity (**B**) are recorded. The  $E/e'$  ratio of 25 is consistent with elevated left ventricular filling pressure.

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.

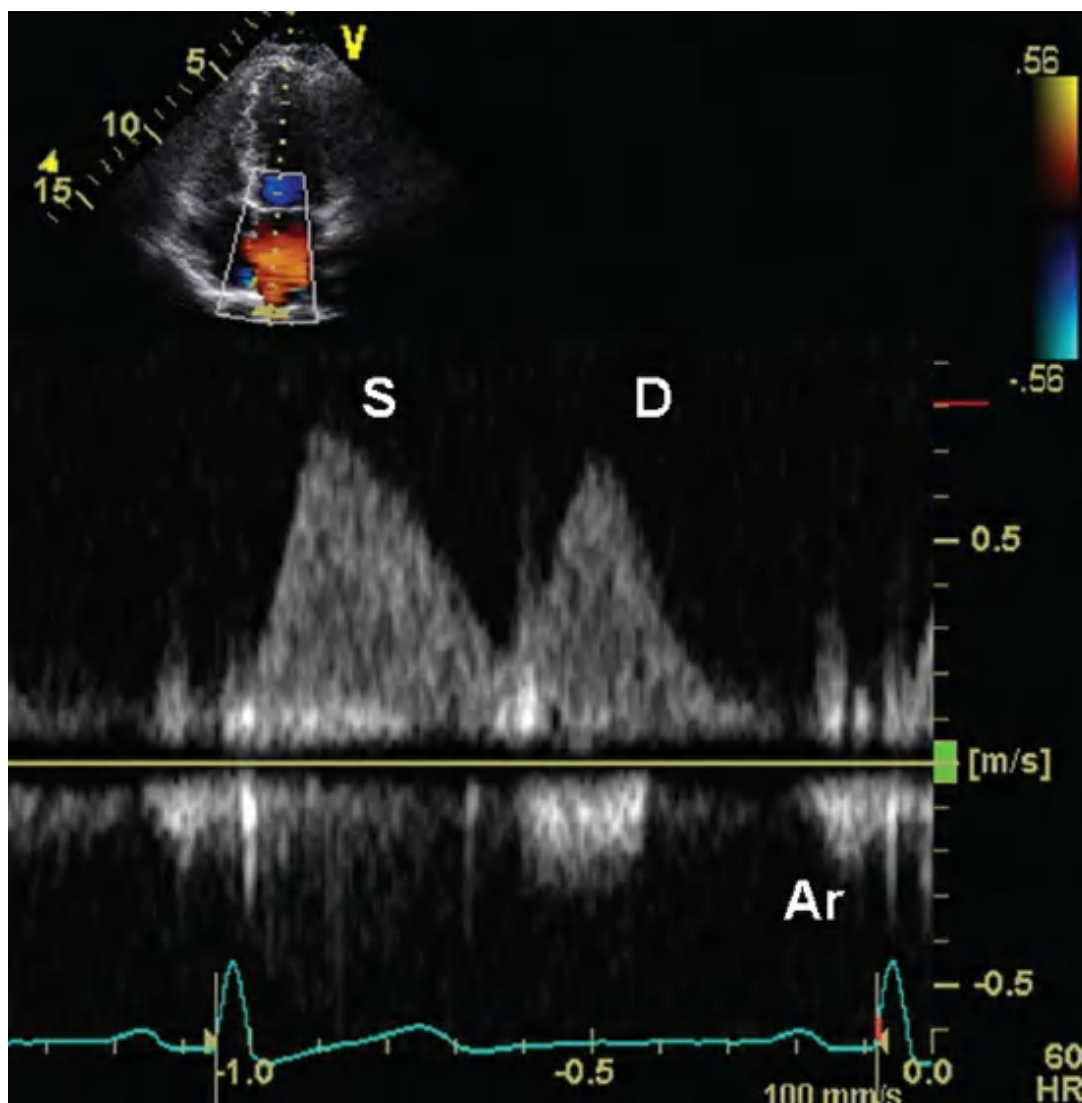


**FIGURE 6.25.** An example of normal pulmonary venous flow is illustrated. Normal flow consists of a systolic wave (S), a diastolic wave (D), and a small wave of flow reversal (Ar) that occurs during atrial systole.

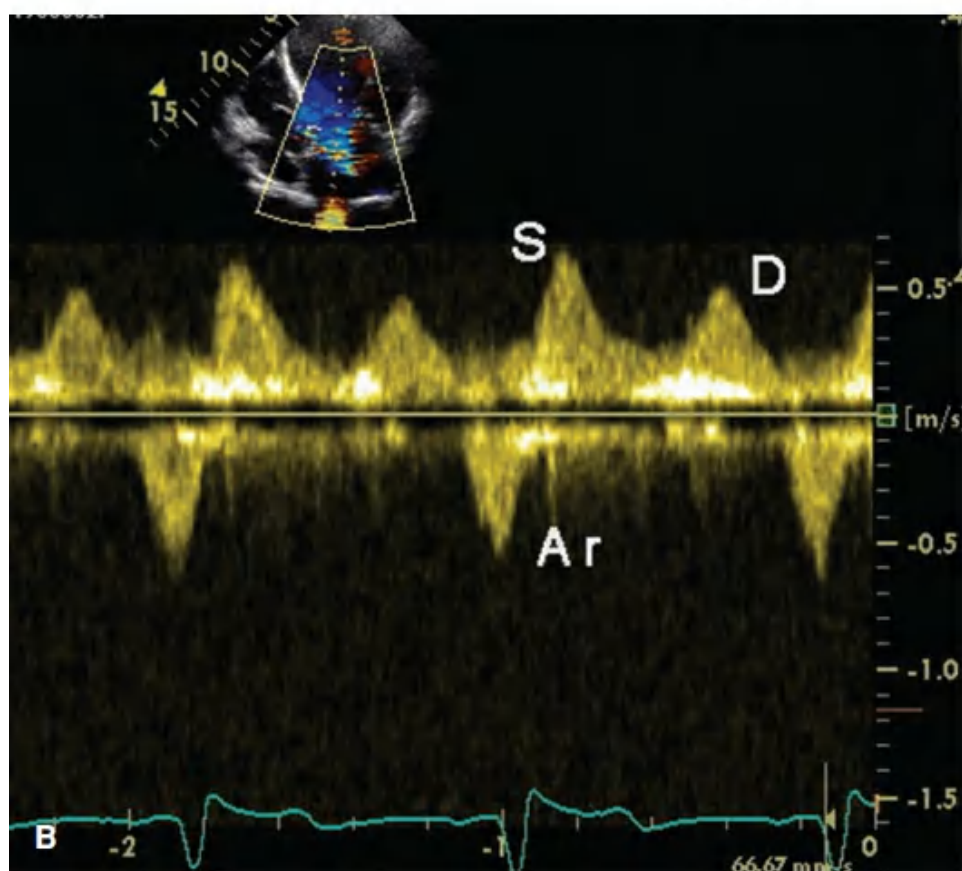
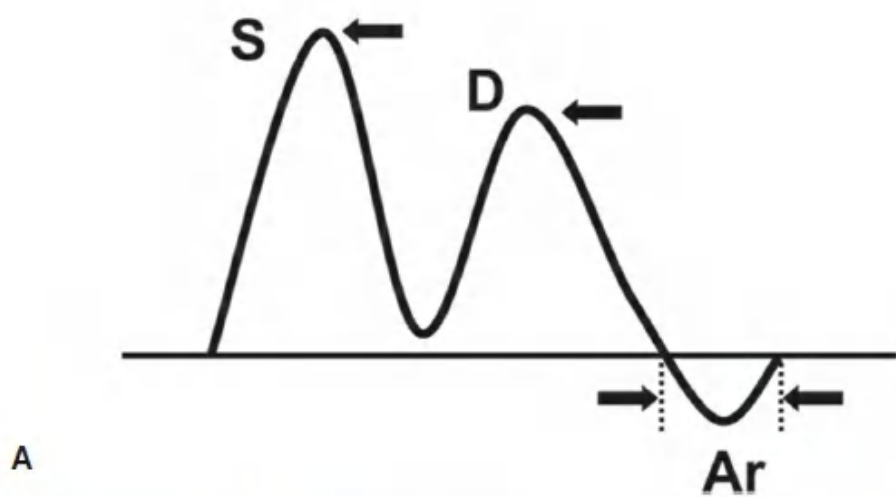


**FIGURE 6.26.** In **A**, the schematic demonstrates the relationship between the three pulmonary venous flow components, recorded using pulsed Doppler, and the electrocardiogram (ECG). The drawing shows how the duration of retrograde flow during atrial systole (Ar) is measured. An example is provided in **B**, showing a very prominent atrial reversal wave (Ar). See text for details.



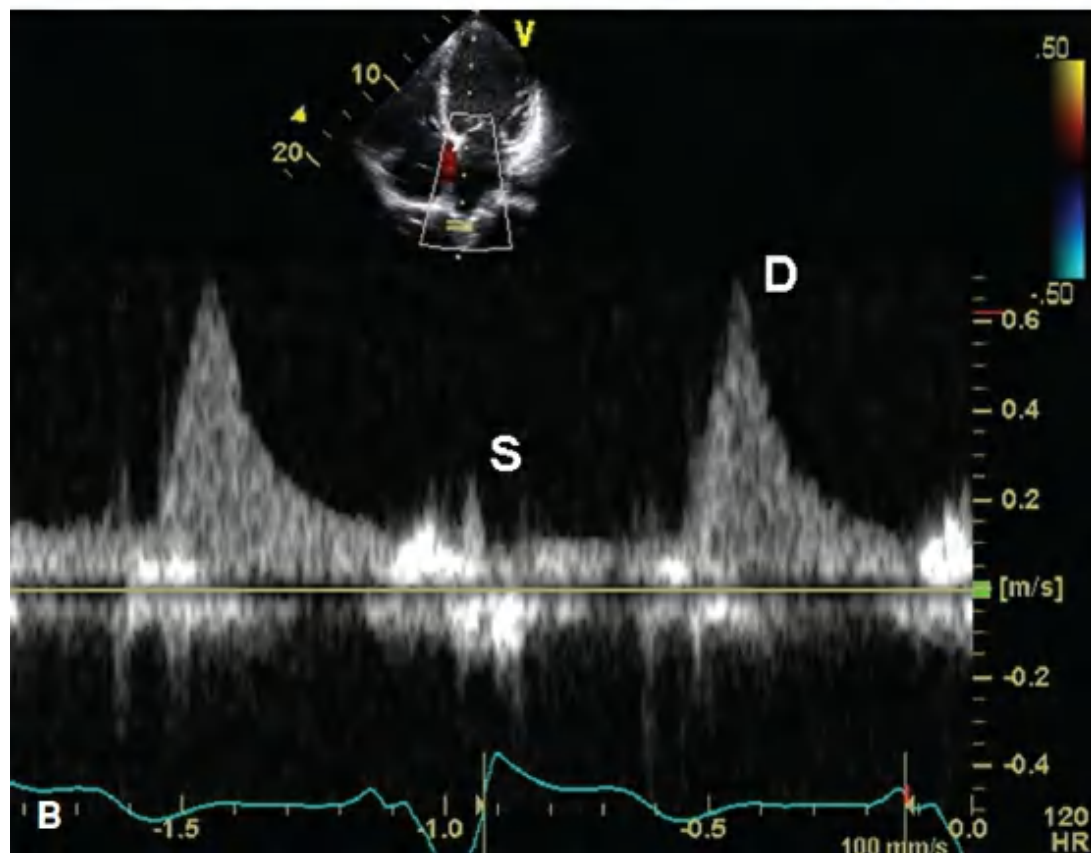
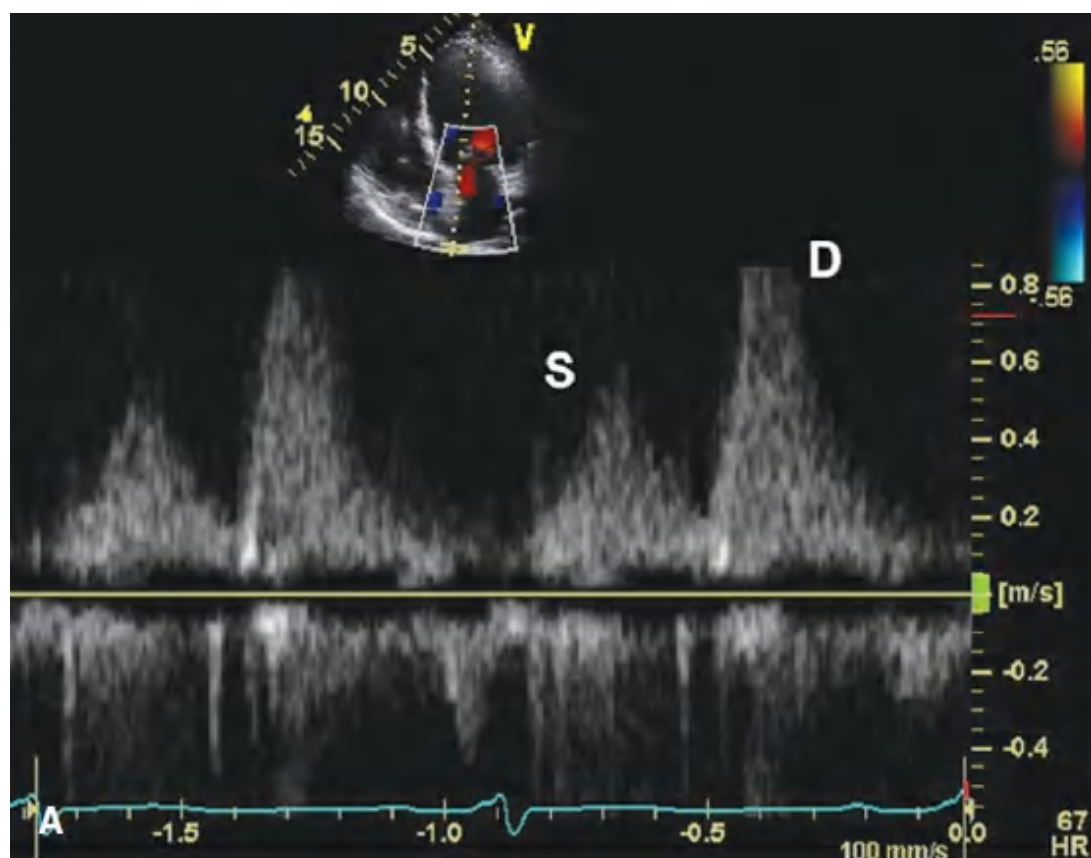




**FIGURE 6.27.** Abnormal pulmonary venous flow patterns are demonstrated. In **A**, diastolic predominance ( $D > S$ ) is present. In **B**, the systolic wave is absent and forward flow occurs exclusively during diastole.

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/m<sup>2</sup>.

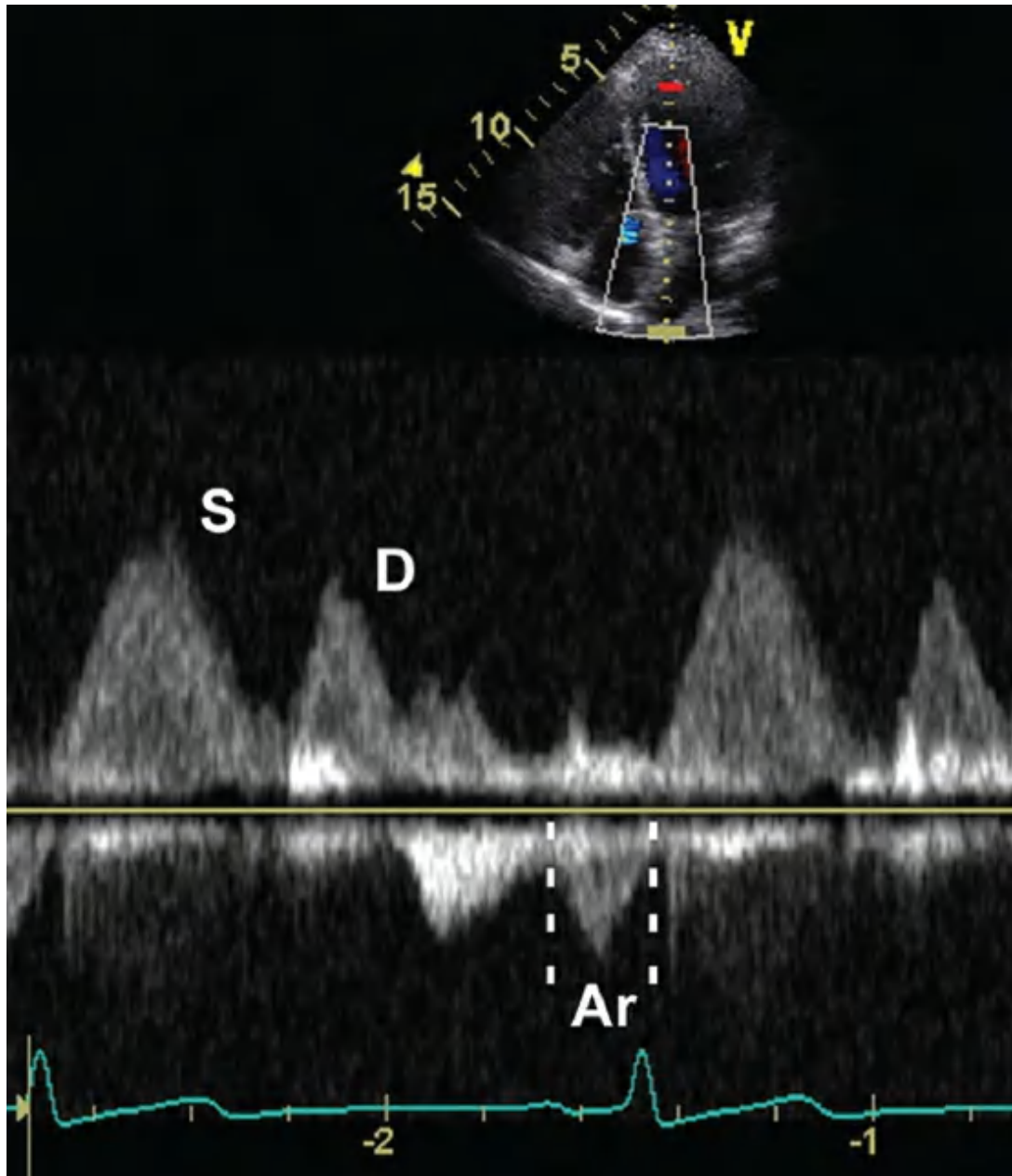


**FIGURE 6.28.** The illustration demonstrates how the S and D waves are recorded as well as the duration of the retrograde wave during atrial systole (Ar). See text for details.

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 phase, 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 phase, 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.

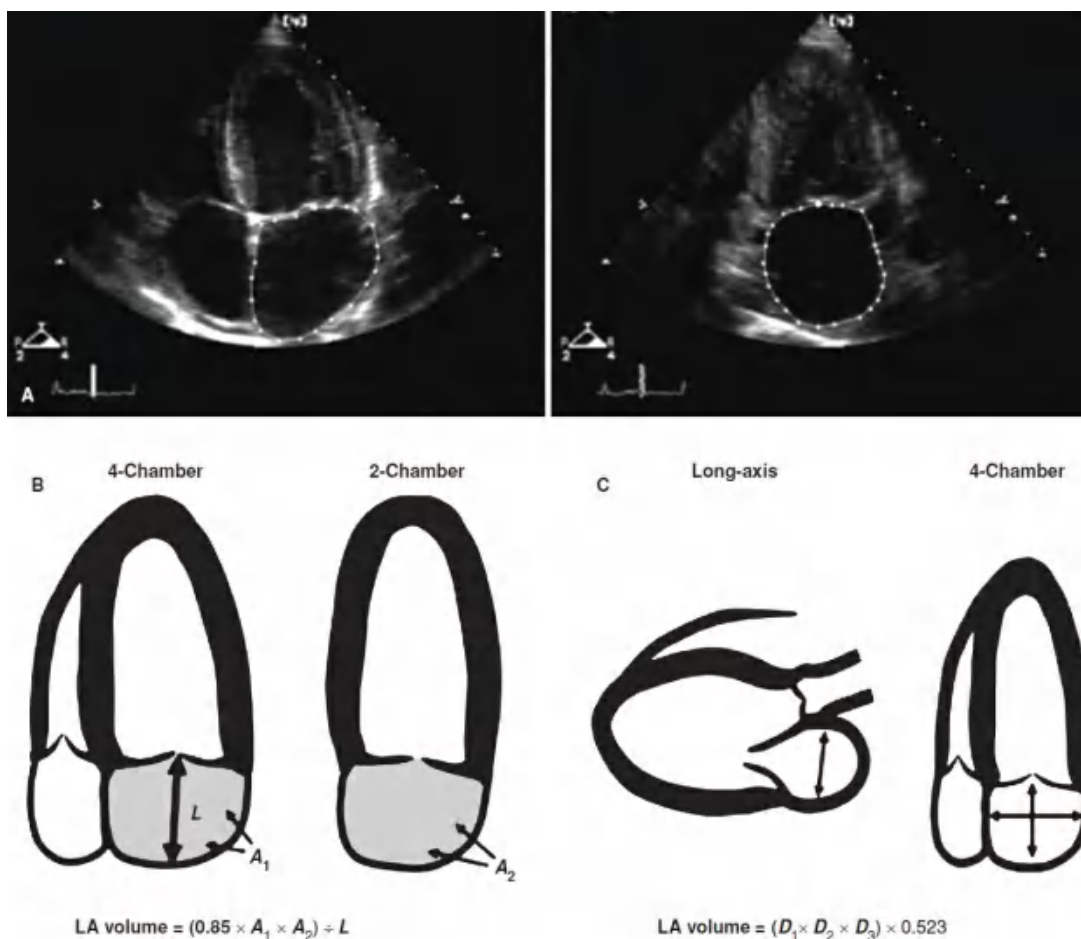


**FIGURE 6.29.** Formulae used to calculate left atrial volume are demonstrated. In **A**, left atrial area is determined by planimetry from the apical four-chamber (left) and two-chamber (right) views (*white dotted lines*). In **B**, determination of left atrial volume using the area-length method is shown. The calculation involves two area measurements ( $A_1$  and  $A_2$ ) and one linear dimension ( $L$ ). In **C**, an alternative formula, using three orthogonal diameters ( $D_1$ ,  $D_2$  and  $D_3$ ) of the left atrium is demonstrated.

## 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