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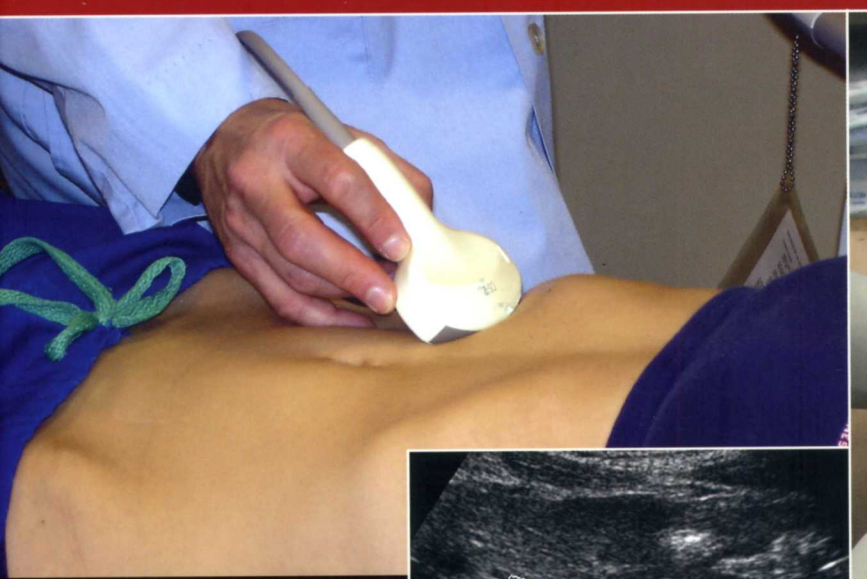
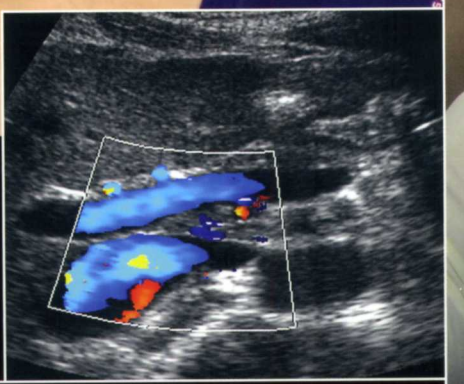
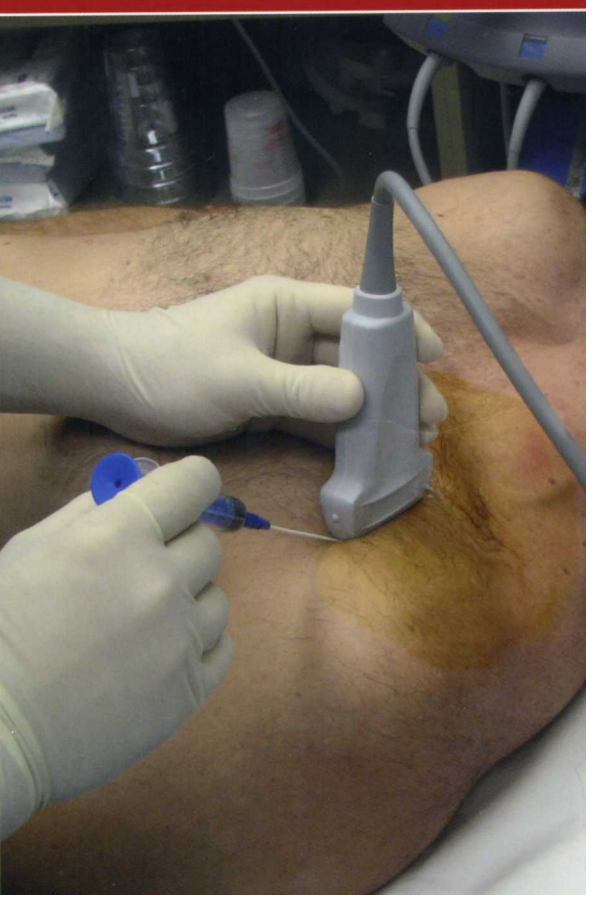






SECOND EDITION

CHAPTER 6

Cardiac

Robert F. Reardon and Scott A. Joing

Echocardiography is the gold standard for the diagnosis of many cardiac and pericardial abnormalities.[1] Echocardiography provides critical information about cardiac structure and function in real time. Since it is impossible for expert echocardiographers to be present in a timely manner for most critical resuscitations, emergency physicians have begun to incorporate focused bedside echocardiography into their daily clinical practice.[2–7] During the past 20 years, there is a growing body of evidence that noncardiologists can use focused echocardiography safely and accurately in a variety of clinical settings.[5,8–23]

Focused echocardiography is a goal-directed examination that is used only to answer defined clinical questions and not to detect all possible cardiac pathology. The key is to keep the examination straightforward by evaluating for gross abnormalities and overall cardiac function.[2,24,25] Focused echocardiography is not meant to replace comprehensive echocardiographic examinations; rather, its purpose is to provide clinicians with vital, real-time information when comprehensive echocardiography is unavailable.[24,26]

▶ CLINICAL CONSIDERATIONS

Focused transthoracic echocardiography is an ideal diagnostic tool for detecting life-threatening cardiac conditions in the emergency department. Much of the information obtained from a focused bedside echocardiographic examination could also be obtained by invasive monitoring techniques. Although emergency physicians and critical care physicians routinely use invasive monitoring, it is not practical to use invasive techniques on all patients with potentially life-threatening conditions. Patients who have quickly reversible hemodynamic compromise do not need invasive monitoring. In addition, placement of invasive monitoring devices is time consuming and has complications.

Without bedside echocardiography or invasive monitoring, clinicians would be left to manage critically ill patients with only indirect information about cardiac structure and function. "Classic" physical examination findings and changes in vital signs are often absent and unreliable for making critical diagnoses. An electrocardiogram (ECG) is very helpful in patients with certain

cardiovascular problems who have diagnostic findings, but the majority of critically ill patients have nonspecific ECG findings. A chest radiograph may also provide some helpful information, but is just as likely to be nonspecific as well.

In cardiac arrest with pulseless electrical activity (PEA), it is critical to determine whether the patient has true electromechanical dissociation (EMD) with cardiac standstill or pseudo-EMD with mechanical cardiac contractions too weak to generate a palpable blood pressure.[27] Some patients thought to be in cardiac arrest have extreme hypotension. Other patients with PEA have cardiac tamponade, massive pulmonary embolism (PE), or severe left ventricular dysfunction. All of these conditions can be detected with bedside transthoracic echocardiography. Echocardiography can be performed serially during a critical resuscitation as long as the examination itself does not interfere with resuscitative efforts.

A controversial use of focused echocardiography is for patients who are stable and minimally symptomatic. Stable patients presenting with nonspecific symptoms may benefit from a focused echocardiographic examination. Pericardial effusions often cause nonspecific or minimal symptoms until tamponade develops. Focused echocardiography is the most efficient method to evaluate for "silent" pericardial effusion since it is not reasonable to order a comprehensive echocardiographic examination on every patient with such vague complaints in the emergency department. Also, 6% of all patients over 45 years of age have "silent" heart failure.[30] Many patients also have "silent" valvular disease and may benefit from focused echocardiography, even if the only information gained is an estimate of gross chamber size.

Transesophageal echocardiography (TEE) may be used as an alternative or complementary diagnostic tool to transthoracic echocardiography. While transthoracic echocardiography does not require sedation or airway protection, TEE is a more invasive procedure that may necessitate those maneuvers. Nevertheless, although not yet widely used by emergency or acute care physicians, TEE has the capability of obtaining excellent resolution of intracardiac abnormalities with little artifact or ultrasound window difficulty. TEE is portable and can be performed at the bedside and during ongoing cardiopulmonary resuscitation without interruption of chest procedures. Other advantages include that it is reproducible, rapid, and accurate. Disadvantages of TEE are that it is very operator dependent, may not be well tolerated by some patients, and may expose patients to vomiting and aspiration. Limitations of TEE are that it may not visualize the extension of aortic dissection into supra-aortic arteries and that it may have difficulty assessing a small distal portion of the ascending aorta and branches of the aortic arch behind the respiratory tract.[28–31] Table 6-1 contrasts transthoracic echocardiography with various other diagnostic testing and evaluation methods.

▶ CLINICAL INDICATIONS

Any patient at risk for significant cardiovascular compromise is a candidate for a focused bedside echocardiographic examination. Patients with significant disease have widely varying presentations, from cardiac arrest to vague symptoms of dizziness, shortness of breath, or chest discomfort.[32,33] The challenge is to determine which echocardiographic findings can be readily recognized by noncardiologists with minimal training and which patients need comprehensive echocardiography.

Primary indications for performing focused echocardiography include

- cardiac arrest,
- pericardial effusion,
- massive pulmonary embolism,
- assessment of left ventricular function,
- unexplained hypotension,
- estimation of central venous pressure, and
- external cardiac pacing.

Other indications that require more training and experience with focused echocardiography include

- severe valvular dysfunction,
- proximal aortic dissection, and
- myocardial ischemia.

▶ CARDIAC ARREST

Bedside echocardiography is invaluable in helping resuscitate patients with PEA. Prior to the 1980s all patients with an organized electrical rhythm but no pulse were thought to have EMD. In the mid-1980s, using arterial catheters and echocardiography, physicians discovered that many patients with apparent EMD have mechanical contractions that are too weak to produce a blood pressure detectable by palpation.[2,3,34–36]

A 1988 study described two very different categories of EMD, those with "true" EMD and those with mechanical cardiac contractions. It observed that those with "true" EMD, or cardiac standstill, have a dismal prognosis, similar to the prognosis for asystole.[2]

It has been reported that 86% of patients with PEA have mechanical cardiac contractions.[36]

Studies have confirmed that palpation of pulses is an unreliable means of assessing cardiac function and blood pressure. One study questioned whether carotid, femoral, or radial pulses correlated with certain blood pressure measurements.[37] They attempted to palpate pulses in 20 hypotensive patients who had arterial lines

▶ TABLE 6-1. EMERGENT TRANSTHORACIC ECHOCARDIOGRAPHY CONTRASTED WITH OTHER DIAGNOSTIC TESTS

	TTE	Clinical Examination	CVP	PA Line	EKG	Chest Radiograph	Arteriography	CT	MRI	Pericardial Window	Thoracotomy
Ease of use	+ + +	+ + +	+	+	+ + +	+ + +	+	+ +	+ + +	+	+
Diagnostic cardiac accuracy	+ + +	+	+	+ +	+ +	+ +	+ + +	+ +	+ + +	+ + +	+ + +
Lack of invasiveness	+ + +	+ + +	+	+	+ + +	+ + +	+	+ +	+ +	+	+
Limitations	Aortic anatomy, coronary anatomy, pulmonary anatomy, valvular vegetation	Pulmonary embolism, myocardial ischemia	LVEDP not measured	Invasive pressures only measured	Lack of anatomical findings, nonspecificity for valvular lesions and aortic disease	Chamber size, hemodynamics, location of disease	Dye load, catheterization laboratory availability, valvular anatomy, right and left heart—different procedures	Breath hold, intracardiac anatomy, pressure readings	Breath hold, need for stable patient	Intracardiac anatomy	Invasiveness, need for bypass
Strengths	Repeatable, noninvasiveness, bedside intracardiac anatomy	Valvular, lack of invasiveness	Availability, right heart pressures	Blood sampling, LVEDP accurate measurement	Availability	Pulmonary disease, availability	Coronary anatomy, chamber pressure, aortic disease	Other thoracic/abdominal anatomy, aortic disease	Other thoracic/abdominal anatomy, aortic disease	Avoidance of thoracotomy	Proximal control, repair of cardiac/thoracic lesions

Note. TTE = Transthoracic echocardiography; CVP = central venous pressure; PA = pulmonary artery; EKG = electrocardiogram; CT = computed tomography; MRI = magnetic resonance imaging; LVEDP = left ventricular end-diastolic pressure
0 = None; + = Minimal; + + = Moderate; + + + = Large.

and invasive blood pressure measurement already established. They found that as systolic blood pressure declined the radial pulse always disappeared before the femoral pulse, which always disappeared before the carotid pulse. Surprisingly, the disappearance of pulses from a specific location did not correlate with an absolute blood pressure but was widely variable; for instance, several patients had palpable carotid pulses with a measured systolic blood pressure between 30 and 60 mmHg. More worrisome was the finding that about 10% of these patients had no palpable carotid pulse with measured systolic blood pressures between 50 and 80 mmHg.[37]

Since carotid pulses are an unreliable means for determining true cardiac arrest, basic life support guidelines put forth by the American Hearth Association no longer recommend that lay people even try to check for a carotid pulse.[38] One study found that health care providers may not be any better than lay people when palpating carotid pulses and questioned the use of carotid pulse checks during cardiopulmonary resuscitation (CPR), even by health care providers.[39] Bedside echocardiography allows clinicians to directly visualize the heart and determine the presence and quality of mechanical cardiac function during a cardiac arrest. If a carotid pulse is absent but echocardiography shows reasonable mechanical cardiac function, then clinicians should proceed with aggressive resuscitation.

Several studies have examined whether bedside echocardiography in PEA could predict the outcome of cardiac arrest resuscitation.[8,21,22,40] A 2001 study reported that 56% of patients presenting to two community hospitals with PEA had cardiac contractions on bedside echocardiography; 26% of those with contractions and 4% (one patient) with cardiac standstill survived to hospital admission.[22] In a multicenter trial from four academic centers, 32% of patients with PEA had mechanical contractions. No patient with cardiac standstill had return of spontaneous circulation (ROSC) and 73% of those with mechanical contractions had ROSC.[21]

The data suggest that when echocardiography shows cardiac standstill it may be reasonable to consider terminating resuscitative efforts although return of mechanical contraction was reported in 4 of 18 patients who initially had cardiac standstill.[41] In current practice, many emergency physicians use echocardiography to confirm cardiac standstill before terminating resuscitation in all cardiac arrests. Beyond predicting the outcome of resuscitation, echocardiography is essential in helping to rapidly identify the cause of the cardiac arrest since PEA is often associated with specific clinical states that may be readily reversed when identified and treated appropriately.[27] The most common causes of PEA are hypovolemia, hypoxia, acidosis, hypo/hyperkalemia, hypoglycemia, hypothermia, drug overdose, cardiac tamponade, tension pneumothorax, massive myocardial infarction, and massive PE.[34] Hypovolemia, cardiac tamponade, massive PE, and massive myocardial infarction can be detected by bedside echocardiography so that early aggressive management of these abnormalities can then be instituted.[41–44]

The first question to answer using bedside echocardiography is whether the etiology of the arrest is likely to be cardiac or noncardiac. Severe left ventricular dysfunction from massive myocardial infarction, acidosis, drug overdose, or electrolyte abnormality will be apparent. Kaul and colleagues reported using a similar approach to determine whether hypotension had a cardiac or noncardiac etiology. The authors compared 2-D echocardiography to hemodynamic measurements from pulmonary artery catheters and found that in 86% of cases echocardiography correctly determined whether the etiology of shock was cardiac or noncardiac.[45] In addition, studies have shown that emergency physicians can accurately estimate left ventricular function, especially when left ventricular dysfunction is severe.[15,19,46]

Patients in cardiac arrest with PEA as a result of severe hypovolemia will have a small, empty appearing heart on bedside echocardiography.[42] Both the right and left ventricles will be poorly filled and the right ventricle will be almost completely collapsed. The left ventricle will usually be vigorous. The inferior vena cava (IVC) will have a small diameter and its lumen will disappear completely during inspiration (or expiration when positive pressure ventilation is being used). These findings on bedside echocardiography should prompt the clinician to aggressively replace volume and consider the etiology of hypovolemia.

The most straightforward application of bedside echocardiography during cardiac arrest is evaluating for a pericardial effusion.[2,18,47] A pericardial effusion presents as an anechoic stripe surrounding the heart and should be obvious if it is large enough to cause cardiac tamponade. This finding should prompt the clinician to perform an immediate pericardiocentesis using echocardiography guidance (see chapter 20). Pericardiocentesis can be life-saving and the removal of just a small amount of fluid may result in significant improvement in cardiac output.[2,3,5,44]

Massive PE is responsible for about 10% of cardiac arrests in cases where a primary cardiac etiology is clinically suspected.[48,49] The routine use of bedside echocardiography in cardiac arrest may allow immediate detection of massive PE, even in cases where the diagnosis is not clinically suspected.[43,50–54] It is important to immediately recognize that PE is the cause of a cardiac arrest because early thrombolytic therapy has been shown to significantly improve the chance of ROSC. A review of 60 cases of cardiac arrest caused by massive PE found that 81% of patients who received early thrombolysis had ROSC compared with 43% for those who did not receive the therapy.[48]

▶ PERICARDIAL EFFUSION

In 1992, Plummer reported 100% sensitivity for recognizing pericardial effusion in the first 6 years after his group of emergency physicians began screening penetrating trauma patients with echocardiography. More importantly, the use of bedside echocardiography significantly reduced the time to diagnosis and disposition to the operating room from 42.4 minutes to 15.5 minutes while the actual survival improved from 57.1 to 100%.[19] Bedside focused echocardiography is now the standard of care for patients with potential penetrating cardiac injuries.[20,55,56]

Patients at risk for nontraumatic pericardial effusions are more difficult to identify clinically because they usually have nonspecific signs and symptoms. Most patients with a pericardial effusion are stable and have nonspecific symptoms such as dyspnea, chest pain, cough, or fatigue.[57,58] Since cardiac tamponade can develop rapidly, even in those with chronic pericardial effusion, it is prudent to make the diagnosis as early as possible. Patients at risk for a pericardial effusion include those with the following disease processes: idiopathic/viral pericarditis, HIV, hepatitis B, bacterial pericarditis, fungal pericarditis, lupus, rheumatoid arthritis, scleroderma, polyarteritis nodosa, temporal arteritis, early postmyocardial infarction, Dressler syndrome, drug induced (isoniazid, cyclosporine), neoplastic, radiation induced, postcardiac surgery, postcardiac procedure/device, chronic renal failure, hypothyroidism, amyloidosis, aortic dissection, congestive heart failure, and blunt/penetrating chest trauma.[57,59] Effusions associated with neoplastic disease or bacterial, fungal, or HIV infections have a higher risk of progressing to tamponade.[59] Patients who have had recent invasive cardiac procedures, such as coronary angiography and pacemaker or defibrillator placement, are at high risk for pericardial effusion with tamponade.

Any patient with a pericardial effusion is at risk for developing cardiac tamponade, which is a life-threatening condition that occurs when a pericardial effusion causes significant compression of the heart leading to a decrease in cardiac output. When a pericardial effusion develops acutely, tamponade can occur with as little as 150 mL of fluid. Because the parietal pericardium can stretch over time, a chronic effusion can have a volume of more than 1000 mL without causing tamponade. The rate of pericardial fluid accumulation relative to pericardial stretch is the critical factor. The steep rise in the pericardial pressure-volume curve makes tamponade a "last-drop" phenomenon, the last few milliliters of fluid accumulation produce critical cardiac compression and the first milliliters of drainage produce the largest relative decompression.[58,60] Cardiac tamponade presents with hypotension that rapidly progresses to PEA and death if not rapidly diagnosed and treated.[61,62] Echocardiography is now the standard means to evaluate for cardiac tamponade[63] and can also be used to guide pericardiocentesis.

One study evaluated bedside echocardiography on 103 patients with unexplained dyspnea and found that 14 had pericardial effusions. Four had large effusions requiring pericardiocentesis and 3 had moderate-sized effusions that were treated conservatively. The authors recommended that emergency department patients with unexplained dyspnea should be evaluated for pericardial effusion.[32] Mandavia and colleagues performed bedside echocardiography on 515 patients presenting to their emergency department with high-risk risk criteria to determine the accuracy of bedside echocardiography performed by emergency physicians to detect pericardial effusions. These criteria consisted of unexplained hypotension or dyspnea, congestive heart failure, cancer, uremia, lupus, or pericarditis. They found 103 pericardial effusions in this high-risk population and determined that bedside echocardiography by emergency physicians was 97.5% accurate.[57]

Most nonhemorrhagic pericardial effusions that cause tamponade are moderate to large (300–600 mL) in size.[58] Therefore, an indication for emergent pericardiocentesis is the finding of a moderate or large effusion in a patient with clinical signs of tamponade. Large pericardial effusions surround the entire heart while small effusions collect first around the more dependent, mobile ventricles.[47] Effusions can be categorized by the maximal width of the echogenic pericardial stripe. A stripe less than 10 mm is small, 10–15 mm is moderate, and greater than 15 mm is large.[32] These are gross measurements and do not correlate perfectly to the volume of the effusion. Also, hemorrhagic effusions can occur outside the setting of trauma and tend to accumulate rapidly and cause tamponade even when they are small.

▶ MASSIVE PULMONARY EMBOLISM

Massive pulmonary embolism (PE) is a condition for which bedside echocardiography is invaluable for making a rapid diagnosis.[51,52,64,65] Echocardiography can also help exclude diagnoses that mimic PE such as pericardial tamponade, pneumothorax, and myocardial infarction.[64] Patients with massive PE often present with significant symptoms or with impending cardiac arrest. Patients who present in extremis require rapid intervention since 70% of patients who die from PE die within the first hour. Early thrombolytic therapy or embolectomy is required and rapid treatment often precludes obtaining time-consuming imaging studies.[66–74] This is a challenging scenario for clinicians because thrombolytic therapy can have significant morbidity and embolectomy requires mobilization of significant resources. Bedside

echocardiography can help clinicians quickly confirm or refute the clinical diagnosis of massive PE. The echocardiographic findings in massive PE are not subtle and include massive right ventricular dilatation and right-sided heart failure with a small vigorously contracting left ventricle.[43] In some cases, thrombus can actually be seen in the right atrium or ventricle.

The detection of massive PE by bedside echocardiography should not be confused with using echocardiography to evaluate hemodynamically stable patients suspected of having PE. In stable patients without severe symptoms right-sided heart strain is often subtle and difficult to appreciate on bedside echocardiography. Also, there are many other underlying diseases that cause chronic right-sided heart strain, thus making the diagnosis of acute cor pulmonale in these patients difficult and unreliable.[75]

▶ ASSESSMENT OF LEFT VENTRICULAR FUNCTION

Echocardiography is the preferred first-line test for patients with symptoms or signs consistent with left ventricular dysfunction.[76] Patients who present with nonspecific complaints may have unexpected left ventricular failure. More than half of patients with moderate to severe systolic dysfunction have never been diagnosed with heart failure.[28] Patients with moderate to severe left ventricular dysfunction are at higher risk for complications regardless of their presentation. In addition, when patients present with cardiogenic shock both short- and long-term mortality are associated with the degree of initial left ventricular systolic dysfunction and the degree of mitral regurgitation.[77]

Several studies have demonstrated that noncardiologists can use focused ultrasound in assessment of left ventricular function by estimating left ventricular ejection fraction. One study reported that emergency physicians with prior ultrasound training and 16 hours of additional training could accurately measure left ventricular ejection fraction (LVEF) in hypotensive emergency department patients.[15] All of the echocardiographic examinations were recorded and reviewed by two masked cardiologists. Emergency physicians and cardiologists had 84% overall agreement compared with 88% interobserver agreement between the two cardiologists. The study concluded that bedside echocardiography adds diagnostic value in patients presenting to the emergency department with unexplained hypotension.

Another study reported that emergency physicians with only 3 hours of additional training could accurately estimate left ventricular ejection fraction. They performed a bedside echocardiography on emergency department patients who had a "comprehensive echocardiography" ordered for any reason regardless of hemodynamic status or symptoms. The authors found 86% overall agreement between emergency physicians and cardiologists. Their technique for determining LVEF was restricted to a subjective visual estimate of the change in left ventricular size in diastole versus systole. There was no attempt to clinically correlate these results but the authors suggested that emergency physicians could use bedside echocardiography to differentiate patients with primary pump failure from those with other potential causes of hypotension.[19]

Noncardiologists who use echocardiography to assess left ventricular function should be aware that left ventricular function includes more than just the LVEF. Ejection fraction is largely an assessment of systolic function. Measuring LVEF usually leads to correct interpretation of pathophysiology, especially in cases of heart failure caused by coronary disease and cardiomyopathy. Ejection fraction may not be a good indicator of cardiac output for aortic stenosis, mitral regurgitation, and concentric left ventricular hypertrophy.[78] Diastolic dysfunction is also an important factor in heart failure. Approximately 50% of patients with overt congestive heart failure have diastolic dysfunction without reduced ejection fraction. Determination of diastolic dysfunction requires Doppler measurements.

▶ UNEXPLAINED HYPOTENSION

Unexplained hypotension or shock is a common presentation in the emergency department or critical care setting. The main utility of bedside echocardiography in shock is to allow clinicians to immediately narrow the differential diagnosis and begin early aggressive resuscitation. A simple assessment of global cardiac function and chamber size allows clinicians to assign hypotensive patients to one of four diagnoses: cardiogenic shock from severe left ventricular dysfunction, cardiac tamponade, massive PE, or severe hypovolemia.[47] In addition to assessment of cardiac chamber size and left ventricular function, evaluation of the proximal IVC can be easily used to estimate right atrial filling pressure. Hypovolemia should be considered whenever low (or moderate) right atrial filling pressure is found in combination with a hyperdynamic left ventricle. When hypovolemic shock is suspected from echocardiographic findings, a search for hemorrhagic etiologies may include sonographic examinations for free intraperitoneal fluid and ruptured abdominal aortic aneurysm.[79]

Shock resulting from severe left ventricular dysfunction, cardiac tamponade, or massive PE should be readily apparent on bedside echocardiography.[15,18,19,43,44,46] The finding of moderate or severely reduced left ventricular ejection fraction does not prove that an acute or

primary cardiac abnormality is the cause. Clinicians must realize that there are many other factors to consider such as preexisting cardiac disease and the effects of drugs or acidosis on left ventricular function. As with all echocardiographic findings, clinical correlation is required, but the unexpected finding of significant left ventricular dysfunction in any hypotensive patient is always clinically important.

A prospective study analyzed bedside echocardiography to evaluate the left ventricular function of emergency department patients with atraumatic, unexplained hypotension. Seventeen percent of the patients were found to have a hyperdynamic left ventricle. Of those with a hyperdynamic left ventricular, 76% had a final diagnosis of sepsis. The authors concluded that in emergency department patients with atraumatic, unexplained hypotension the finding of a hyperdynamic left ventricle is highly specific for sepsis as the etiology of shock.[15] Extending the bedside ultrasound examination to include assessment for intraperitoneal fluid or abdominal aortic aneurysm is appropriate in some settings.[47] Rose and coinvestigators reported using an undifferentiated hypotensive patient protocol for the use of ultrasound to rapidly evaluate for cardiac tamponade, intraperitoneal blood, and abdominal aortic aneurysm.[79]

▶ ESTIMATION OF CENTRAL VENOUS PRESSURE

Assessment of the size and respiratory variations of the proximal IVC can provide information about central venous pressure (CVP) and fluid status. The IVC can dilate or collapse depending on intraluminal pressure. In general, a large IVC correlates with higher CVP and a small IVC correlates with a lower CVP. There is usually significant respiratory variation in the size of the IVC because inspiration produces negative intrathoracic pressure and draws blood out of the IVC and into the right atrium, causing the IVC to collapse. The reverse occurs during expiration and the IVC expands. Several investigators have attempted to correlate IVC findings with CVP values, which is difficult since absolute IVC size varies between patients and the IVC can completely collapse with inspiration in a normovolemic patient. Although IVC measurements cannot accurately measure CVP values, they can be used to effectively estimate whether CVP is very low or very high.

The proximal IVC is found in the subxiphoid view as it courses posterior to the liver and into the right atrium. It is usually measured about 3–4 cm distal to its junction with the atrium or 2-cm distal to the entry to the hepatic viens.[80–82] It is measured in the AP diameter in the sagittal plane.[80] One study measured the IVC in 27 patients who had pulmonary artery catheters in-place with right atrial ports and found that in general patients with high right atrial pressure had a larger maximal IVC. There was a stronger correlation between inspiratory collapse of the IVC and CVP. Specifically, the CVP was nearly always >10 mmHg if the IVC did not collapse more than 50% with full inspiration. Patients with low CVP had maximal collapse with low inspiratory pressures and those with higher CVP had a gradual collapse of the IVC that was greatest at high inspiratory pressures.[82] Another study found that 86% of patients with CVP <10 mmHg had IVC collapse >50%, and 89% with CVP ≥10 mmHg had IVC collapse ≤50% with inspiration.[83]

One study found that nearly all trauma patients who presented in shock or later required blood transfusion had an IVC ≤9 mm and those who were stable had IVC >9 mm. The group who presented in shock had a mean hemoglobin level of 9.1 g/dL while the control group had a mean hemoglobin level of 11.3 g/dL.[84] These findings suggested that measurements of IVC may be useful in identifying patients who are in early shock before they develop classic signs of shock. The data also suggested that serial IVC measurements may be useful for monitoring patients with known or suspected blood loss or any other process that may lead to shock.

From a practical standpoint, the initial size and respiratory variation of the IVC is not as helpful (except when at the extremes) as the changes that occur in these parameters in response to a fluid challenge. When a patient presents with undifferentiated hypotension, the initial treatment often involves a fluid challenge. Serial monitoring of the IVC can help the clinician evaluate the effect of this treatment more accurately. In general, if the patient's hemodynamics improve and the IVC changes little on ultrasound, more fluid can be given. When the IVC measurements indicate rapidly increasing fluid pressures, further fluid administration should be limited.

▶ EXTERNAL CARDIAC PACING

Transcutaneous cardiac pacing is a common treatment for hemodynamically unstable bradycardias. Application and use of external pacing devices is simple but assessment of mechanical ventricular capture during pacing can be confusing. If the pacing unit is used for electrocardiographic monitoring, then a pacing filter may allow correct assessment of electrical capture. Even with a filter pacer spikes can drown out the native QRS complexes and give a false impression that there is electrical capture when there is none. An alternative means of assessing the capture is to feel the patients pulse and confirm that pulses correspond to pacer output. However, this can also be difficult especially if pacing causes significant skeletal muscle contractions.[85]

The use of bedside echocardiography to assess pacemaker capture is straightforward and was described in the earliest report of emergency department bedside echocardiography.[4] The utility of echocardiography for determining the capture during external pacing was studied using an animal model and showed that blinded interpreters could identify ventricular capture with good agreement.[86]

Bedside echocardiography can also be used to help guide placement of a temporary transvenous pacemaker wire. Emergency physicians have used echocardiography guidance to correctly place pacer wires into the apex of the right ventricle and confirm ventricular capture with excellent success.[87]

▶ VALVULAR ABNORMALITIES

Valvular dysfunction and structural abnormalities have not traditionally been a focal part of the emergent echocardiography examination. In select cases of acute hemodynamic compromise, detection of significant valvular abnormalities by echocardiography may be lifesaving. Since the mitral valve is the most straightforward valve to visualize by echocardiography, severe abnormalities of this valve may be apparent. Acute rupture of chordae tendineae or papillary muscles can be seen as a result of myocardial infarction or infective endocarditis.[88] Rupture is more likely to result from an inferior wall myocardial infarction with involvement of the right coronary artery. Acute mitral valve incompetence causes dyspnea, pulmonary edema, and cardiogenic shock. Rupture of the entire papillary muscle usually results in acute severe mitral regurgitation.[89] This process may be suspected in patients presenting with new onset severe pulmonary edema. Rapid diagnosis and emergency surgery is the key to survival for these patients.

Both leaflets of the mitral valve are usually clearly visualized if a good parasternal long axis view can be obtained. In this view, rupture of one of the mitral valve leaflets will usually show a clear flail leaflet if the entire papillary muscle is ruptured. Alternatively, a four-chamber apical view will clearly show both mitral valve leaflets. Color Doppler will demonstrate regurgitant flow in an eccentric distribution opposite in direction to the leaflet with the anatomic defect.[89]

Aortic stenosis is typically seen in the elderly and is caused by degenerative heart disease or calcific aortic stenosis. Critical aortic stenosis can cause angina, syncope, and eventually heart failure. Sudden death from dysrhythmias occurs in 25% of such patients. Patients who present with congestive heart failure should be treated with oxygen and diuretics but nitrates are not well tolerated in patients with severe aortic stenosis as decreasing preload may result in significant hypotension.[90] Recognizing aortic disease on bedside echocardiography may not be as straightforward as detecting mitral abnormalities because movements of the aortic valve leaflets are much more difficult to visualize. Most patients with aortic stenosis will have significant calcifications and thickening of the aortic valve leaflets that may be visualized by echocardiography.

Aortic valve incompetence is an acute process in about 20% of cases and most commonly caused by infective endocarditis or proximal aortic dissection. In acute aortic valve incompetence, left ventricular failure develops rapidly and mortality from pulmonary edema and cardiac arrest is high, even with intensive medical therapy. The key to survival for these patients is rapid recognition of their condition and emergency surgery. Detecting aortic incompetence will require utilization of color Doppler flow across the aortic valve. Significant incompetence should be apparent with subjective visualization of color Doppler.

▶ AORTIC DISSECTION

Aortic dissection occurs when the intima is violated, allowing blood to enter the media and dissect between the intimal and adventitial layers. Common sites for tear include the ascending aorta and the region of the ligamentum arteriosum. The Stanford classification categorizes aortic dissection as Type A, which involves the ascending aorta; and Type B, which involves only the descending aorta.

Aortic dissection and intramural hematoma may be detected by transthoracic echocardiography on parasternal long, parasternal short, and suprasternal views. A linear echogenic flap, indicative of aortic dissection, may be seen across the aortic lumen anywhere along its length. The ascending aorta can be seen on the parasternal views while the descending aorta is usually seen only in cross section on parasternal views. The aortic arch may be visualized on the suprasternal view in a segment of the population.[28–31] A dissection that extends beyond the thoracic aorta may be detected with abdominal sonography.

TEE provides much better resolution and visualization of aortic dissection than transthoracic echocardiography. Spiral CT, TEE, and magnetic resonance imaging (MRI) have been demonstrated to have comparable sensitivity, and specificity, with accuracy rates approaching 100%.[28–31] One study found that all three modalities approach 100% sensitivity; the specificities for spiral CT, TEE, and MRI were 100, 94, and 94%, respectively.[91]

Important issues to address with aortic dissection include (1) presence of pericardial effusion as a sign of imminent mortality without surgical intervention; (2) presence of ascending aorta involvement without pericardial involvement; (3) evidence of isolated descending

aorta involvement; (4) location of the entry site; and (5) evidence of involvement of major branch vessels.[28–31]

▸ MYOCARDIAL ISCHEMIA

The diagnosis of acute myocardia ischemia can be made by echocardiographic findings of wall motion abnormalities. Myocardial function is immediately affected by ischemia and may precede ECG changes. New regional wall motion abnormalities, however, may be difficult to differentiate from old wall motion changes without reviewing prior echocardiograms. Studies have demonstrated that the recognition of regional wall motion abnormalities by echocardiography in patients with acute chest pain is a sensitive predictor for Q-wave myocardial infarction. Sensitivity for acute myocardial infarction generally is high, but specificity remains moderate because of old wall motion abnormalities.[92,93] Resting echocardiography in patients with acute chest pain is not sufficiently sensitive for exclusion of cardiac ischemia.[94,95] In the emergency department, however, the combination of resting echocardiography and cardiac enzyme serum markers may be a promising combination for the stratification of patients at risk for complications within the hospital and on discharge.[96] The identification of echocardiographic abnormalities may expedite admission for stable patients who are being evaluated in an emergency department.

Echocardiography plays an important role in the noninvasive evaluation of patients with known myocardial infarction, including evaluation of its complications such as left ventricular systolic dysfunction, development of ventricular septal defects, left ventricular rupture, and mitral regurgitation.[97] In fact, one study on emergency department patients reported six cases of myocardial rupture with hemopericardium from acute myocardial infarction with early ventricular rupture. As four of these patients met the criteria for emergency thrombolytic therapy, the emergency management was significantly changed by these findings on the focused echocardiogram. [18] Chronic complications such as pericarditis, pericardial effusion, left ventricular aneurysm, and left ventricular thrombus may also be evaluated by echocardiography.

▸ ANATOMIC CONSIDERATIONS

HEART

The heart is a hollow muscular organ, placed between the lungs, and enclosed within the pericardium. It is divided by a septum into two halves, right and left, each half being further subdivided into two cavities, the atrium and the ventricle (Figure 6-1). Blood flows from the right atrium into the right ventricle through the tricuspid valve. From the right ventricle, unoxygenated blood is carried to the lungs through the pulmonary artery. Blood flows from the left atrium into the left ventricle through the mitral valve. From the left ventricle, oxygenated blood is distributed to the body through the aorta. The outflow tract through the aortic valve into the proximal aorta starts anteriorly and revolves posteriorly into the posterior mediastinum next to the esophagus. The aorta is divided into the ascending aorta, the aortic arch, and the descending aorta. The ascending aorta is approximately 5 cm in length; the only branches of the ascending aorta are the coronary arteries. The aortic arch has three branches: the innominate artery, the left common carotid artery, and the left subclavian artery.

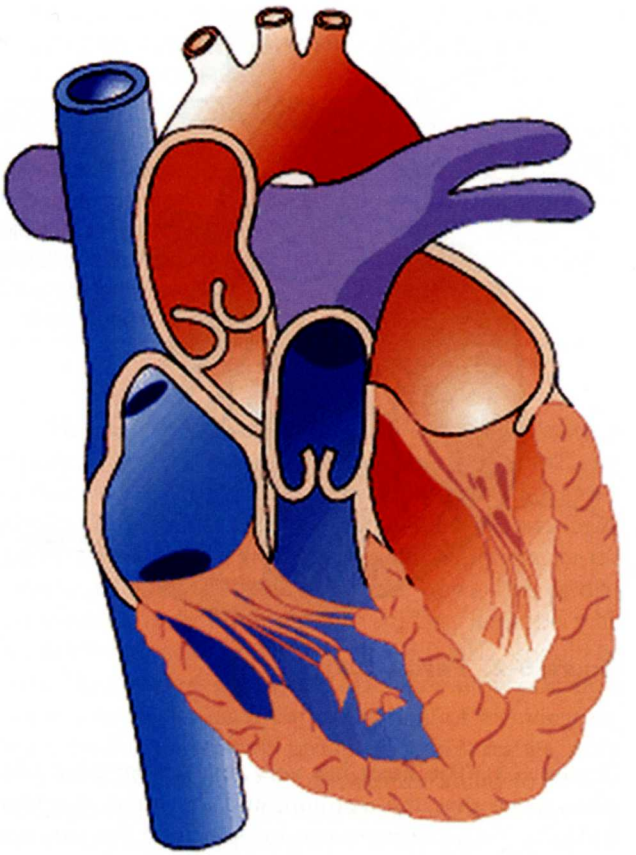


Figure 6-1. Cardiac circulation.

The pericardium is composed of two layers, the parietal and visceral layers, which normally oppose each other without any significant fluid accumulation. The pericardium attaches to the superior left atrium and envelopes the proximal aspects of the great vessels (Figure 6-2).

THORACIC CAVITY

The thoracic cavity provides both windows and impediments to the accurate sonographic view of the heart

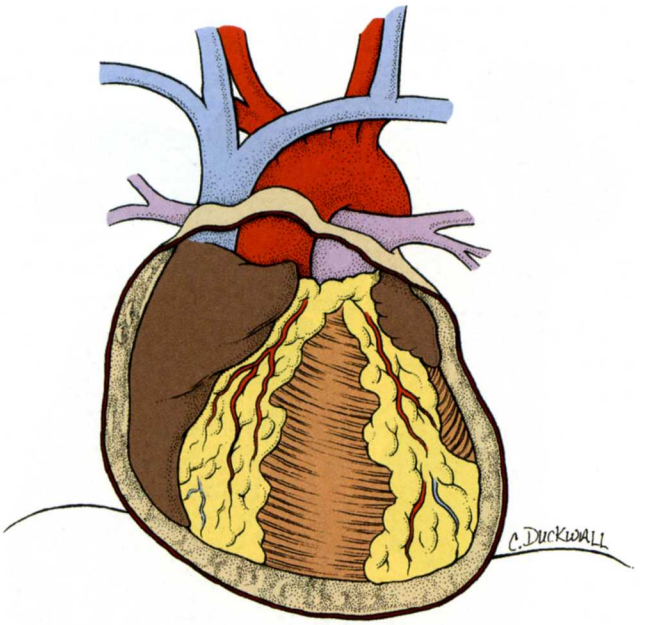


Figure 6-2. Pericardium.

(Figure 6-3). The heart has very few sonographic windows since ribs, the sternum, and the lungs surround it (Figure 6-4). Common windows include the parasternal, apical, subxiphoid, and suprasternal views. The left parasternal interspace allows for small sonographic windows into the mediastinum. The superior aspect of the abdomen also allows for soft tissue windows via the left lobe of the liver. The heart can shift closer to the chest wall in the left decubitus position.

CARDIAC AXES

The heart has a long axis from the right shoulder to the left hip. The transverse view, or short axis of the heart, is rotated 90° from the long axis of the heart (left shoulder to right hip) (Figure 6-5). The apical view, or four-chamber axis, is a coronal image of the heart from its apex to its base. The apex of the heart is usually located at the nipple line in the anterior axillary line. The base of the heart lies in an axis, anterior to posterior, from the parasternal right 2nd intercostal space to the posterior thorax next to the esophagus.

▶ GETTING STARTED

Novice sonologists may find transthoracic echocardiography difficult to learn initially because of the complex anatomy of the heart and difficulty obtaining standard images due to the surrounding air-filled lungs (Figures 6-1 and 6-4). It is important to note that the long axis

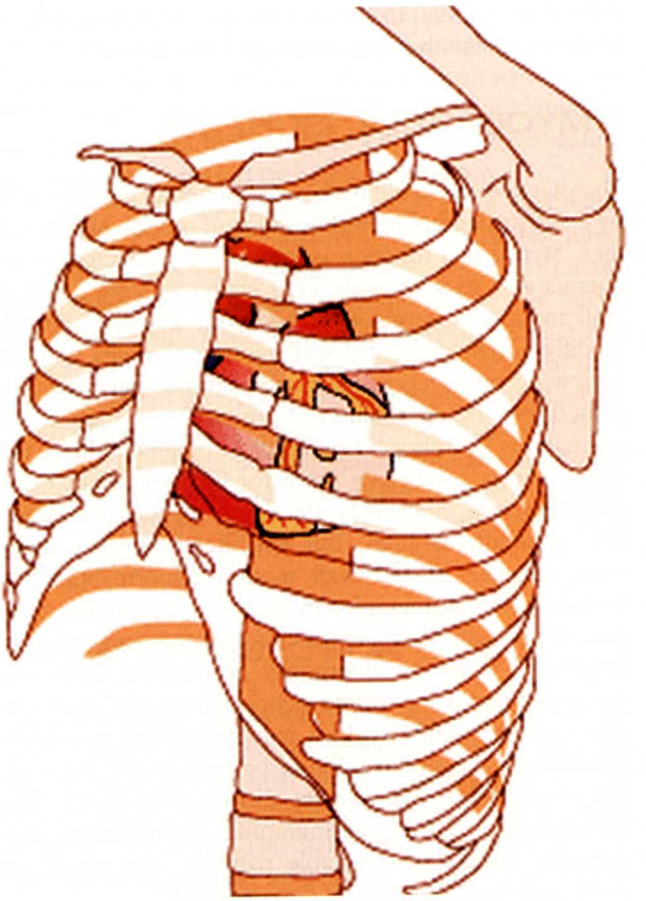


Figure 6-3. Chest cage.

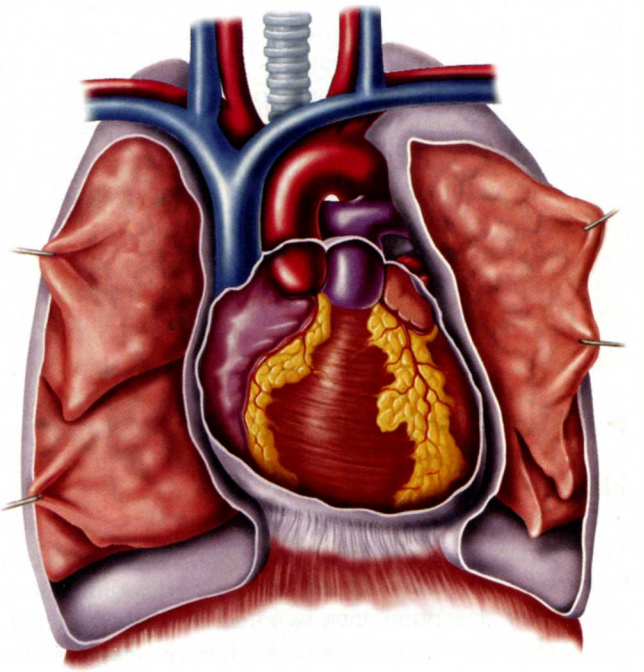


Figure 6-4. Surrounding structures.

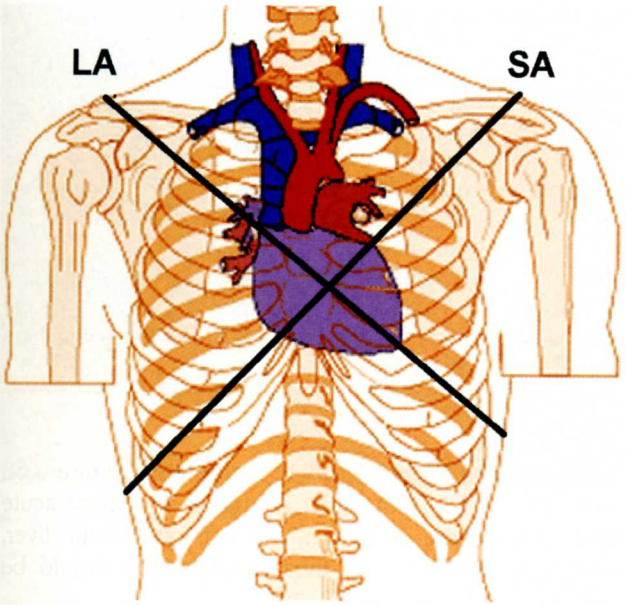


Figure 6-5. Cardiac axes. Long axis (LA), short axis (SA).

of the heart lies diagonal to the long axis of the torso (Figure 6-5). The long axis of the heart is more horizontal in short obese patients and more vertical in tall thin patients. Standard images of the heart are usually obtained from three anatomic locations on and below the chest wall: the subxiphoid window, the parasternal window, and the apical window. The heart lies higher in the chest cavity in obese patients and those who are supine. It lies lower in the chest cavity in thin patients and those who are sitting upright. The apex is found more medially in normal hearts and more laterally in enlarged hearts. Parasternal and apical images may be very difficult to obtain in patients with hyperexpanded lungs.

While most echocardiographers prefer to scan from the patient's left side, many clinicians scan from the patient's right. Cardiac presets should be selected if available on the machine. Patient positioning and ability to cooperate with inspiratory and expiratory maneuvers are critical to obtaining good images. The subxiphoid views are best obtained in the supine position, the parasternal views may be acquired with supine or left lateral decubitus (LLD) positioning and the apical views are usually best obtained in LLD. Windows through intercostal spaces can be improved by positioning the patient's left hand behind their head, which may slightly widen the window. Nearly any change in patient position may improve cardiac windows and the ability to obtain good images. The subxiphoid image is often significantly improved by having the patient take and hold a deep breath, the parasternal and apical views may at times be aided by an expiratory hold.

▶ TABLE 6-2. COMPARISON OF CURVED LINEAR ARRAY WITH PHASED ARRAY TRANSDUCERS

Transducer Type	Curved linear Array	Flat phased Array
Image on monitor	Sector	Sector
Moving structures capture	Poor	Good
Small window capability	Generally worse	Excellent
Gray scale differentiation of echogenic structures	Good	Poor

Having proper equipment and equipment settings is critical to obtaining good images. Using a phased array cardiac probe is better than a curvilinear for echocardiography (Table 6-2). The phased array probe allows imaging between the ribs and is especially important for the parasternal short axis and all apical views. For cardiac transthoracic echocardiographic studies, a probe with median frequencies of 3.5 MHz (2.5–5.0 MHz) should be used. Most modern ultrasound machines allow operators to use presets for each particular type of examination. Using cardiac presets will produce the best results. Also, modern machines give the operator the ability to change the frequency range of the ultrasound probe and activate tissue harmonics with the touch of a button. Testing different frequency ranges and activating tissue harmonics during each examination allow the operator to optimize images.

Overall gain and time-gain compensation are also simple to adjust and can be used to optimize each image. Ideal equipment settings produce images in which the edges of anatomic structures are sharply demarcated and the inside of the cardiac chambers appears black, not grey. It is important for novice sonologists to learn how to obtain images from all three standard anatomic locations and not just the subxiphoid location because on any given patient one location may produce excellent images while the others are less optimal. Every patient has unique anatomy and no one anatomic location allows adequate imaging in all patients.

▶ TECHNIQUE AND NORMAL ULTRASOUND FINDINGS

TRANSTHORACIC ECHOCARDIOGRAPHY

The arrow (right/left indicator) on the monitor is usually oriented to the right side of the image, which is the opposite of abdominal or pelvic imaging and is done automatically via a cardiac preset or manually by toggling the flip image (horizontal) button. The probe indicator, therefore, is oriented to the patient's left for transverse

▶ **TABLE 6-3. TRANSTHORACIC TRANSDUCER ORIENTATION ON THE SUPINE PATIENT**

Ultrasound Preset	Echocardiography	Abdomen/Pelvis
Machine/probe location	To the patient's left	To the patient's right
Monitor indicator	Right side of the image	Left side of the image
Subcostal	Probe marker directed to the patient's left flank	Probe marker directed to the patient's right flank
Apical four-chamber	Probe marker directed to the left side Probe aimed to right shoulder	Probe marker directed to the right side Probe aimed to right shoulder
Parasternal long	Probe marker directed to the patient's right shoulder (10 o'clock)	Probe marker directed to the patient's left hip (4 o'clock)
Parasternal short	Probe marker directed to the patient's left shoulder (2 o'clock)	Probe marker directed to the patient's right hip (8 o'clock)

images. Table 6-3 provides a comparison of imaging techniques for the heart when using an echocardiography preset versus an abdominal/pelvic preset. Regardless of the machine setup, what is most important is to orient the images in a standard fashion on the monitor. This facilitates recognition of the cardiac chambers for the sonologist and for any others who may review the images.

▶ CARDIAC WINDOWS

TRANSTHORACIC

Subxiphoid Four-Chamber View

The subxiphoid view is the most useful view for emergency ultrasound. It usually does not interfere in resuscitative measures such as thoracostomy, CPR, subclavian line insertion, or intubation. It is easily learned, repeated, and performed as part of both the cardiac and trauma ultrasound evaluations.

The subxiphoid view should be performed at the subxiphoid position of the abdomen (Figure 6-6A). The probe should be held at a 15° angle to the chest wall and aimed toward the left shoulder. The probe marker should be aimed toward the patient's left flank (using a cardiac preset). The transducer should be angled up or down depending on the depth of the chest cavity to obtain images of the beating heart. The depth should then be adjusted to visualize the atria at the bottom of the monitor screen. Poor quality initial images may be improved upon by using an appropriate amount of ultrasound gel, using a shallow angle to the chest wall, moving the transducer to the right to use the left lobe of the liver as a window, and moving off the xiphoid and over to the lower intercostal spaces to image the barrel-chested patient with a larger anterior–posterior diameter.

The subxiphoid four-chamber view should be seen as primarily a diagonal view for the ventricles, atria, pericardium, and the left lobe of the liver (Figure 6-6B and 6-6C). If the transducer is angled at a more acute angle toward the abdomen, the left lobe of the liver, inferior vena cava, and the hepatic veins should be visualized.

Subxiphoid Short-Axis View

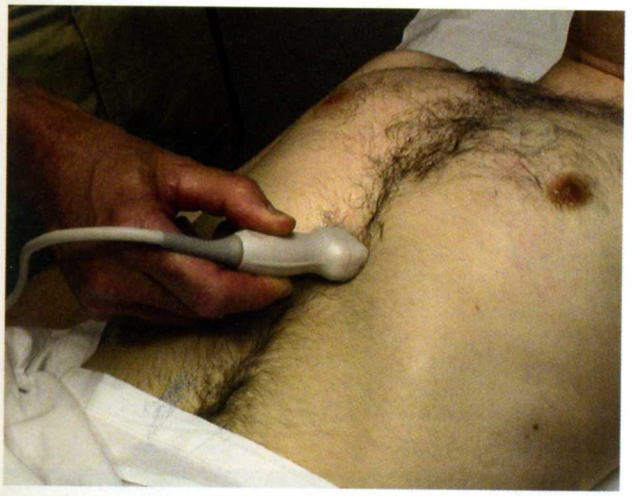
The subxiphoid short axis view can be achieved by rotating the ultrasound probe 90° counterclockwise from the four-chamber view (using a cardiac preset) and aiming the probe toward the patient's left arm (Figure 6-7A). This orientation will resemble the parasternal short axis ("doughnut") view of the left ventricle and may provide virtually all of the same information (Figure 6-7B and 6-7C).

Subxiphoid Long-Axis View

The subxiphoid long axis view uses a sagittal body axis and the probe marker can be aimed toward the patient's feet (using a cardiac preset) (Figure 6-8A). This will place the atrium/diaphragm on the left side of the screen as is the standard for abdominal longitudinal imaging. Echocardiographers (who are envisioning the longitudinal view from the patient's left side) often orient this image with the probe marker pointing cephalad and the atrium/diaphragm on the right side of the screen. A sagittal section of the body views the heart, the left lobe of the liver, the IVC, and hepatic veins (Figure 6-8B). This view allows evaluation of the proximal IVC during expiration and inspiration (Figure 6-8C and 6-8D). The anterior–posterior diameter of the proximal IVC usually measures about 1.5–2.0 cm during expiration and collapses with inspiration. Collapse of less than 50% during inspiration indicates elevated right-sided heart pressures.

Parasternal Long-Axis View

The parasternal long axis view can be best obtained by accepting the long axis of the heart to be roughly from the right shoulder to the left hip (Figure 6-5). The



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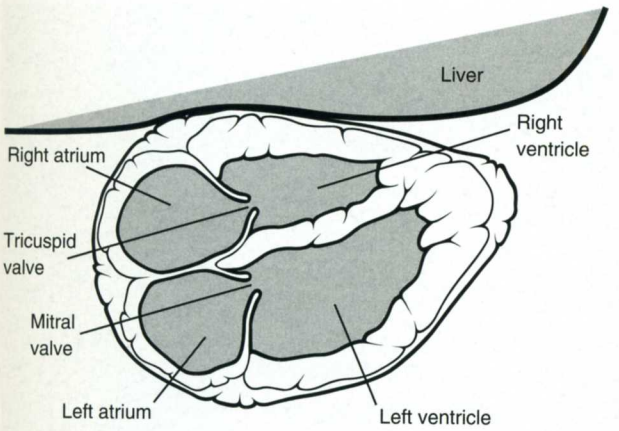

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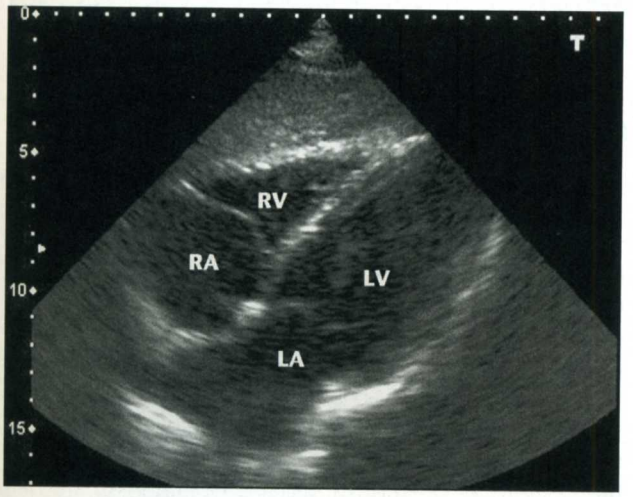
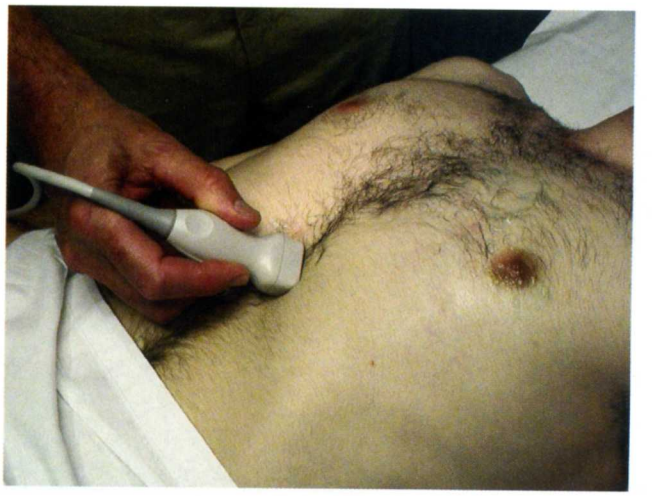


C

Figure 6-6. Probe position for subcostal 4 chamber view (A). Subcostal four-chamber diagram (B). Subcostal four-chamber normal ultrasound (C). RA = right atrium, RV = right ventricle, LA = left atrium, LV = left ventricle. (Courtesy of Hennepin County Medical Center for (C))



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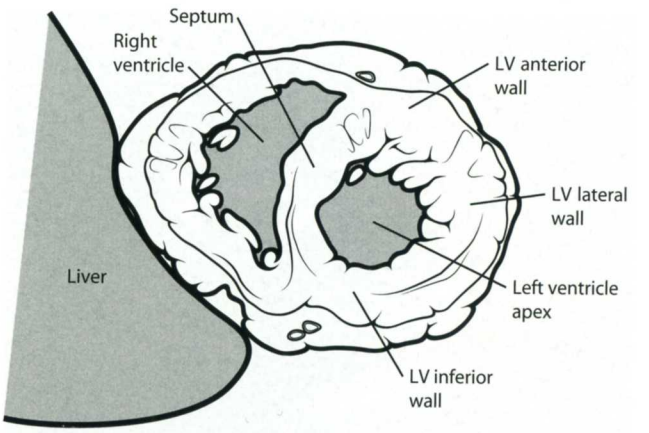

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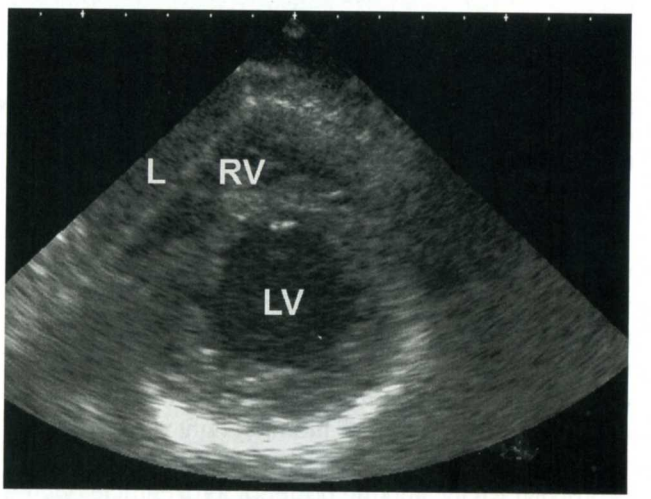
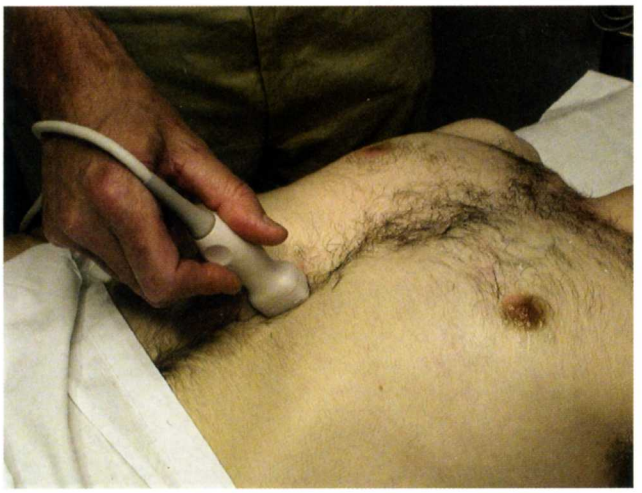


C

Figure 6-7. Probe position for subcostal short axis view (A). Subcostal short axis diagram (B). Subcostal short axis normal ultrasound (C). L = liver, RV = right ventricle, LV = left ventricle. (Courtesy of James Mateer, MD, for (C))



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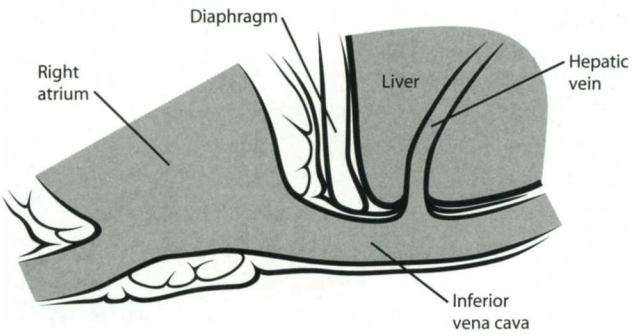

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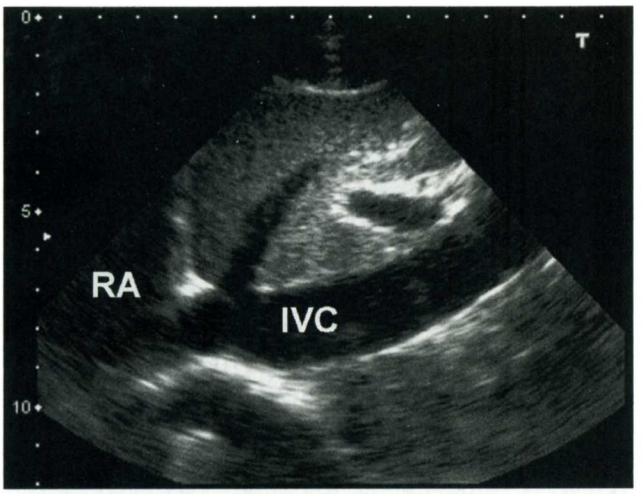

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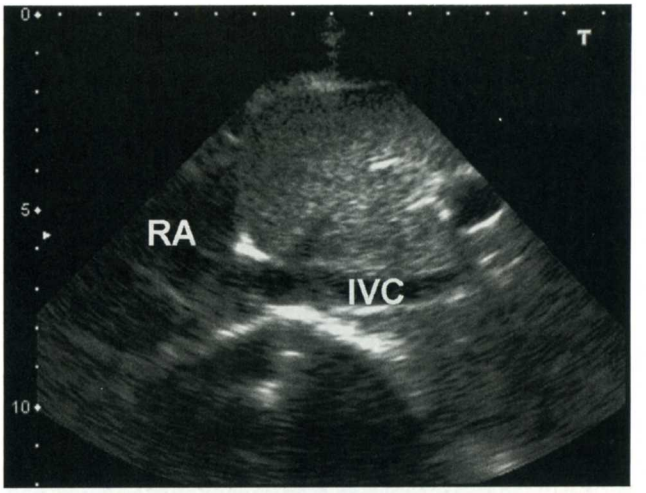


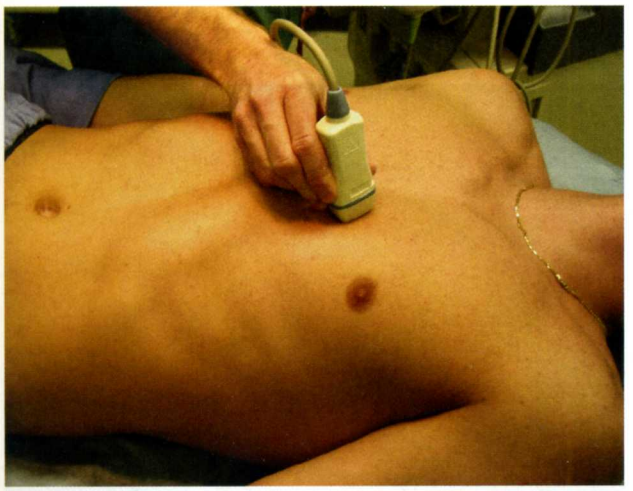
D

Figure 6-8. Probe position for subcostal long axis view (A). Subcostal long axis diagram (B). Proximal IVC during expiration (C) and inspiration (D). IVC = inferior vena cava, RA = right atrium. (Courtesy of Hennepin County Medical Center for (C,D))

transducer should be placed perpendicular to the chest wall at the 3rd or 4th intercostal space immediately to the left of the sternum with the probe indicator directed toward the right shoulder (using a cardiac preset) (Figure 6-9A). The following structures can be visualized from anterior to posterior on the monitor: right ventricular free wall, right ventricular cavity, interventricular septum, left ventricular cavity, and the posterior left ventricle (Figure 6-9B). On the basal side of the image, the aortic valve with its inflow and outflow tracts, the mitral valve with its inflow and outflow tracts, the left atrium, the posterior pericardium, and possibly the descending aorta should be seen (Figure 6-9C). The probe should be rotated to obtain the best axis to view these structures. Angling and tilting may be needed, but less so than for the short axis view. A reduction in size of the image may be needed to focus on certain structures or enlargement of the field of view to see the entire left ventricle and left atrium.

Parasternal Short-Axis View

The imaging plane for the parasternal short axis view of the heart stretches from the left shoulder to the right hip (Figure 6-5), and should be obtained in the left 3rd or 4th intercostal space next to the sternum (Figure 6-10A). If the parasternal long axis view has already been obtained, the parasternal short axis views should be obtained by rotating the probe marker 90° clockwise toward the left shoulder (using a cardiac preset). With the probe in this position, several different short axis views can be obtained by sweeping the image from base to apex (Figure 6-10B). Parasternal short axis views can be



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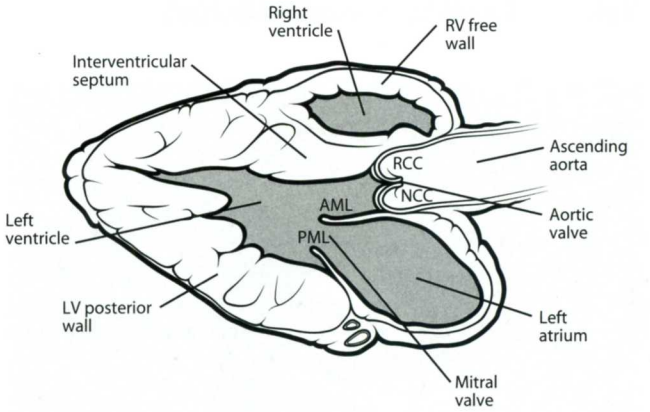

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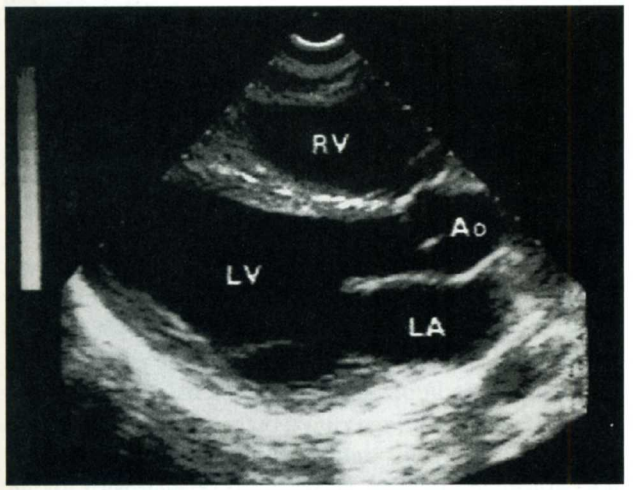
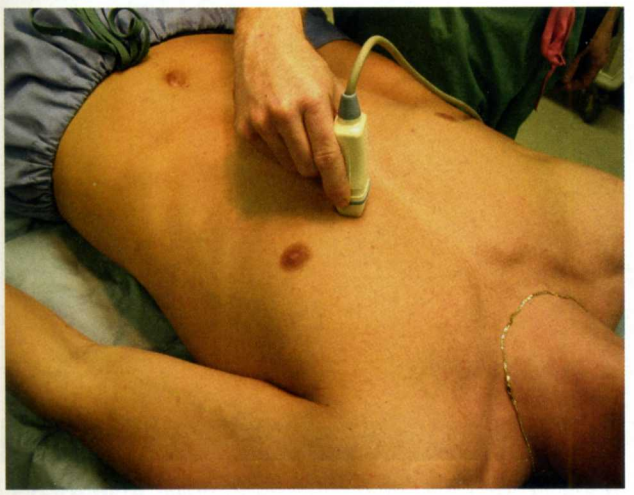


C

Figure 6-9. Probe position for parasternal long-axis view (A). Note: May require left lateral decubitus position. Parasternal long-axis diagram (B). Parasternal long-axis normal ultrasound (C). RV = right ventricle, Ao - aorta, LV = left ventricle, LA = left atrium.



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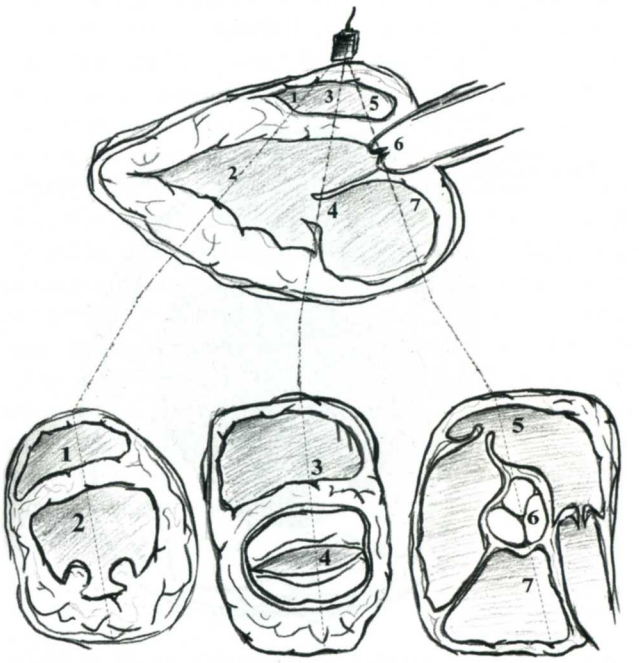

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Figure 6-10. Probe position for parasternal short-axis view (A). Note: May require left lateral decubitus position. Diagram of short axis views from apex to base (B).

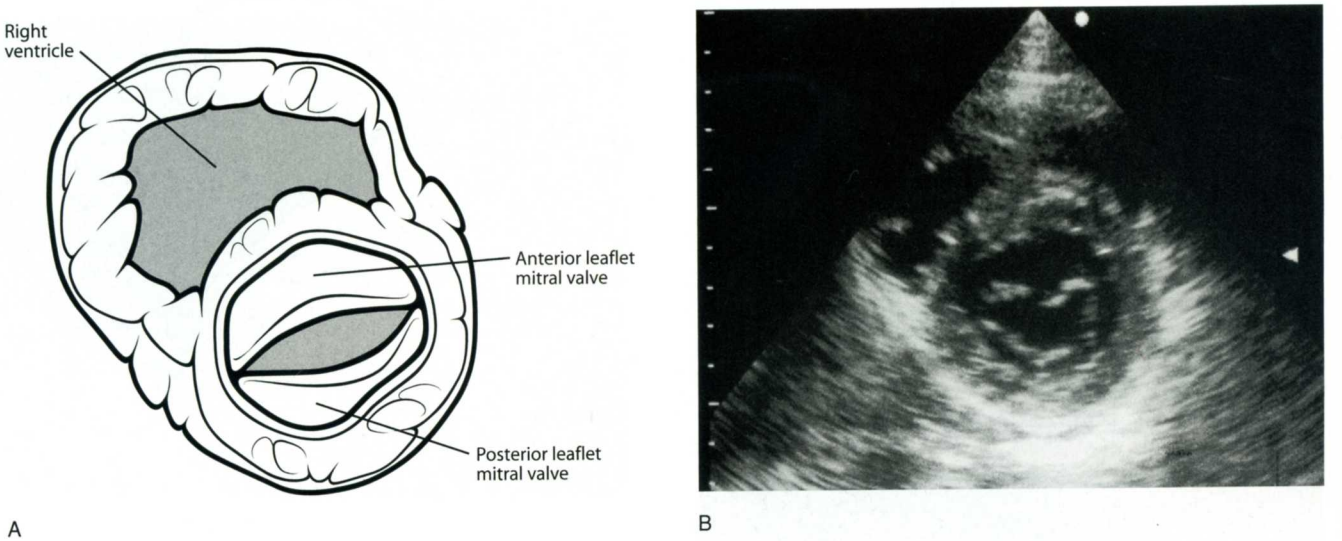


Figure 6-11. Parasternal short-axis diagram at mitral valve (A). Parasternal short-axis normal ultrasound at mitral valve (B).

obtained at the base of the heart, the level of the mitral valve (Figure 6-11A and 6-11B) the level of the papillary muscles, and at the apex. The short axis view at the level of the papillary muscles is an important view because it allows identification of the different walls of the left ventricle (Figure 6-12A and 6-12B). An ideal short axis view at the base of the heart (Figure 6-13A and 6-13B) visualizes the left atrium, right atrium, tricuspid valve, right ventricle, and pulmonary valve encircling the aortic valve ("Mercedes Benz sign") in cross section in the middle of the view (Figure 6-14).

Apical Four-Chamber View

The apical 4-chamber view is a coronal view of the heart that images all four chambers in one plane.

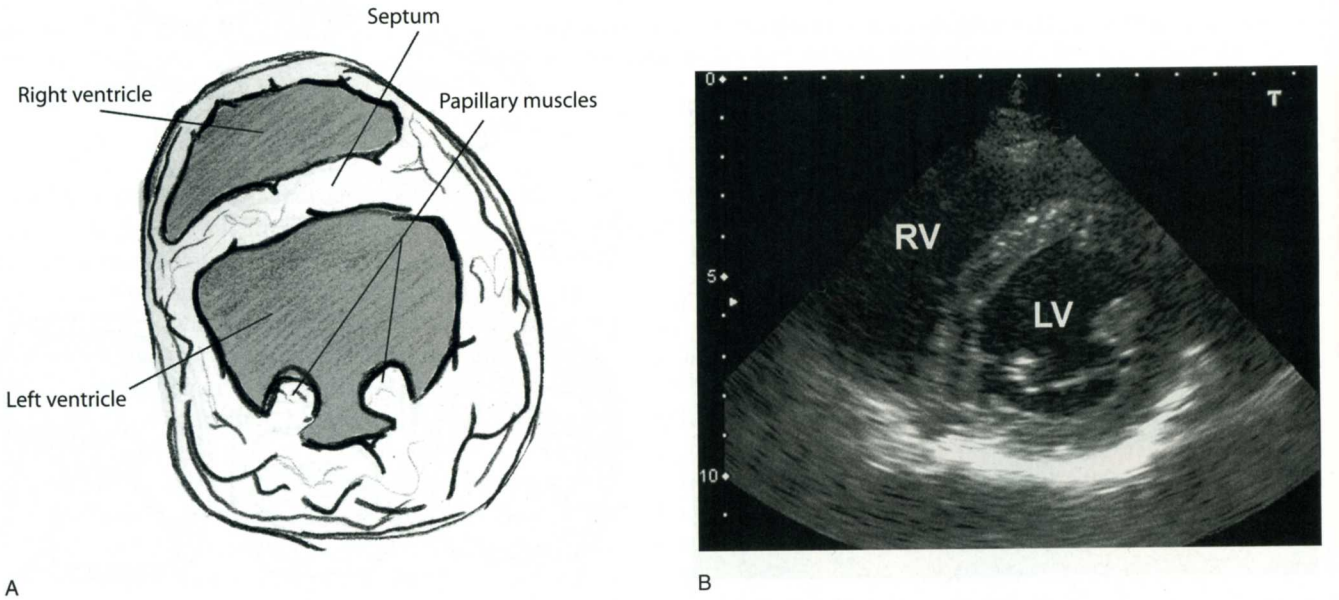


Figure 6-12. Parasternal short axis diagram at papillary muscles (A). Parasternal short axis normal ultrasound at papillary muscles (B). RV = right ventricle, LV = left ventricle. (B: Courtesy of Hennepin County Medical Center)

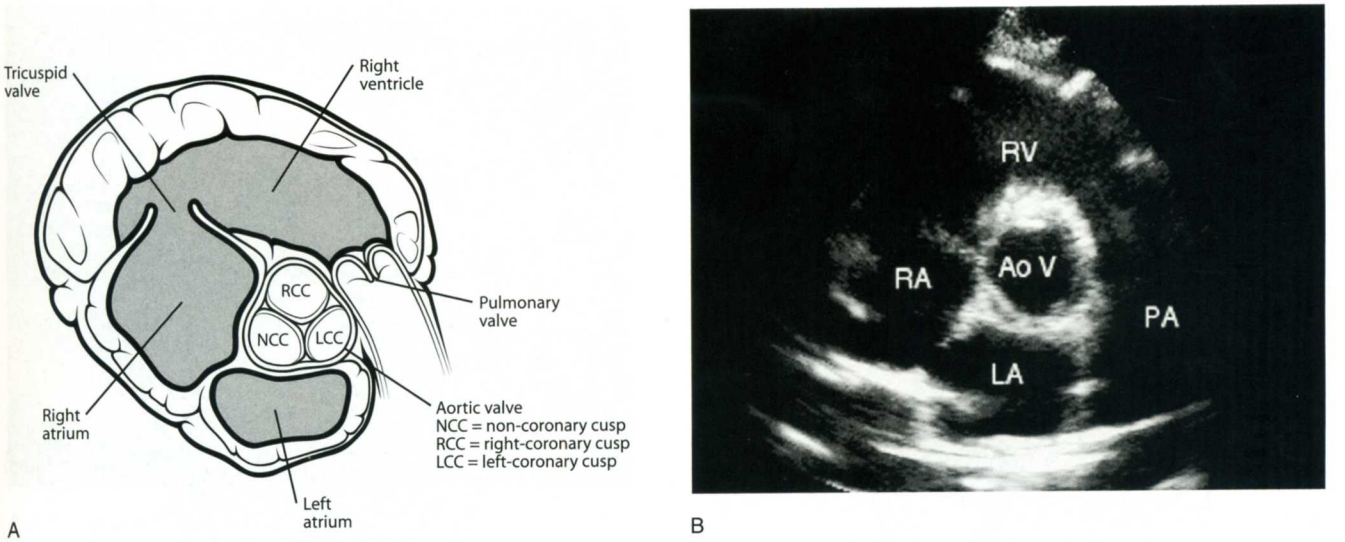


Figure 6-13. Parasternal short axis diagram at aortic valve (A). Parasternal short axis normal ultrasound at aortic valve (B). RV = right ventricular outflow tract, RA = right atrium, LA = left atrium, PA = pulmonary artery, Ao V = aortic valve.

Alterations of this view include the apical two-chamber view, the apical three-chamber view, and the apical five-chamber view. Regardless of the number of chambers, the view is best observed by obtaining the window at the apex of the heart, usually where the point of maximal impulse (PMI) for the heart is located (Figure 6-15A). Alteration of this position may be required to adjust for breast tissue, emphysema, chest deformities, and other anatomic changes. Whenever possible, the patient should be rotated toward their left side to reduce lung artifact and bring the heart closer to the chest wall. The transducer should be placed at this position, generally in the 5th intercostal space or lower, and aimed toward the right shoulder with the marker directed toward the left lateral chest wall (using a cardiac preset). Some rotation may be needed to allow for all four chambers to be viewed. A rounded, foreshortened heart is usually artifactual. To correct this, the transducer should be aimed in a more anterior direction and/or a lower rib interspace should be used. On this view, the right ventricle with its lateral wall, the interventricular septum (septal wall), the left ventricle with the lateral wall, the two atria, the interatrial septum, and the pulmonary veins should be visualized (Figure 6-15B and 6-15C). This view is advantageous for assessing right ventricular function and the left ventricle for function and presence of blood clots. Doppler studies are often best obtained with apical views as blood flow is parallel to the transducer with this position. Intra-atrial abnormalities, such as myxoma, may be well visualized. When the image sector is swept more anteriorly from the four-chamber view, the left ventricular outflow and aortic valve come into view (five-chamber).

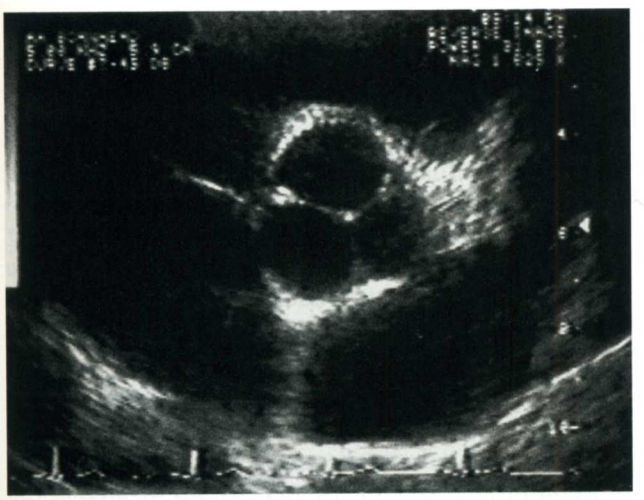
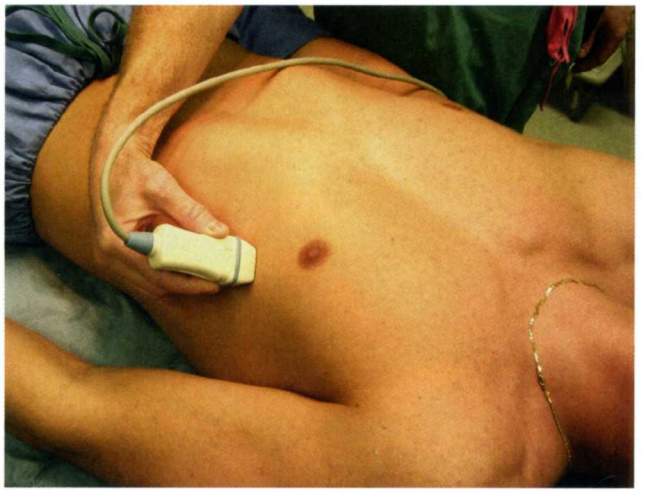


Figure 6-14. Mercedes Benz sign. Parasternal short axis at aortic valve demonstrates closure of all 3 cusps. (Courtesy of Lori Sens and Lori Green, Gulfcoast Ultrasound)

Apical Two-Chamber View

For the apical two-chamber view, the ultrasound probe should be placed in the same orientation as for the apical



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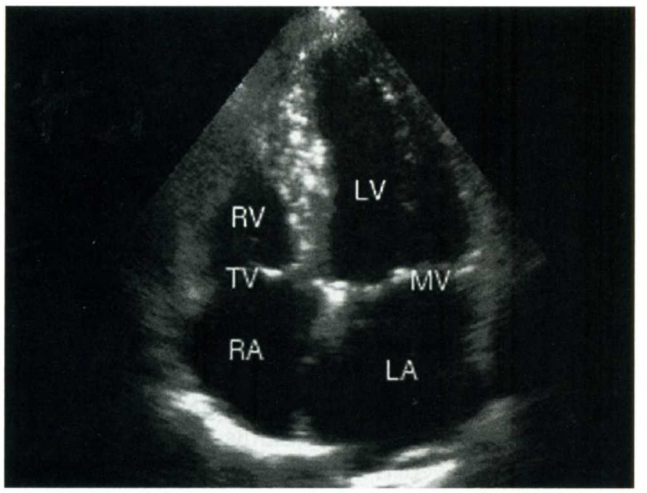

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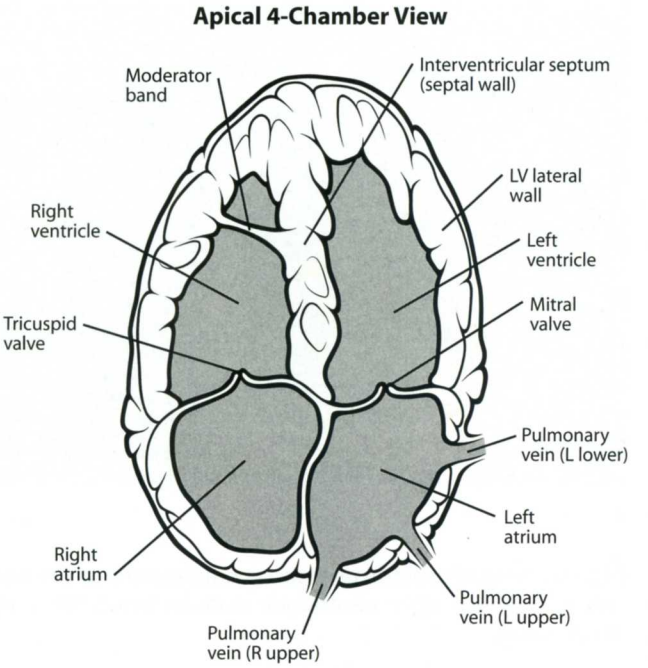


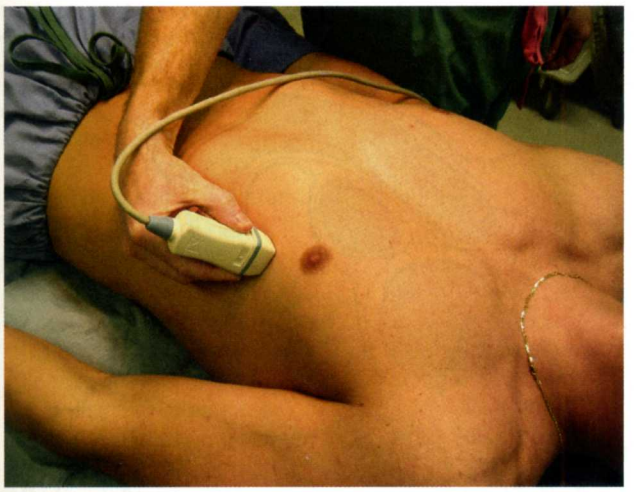
B

Figure 6-15. Probe position for apical 4 chamber view (A). Note: May require left lateral decubitus position with left arm elevated. Apical 4 chamber diagram (B). Apical 4 chamber normal ultrasound (C). RV = right ventricle, LV = left ventricle, MV = mitral valve, LA = left atrium, RA = right atrium, TV = tricuspid valve.

four-chamber view but the transducer should be rotated 90° counterclockwise (using a cardiac preset) until the marker is directed toward the left mid clavicle or head of the patient (Figure 6-16A). This view evaluates anterior and inferior walls, thus complementing the apical four-chamber view of the left ventricle for wall motion and function (Figure 6-16B and 6-16C). Further counterclockwise probe rotation from the two-chamber view (additional 30°) would create the apical long axis (three-chamber) view.

Suprasternal View

The suprasternal view provides a glimpse of the aortic arch with its three main branches: the brachiocephalic artery, the left carotid artery, and the left subclavian



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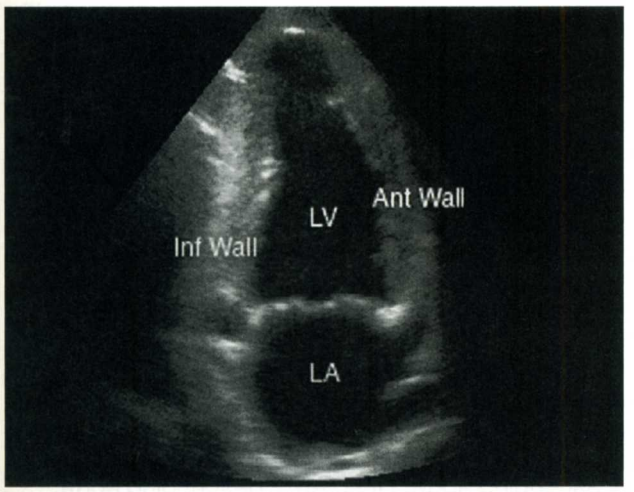

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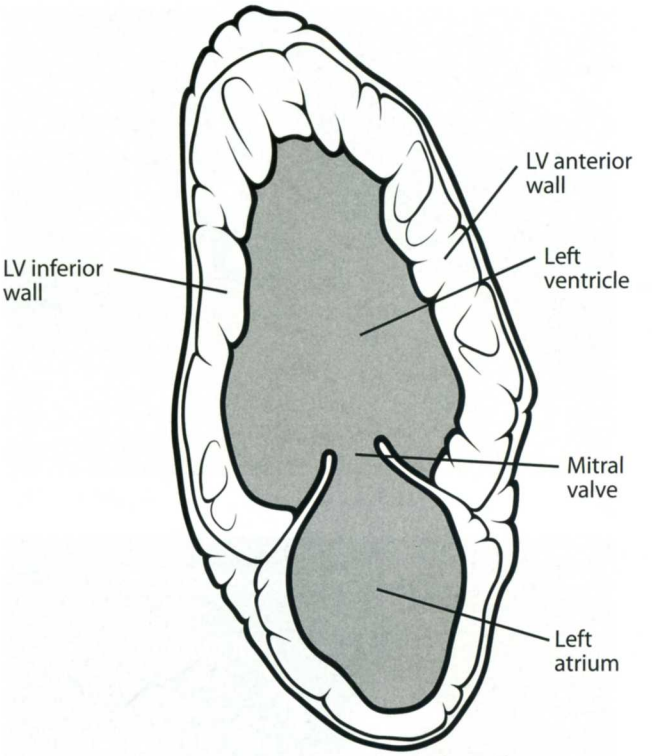


B

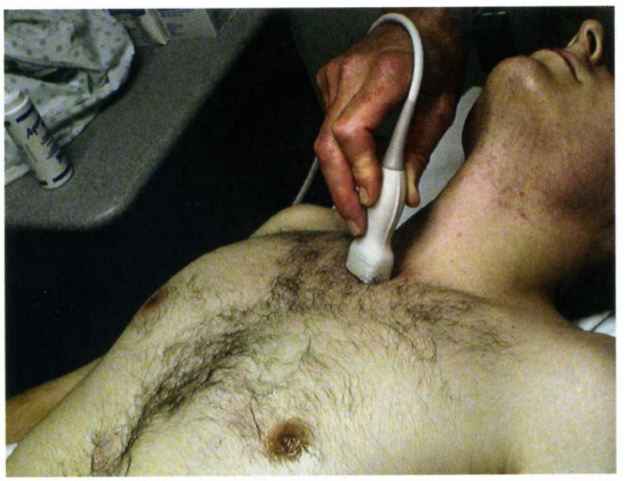
Figure 6-16. Probe position for apical two-chamber view (A). Note: May require left lateral decubitus position with left arm elevated. Apical two-chamber diagram (B). Apical two-chamber normal ultrasound (C). LV = left ventricle, LA = left atrium.

artery. The ultrasound probe should be placed in the sternal notch with the transducer marker pointed toward the patient's left shoulder (using a cardiac preset) and probe aimed as far anteriorly as possible (Figure 6-17A). While it is difficult to obtain in many patients, this view may provide a confirmation of aortic aneurysm or dissection in the patient with an optimal window. The right pulmonary artery in cross section can be viewed below the aortic arch. If the transducer is rotated 90° to visualize the aortic arch in cross section, the left pulmonary artery may be seen. Occasionally, the superior vena cava may be viewed lateral to the ascending aorta. The left atrium lies inferior to the pulmonary arteries and, in an optimal window, all four pulmonary veins may be viewed (Figure 6-17B and 6-17C).

▶ MEASUREMENTS

TWO-DIMENSIONAL MEASUREMENT

Chamber dimensions and sizes should be measured at right angles to the long axis of the respective chamber. Measurement of the chamber sizes, wall thickness, and the left ventricular function may be helpful. By measuring the left ventricular dimensions in systole and diastole, one can calculate the ejection fraction manually or by using the ultrasound machine calculation package. Critical to the 2D measurement is the ability to visualize the endocardium and a cine memory in order to scroll to the correct point in the cardiac cycle for measurement. Table 6-4 lists linear dimensions of normal cardiac structures.



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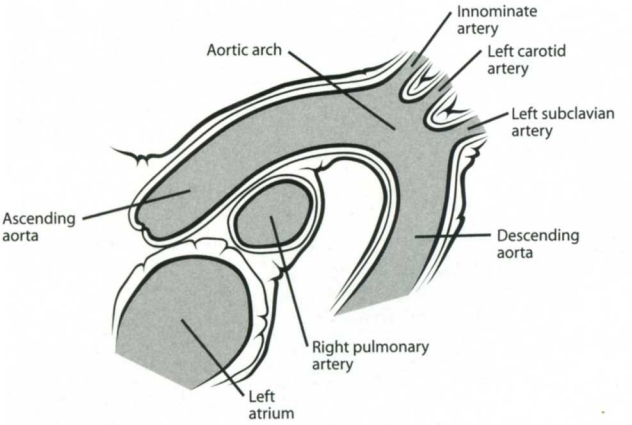

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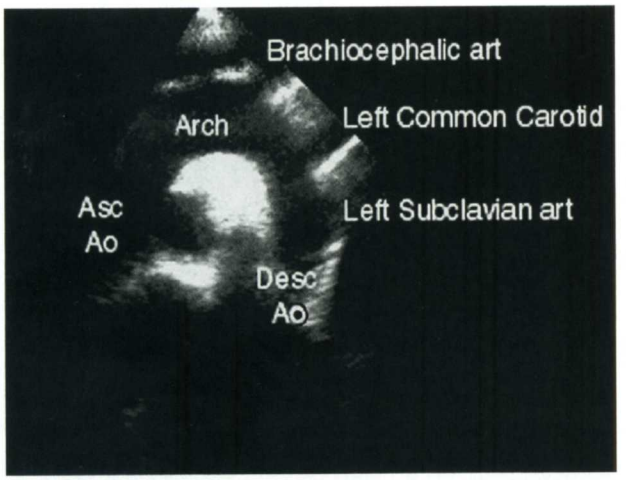


C

Figure 6-17. Probe position for suprasternal view (A). Suprasternal diagram (B). Suprasternal normal ultrasound (C). The branch arteries may be closely approximated as in the diagram or spread apart for some patients as in the ultrasound example. Asc Ao = ascending aorta, Desc Ao = descending aorta.

LEFT VENTRICULAR FUNCTION MEASUREMENT

Several methods exist for the echocardiographic measurement of ejection fraction. These range from observation and fairly simple M-mode measurements to complicated biplane calculations.[98] Many ultrasound software packages have the ability to estimate volume and calculate ejection fraction. This is typically accomplished by border tracing and measurement of cavity length while scanning in an apical four-chamber or apical two-chamber views.

While it may be satisfying to actually calculate a value for ejection fraction, it has been shown that visual estimation of ejection fraction is as good or better than calculated ejection fraction and is much easier to perform.[98–100] This is the most frequent method in clinical use today. With training and practice, noncardiologists may be able to estimate ejection fraction with reasonable accuracy.[15,19] It is important to note that both estimated and calculated ejection fractions are more accurate when the ventricle is regularly shaped and contracts in a symmetric fashion. Wall motion abnormalities and other variations in shape may significantly confound the echocardiographic measurement of ejection fraction.

For those who are more comfortable with a numerical measurement to determine left ventricular function, the EPPS can be determined from the cine review of the mitral valve on a parasternal long axis view. For more discussion, see below: M-Mode Mitral Valve and Left Ventricular Dysfunction.

CENTRAL VENOUS PRESSURE

Right atrial pressures can be estimated by viewing the respiratory change in the diameter of the IVC. Table 6-5 shows common measurements of IVC diameter and change with respiration used to estimate the right atrial pressure. This amounts to a noninvasive CVP measurement and may be helpful in patients with hypotension and uncertain volume status. It is also possible to estimate the end-systolic pulmonary artery pressure (approximates the wedge pressure) by measuring the flow velocity of the tricuspid regurgitant jet and applying it to

▶ TABLE 6-4. NORMAL FINDINGS ON TWO-DIMENSIONAL ECHOCARDIOGRAPHY.

	Range	Range Indexed to BSA	Upper Limit of Normal
Aorta			
Annulus diameter	1.4–2.6 cm	1.3 ± 0.1 cm/m^2	<1.6 cm/m^2
Diameter at leaflet tips	2.2–3.6 cm	1.7 ± 0.2 cm/m^2	<2.1 cm/m^2
Ascending aorta diameter	2.1–3.4 cm	1.5 ± 0.2 cm/m^2	
Arch diameter	2.0–3.6 cm		
Left ventricle			
Short-axis diastole (EDD)	3.5–6.0 cm	2.3–3.1cm/m^2	
Short-axis systole (ESD)	2.1–4.0 cm	1.4–2.1 cm/m^2	
Long-axis diastole	6.3–10.3 cm	4.1–5.7 cm/m^2	
Long-axis systole	4.6–8.4 cm		
End-diastolic volume men	96–157 mL	67 ± 9 mL	
End-diastolic volume women	59–138 mL	61 ± 13 mL	
End-systolic volume men	33–68 mL	27 ± 5mL	
End-systolic volume women	18–65 mL	26 ± 7 mL	
Ejection fraction men	0.59 ± 0.06		
Ejection fraction women	0.59 ± 0.07		
LV – wall thickness end diastole	0.6–1.1 cm		Men ≤1.2 cm Women ≤1.1 cm
LV-mass: men	<294 g	109 ± 20 g/m^2	≤150 g/m^2
LV-mass: women	<194 g	89 ± 15 g/m^2	≤120 g/m^2
Left atrium			
Long-axis AP diameter	2.3–4.5 cm	1.6 –2.4 cm/m^2	
Apical four-chamber Medial–lateral diameter	2.5–4.5 cm	1.6–2.4 cm/m^2	
Apical four-chamber Superior-Inferior diameter	3.4–6.1 cm	2.5–3.5 cm/m^2	
Mitral annulus			
End diastole	2.7 ± 0.4 cm		
End systole	2.9 ± 0.3 cm		
Right ventricle			
Wall thickness	0.2–0.5 cm	0.2 ± 0.05 cm/m^2	
Minor dimension	2.2–4.4 cm	1.0–2.8 cm/m^2	
Length diastole	5.5–9.5 cm	3.8–5.3 cm/m^2	
Length systole	4.2–8.1 cm		
Pulmonary artery			
Annulus diameter	1.0–2.2 cm		
Main pulmonary artery	0.9–2.9 cm		
Inferior vena cava			
At right atrial junction diameter	1.2–2.3 cm		

BSA = body surface area; LV = left ventricle; AP = anterior-posterior.
(Adapted from Otto CM and Pearlman AS: *Normal Cardiac Anatomy in Textbook of Clinical Echocardiography.* Philadelphia, Saunders, 1995; p. 35).

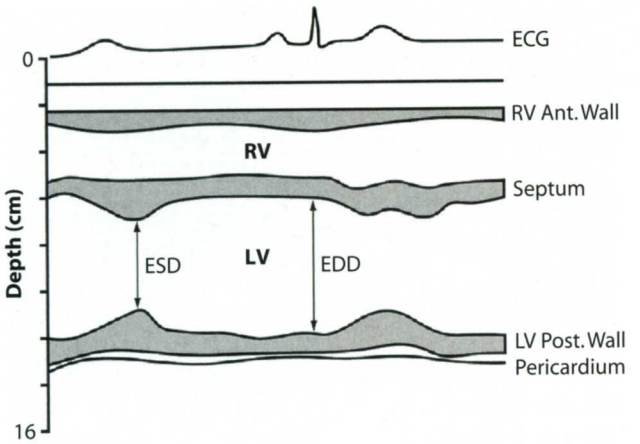
▶ TABLE 6-5. INFERIOR VENA CAVA (IVC) ESTIMATES OF RIGHT ATRIAL (RA) PRESSURE

IVC Size, cm	Respiratory Change	RA Pressure, cm
<1.5	Total collapse	0–5
1.5–2.5	>50% collapse	5–10
1.5–2.5	<50% collapse	11–15
>2.5	<50 % collapse	16–20
>2.5	No change	>20

a formula or calculation package. The specifics of this measurement are beyond the scope of the text.

M-MODE

M-mode (motion mode) allows for a one-dimensional tracing of structures over time. M-mode can record motion of structures faster than human vision and record subtle changes. Measurements of valve diameters, wall motion, wall thickness, and stroke volume are possible.



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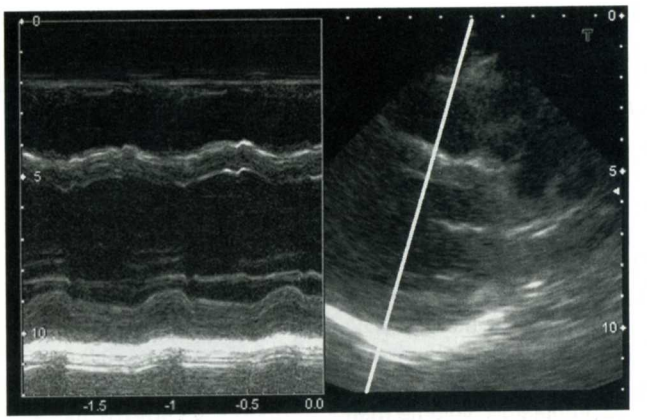

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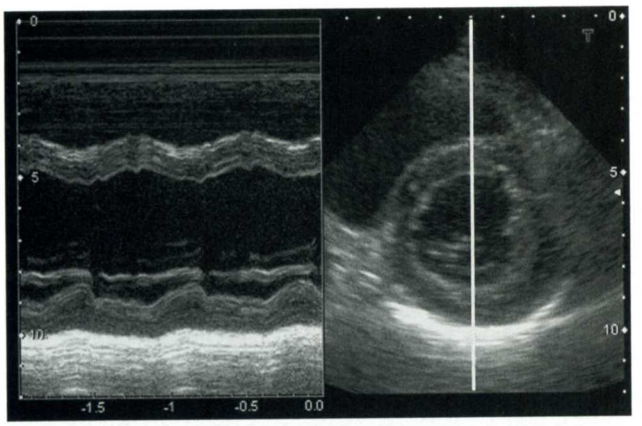


C

Figure 6-18. M-Mode diagram of left ventricle (A). Split screen M-Mode ultrasound from parasternal long-axis view (B). Split screen M-Mode ultrasound from parasternal short-axis view (C). White line indicates approximate position of M-Mode cursor for LV measurements. RV = right ventricle, LV = left ventricle, ESD = end systolic diameter, EDD = end diastolic diameter. (Courtesy of Hennepin County Medical Center for (B,C))

There are several uses of M-mode tracing but the most common in emergency ultrasound is the tracing through the left ventricle for measurement of left ventricular size and function and to confirm the presence of a pericardial effusion.

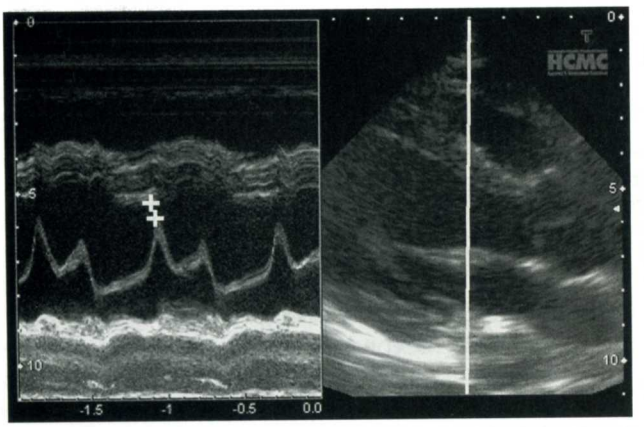


Figure 6-19. Split screen M-Mode ultrasound at mitral valve level. White line indicates approximate position of M-Mode cursor for recording anterior mitral valve leaflet. Measurement cursors are positioned for EPSS which equals 6 mm in this example. (Courtesy of Hennepin County Medical Center)

M-MODE LEFT VENTRICLE

This is usually performed perpendicular to the long axis of the left ventricle in the parasternal long or short axis at the level of the tips of the mitral valve leaflets (Figure 6-18A–C). This tracing cuts through the right ventricle, interventricular septum, left ventricle anterior wall, left ventricle posterior wall, and posterior pericardium. The end systolic diameter (ESD) is measured at the smallest dimensions of the septum and posterior wall. The end diastolic diameter (EDD) is measured just prior to ventricular thickening and contraction. An EDD measurement >6.0 cm is consistent with LV dilation. These measurements can also be used in formulas to calculate left ventricular fractional shortening and ejection fraction.

M-MODE MITRAL VALVE AND LEFT VENTRICULAR DYSFUNCTION

An M-mode tracing at the mitral valve level in the parasternal long axis shows the right ventricular free wall, interventricular septum, the mitral valve leaflets, the posterior left ventricular wall, and the pericardium. An M-mode tracing through the anterior leaflet of the mitral valve produces a double peak pattern (Figure 6-19).

The first peak is the E point and is caused by passive left ventricular filling. The second peak is the A point and is caused by atrial contraction. This double peak pattern is evidence of sinus rhythm. The distance between this E point and the left ventricular (LV) septal wall is measured as the E-point septal separation (EPSS). A large EPSS (in the absence of mitral stenosis) reflects left ventricular systolic dysfunction, left ventricular dilatation, or aortic regurgitation. An EPSS value greater than 8–10 mm is considered abnormal. The cause of the EPSS increase in these conditions is fairly simple: LV systolic dysfunction limits flow velocity, so maximum valve opening is reduced, LV dilatation increases the distance between the open valve and septum; and significant aortic regurgitation produces backflow that pushes the mitral valve away from the septum.

DOPPLER MEASUREMENTS

The use of the Doppler principle in ultrasound depends on the measurement of frequency shifts from moving red blood cells within cardiac structures. The measured velocity of the red blood cells depends on the transducer frequency, the speed of red blood cells in vessels, and the angle between the beam and the direction of flow. The sampling beam should be positioned as parallel to flow as possible.

Laminar flow is similar to that inside a tube, with a leading edge of fast flow and slow flow along the walls. Pressure differences drive blood across the vessel or in the heart. With turbulent flow, blood is swirling, moving in all directions but is moving forward. At stenoses, the flow is faster than at narrower points because the volume of blood moving across the stenosis must be the same as in other areas of the heart. Doppler measurements are taken in different windows to measure flows from different chambers and vessels.

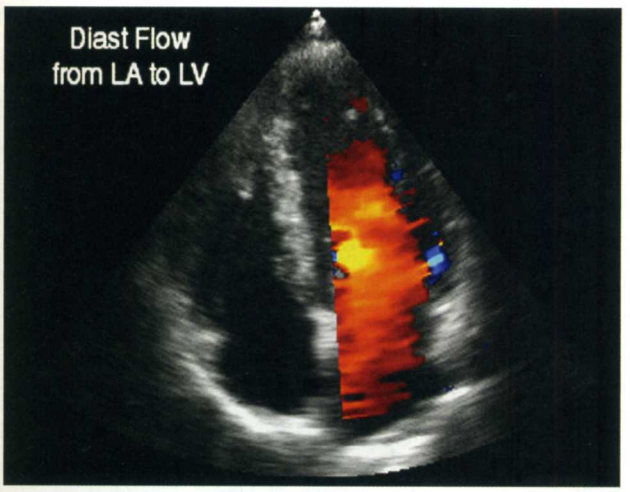
COLOR DOPPLER FLOW

Color Doppler flow is a representation of blood flow, usually as red or blue, in regards to the direction from the transducer. Conventionally, red flow is toward the transducer and blue flow is away from the transducer (Figure 6-20A). It should be noted that the colors red and blue do not necessarily signify arterial or venous flow or vessels. In addition, degrees of velocity are mapped as shades of red and blue. Shades of orange, green, or yellow may represent degrees of velocity, variance, or turbulence (Figure 6-20B).

▶ COMMON AND EMERGENT ABNORMALITIES

CARDIAC TAMPONADE

Cardiac tamponade is not dependent on the amount of fluid in the pericardial sac but on the rate of fluid accumulation within the pericardial sac (Figure 6-21).[101] Emergent echocardiographic findings of cardiac tamponade include a pericardial effusion, right atrial collapse during ventricular systole, right ventricular diastolic collapse (Figures 6-22 and 6-23), and lack of respiratory variation in the IVC and hepatic veins.[101] Left atrial or left ventricular collapse may occur in localized left-sided compression or in severe pulmonary hypertension.



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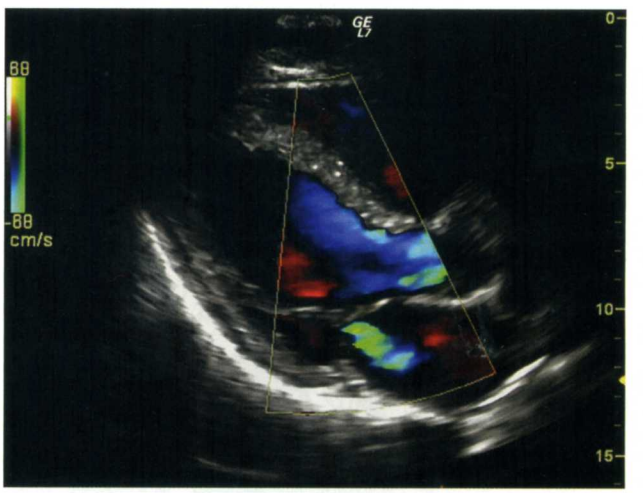


B

Figure 6-20. Color Doppler flow through left ventricle, apical four-chamber view - conventional color key (A). Flow during diastole is toward the transducer and is coded red in this example. Color Doppler flow on parasternal long-axis view - variance mode color key (B). Shades of green have been added to indicate areas of turbulence. (Courtesy of GE Medical for (B))

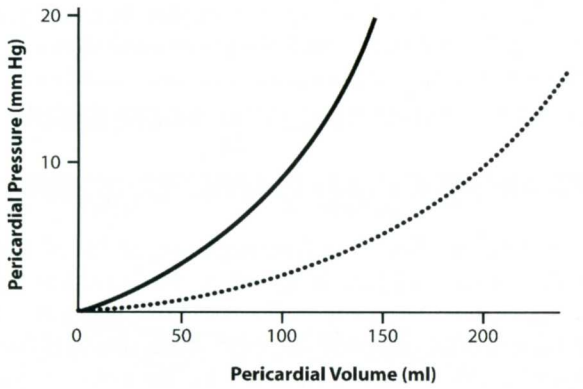


Figure 6-21. Pericardial P/V curve with acute (solid line) vs. chronic effusion (dotted line).

PERICARDIAL EFFUSION

Pericardial effusion is typically characterized by an anechoic fluid collection between the parietal pericardium and the visceral pericardium (Figures 6-24A and 6-24B, 6-25). For all practical purposes, the visceral pericardium is not visualized by transthoracic echocardiography. However, the combined interface of the parietal and visceral pericardium is echogenic.

On transthoracic echocardiography, pericardial effusions may be judged as small or large. Small pericardial effusions are seen as an anechoic space less than 1 cm thick and are often localized, usually between the posterior pericardium and left ventricular epicardium. Large effusions are seen as an anechoic space greater than 1 cm thick, and are usually completely surrounding the heart. In patients with larger effusions, the heart may swing freely within the pericardial sac (Figure 6-26).

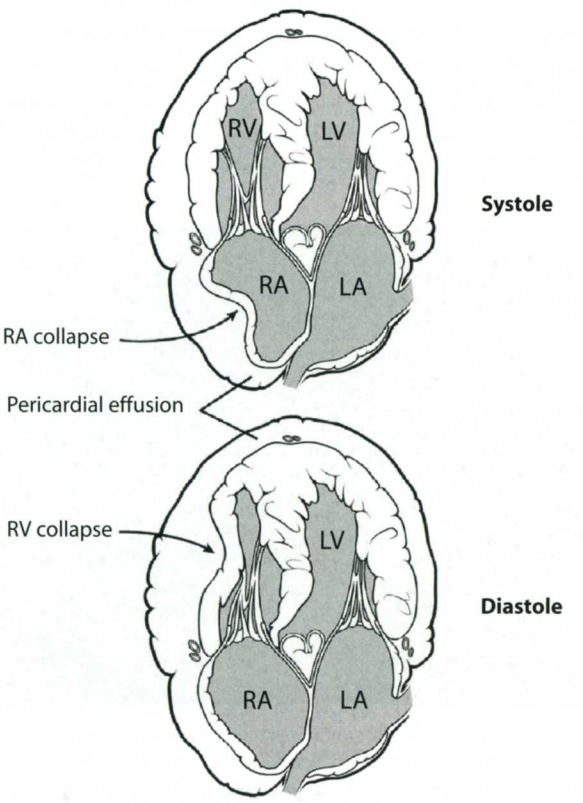


Figure 6-22. Physiology of cardiac tamponade.

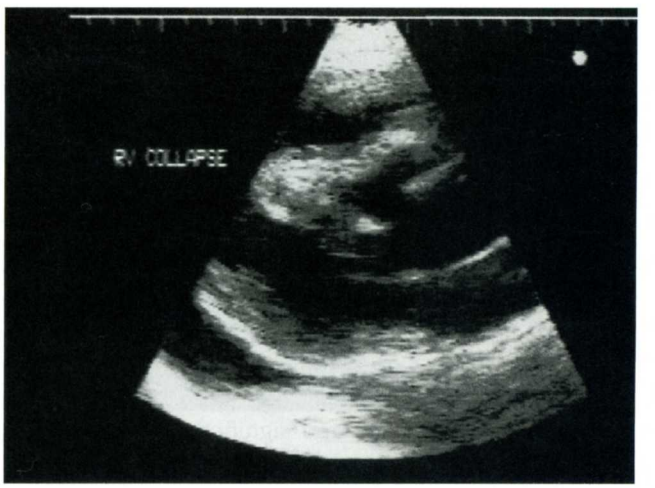
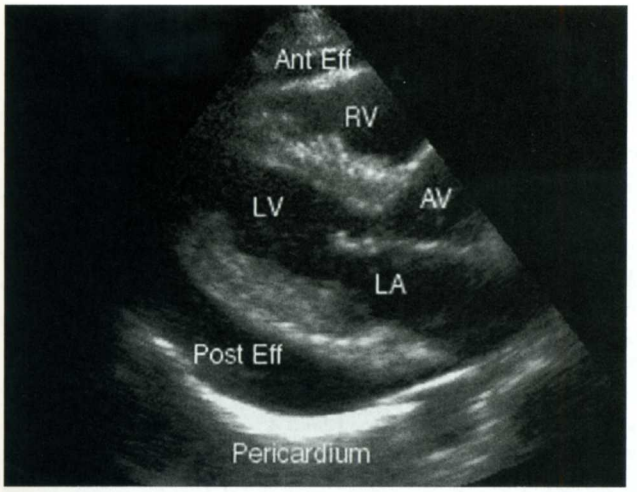


Figure 6-23. Cardiac tamponade. Parasternal long-axis view with diastolic collapse of right ventricle. (Courtesy of James Mateer, MD)

Pericardial volumes of up to 50 mL may be normal; however, pathologic fluid collections, if slow in progression, may accumulate hundreds of milliliters. Pericardial fluid is usually anechoic, but exudative effusions, such as pus, malignant effusions, and blood mixed with fibrin material, may be echogenic (Figure 6-27). Pericardial fluid collections can be complicated by gas-forming infections or by gas-causing tamponade (pneumopericardium).

MYOCARDIAL ISCHEMIA

Abnormal wall motion and abnormal ventricular emptying or relaxation characterizes left ventricular dysfunction (Figure 6-28). Wall motion is graded as hypokinesis (reduced ventricular wall thickening and motion), akinesis (absent wall thickening and motion), and dyskinesia (paradoxical motion of the wall—outward movement of the wall during systole).[92,102] Assessment of wall thickening requires ultrasound visualization of the myocardium and endocardium, which can be significantly limited for the typical emergency or critically ill patient. Wall motion may be characterized by gross ventricular wall dysfunction or segmental wall motion defects that usually follow the distribution of coronary blood perfusion (Figure 6-29A). Multiple views of the left ventricle and a knowledge of the coronary vascular anatomy are



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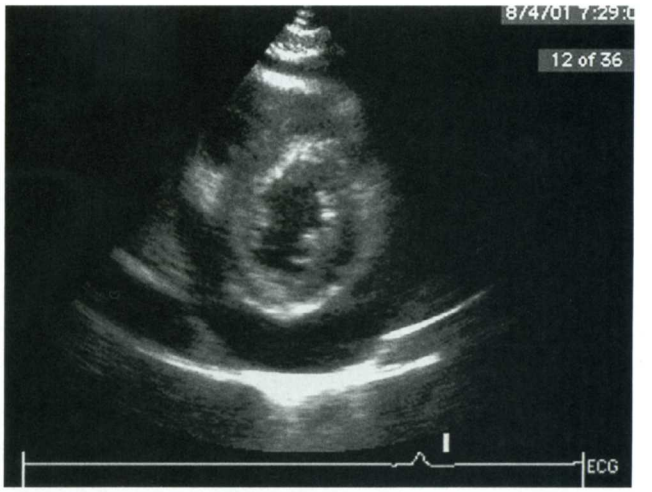


B

Figure 6-24. Pericardial effusion on parasternal long-axis view (A) and parasternal short-axis view (B). RV = right ventricle, LV = left ventricle, AV = aortic valve, LA = left atrium.

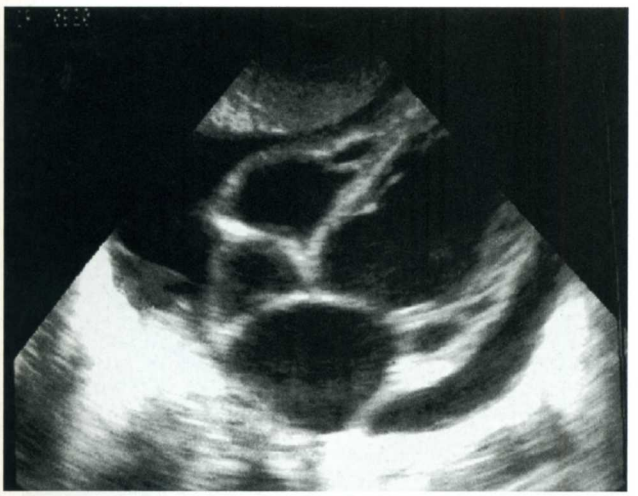


Figure 6-25. Chronic pericardial effusion (Subcostal four-chamber).

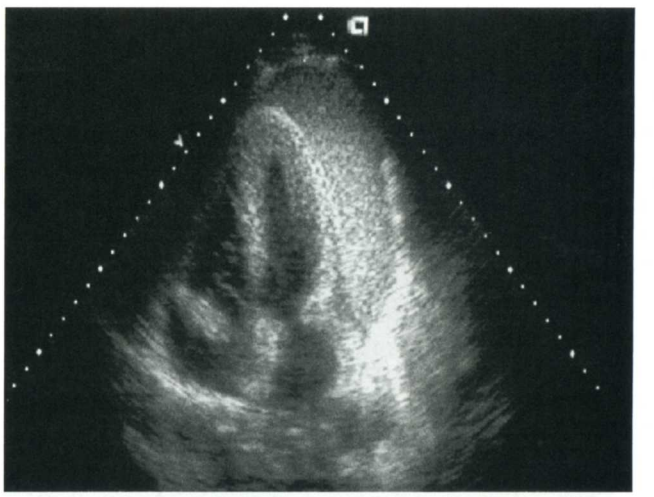


Figure 6-27. Exudative pericardial effusion (apical four-chamber view).

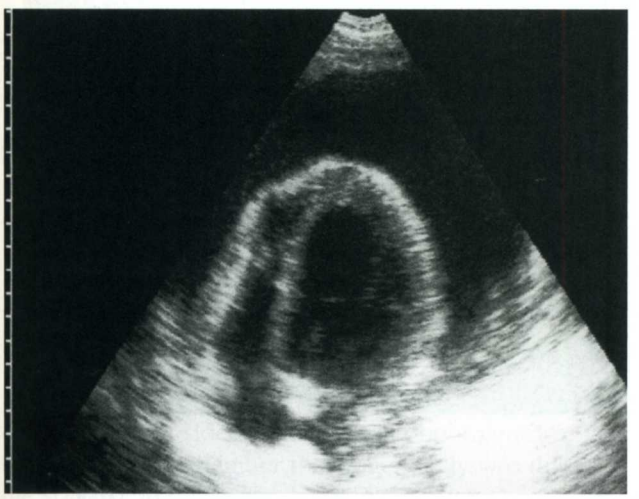


Figure 6-26. Large effusion – apical view. (Courtesy of James Mateer, MD)

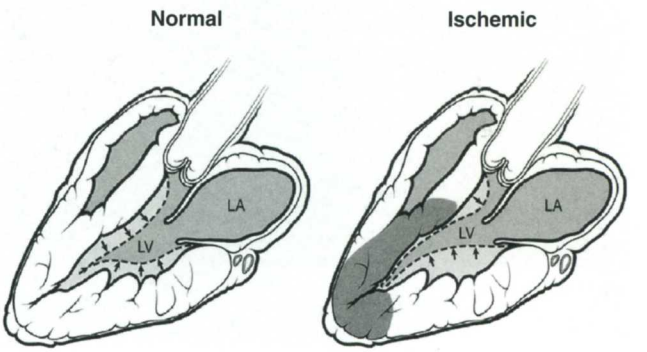
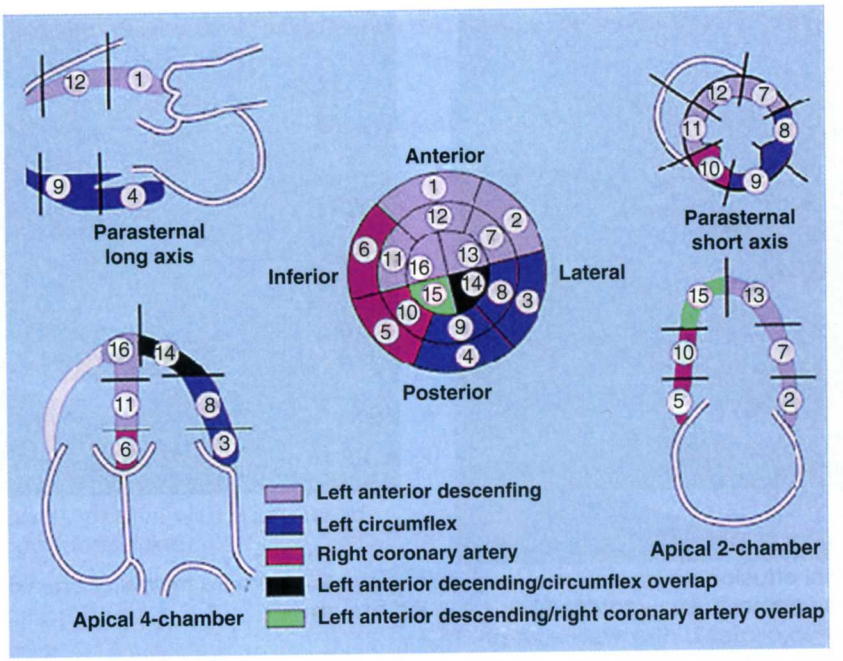


Figure 6-28. Wall motion abnormality.



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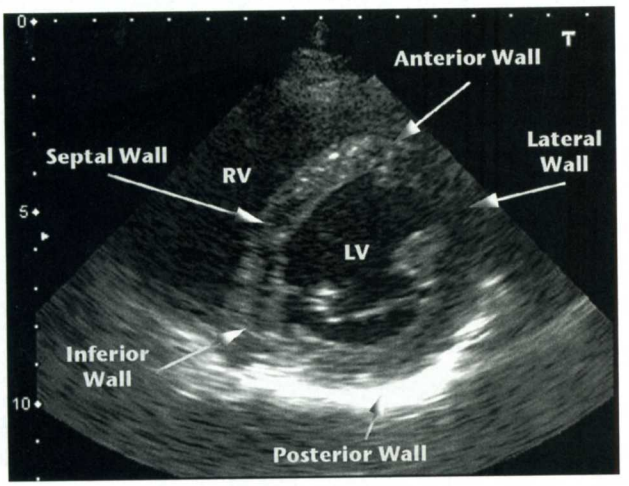

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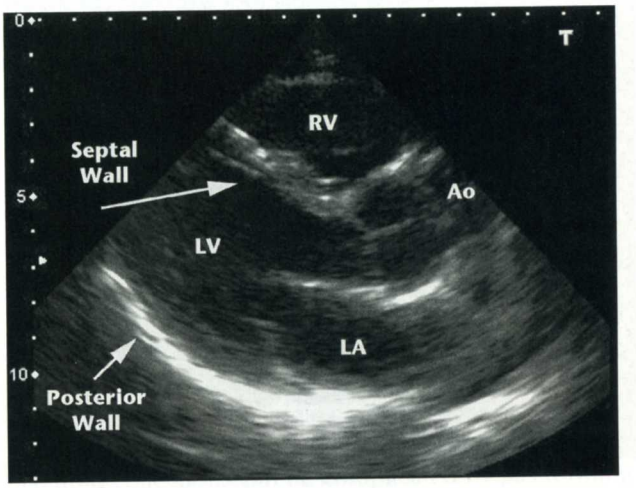

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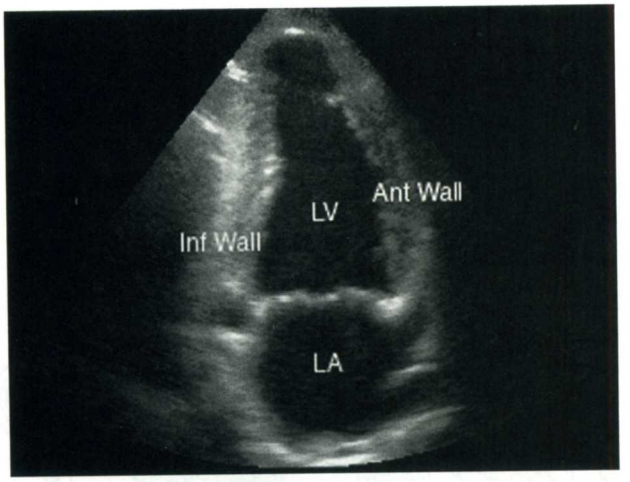

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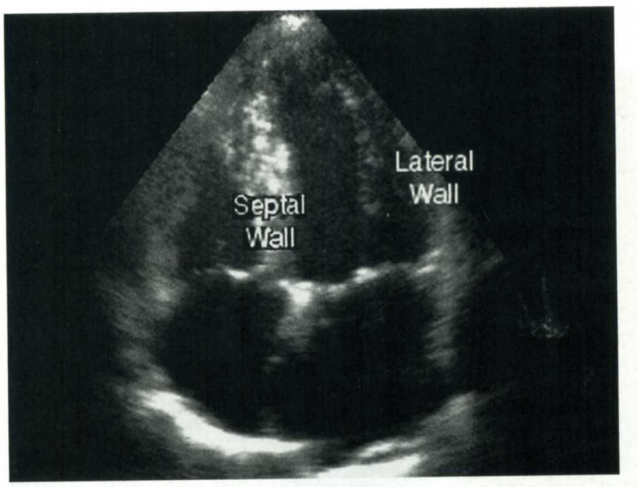
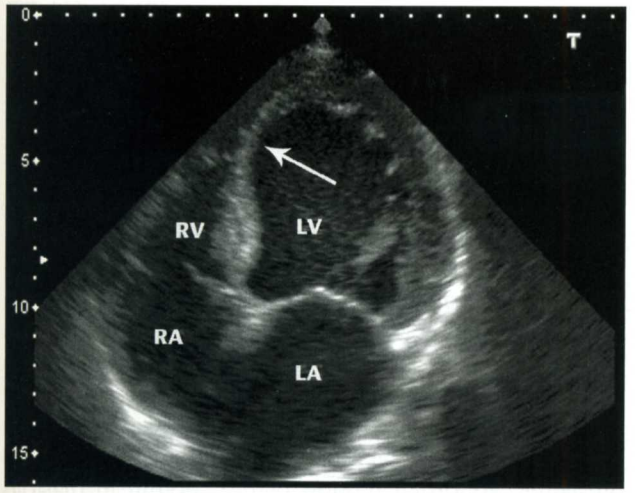


D

Figure 6-29. Left ventricular wall segments (A). Center 'bullseye" represents LV viewed from apex. Wall segments are color and number coded for regional coronary vasculature. Corresponding ultrasound images of LV wall segments in parasternal long-axis view (B), parasternal short-axis view (C), apical four-chamber view (D), apical two-chamber view (E). RV = right ventricle, LV = left ventricle, Ao = aorta, LA = left atrium. (Courtesy of Hennepin County Medical Center for (B,C))



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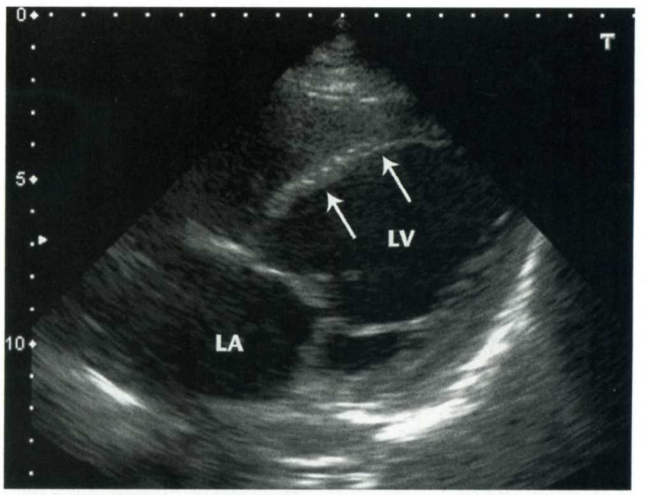


B

Figure 6-30. Chronic left ventricular infarction. Apical four-chamber view (A) demonstrates thinning and increased echogenicity of the apical septum (arrow) with increased size of LV and LA chambers. Subcostal four-chamber view (B) shows chronic thinning of the entire inferior septum (arrows). RV = right ventricle, RA = right atrium, LV = left ventricle, LA = left atrium. (Courtesy of Hennepin County Medical Center)

required if the goal is to determine the region affected and correlate findings with the EKG (Figure 6-29B–E).

Echocardiography can detect acute ischemic changes with diastolic dysfunction, increased left ventricular diastolic filling, decreased left ventricular diastolic compliance, and reduced left ventricular diastolic compliance. As the ischemia transforms to transmural infarction, there is impaired systolic thickening, reduction in endocardial motion, and dyssynchronous contraction of myocardial segments. Left ventricular chamber size increases with reduction in systolic ejection fraction. Ultrasound findings of acute left ventricular ischemia or infarction typically include wall motion abnormalities, usually regional in the distribution of a coronary artery or its branch, but without evidence of chronic thinning or scarring of the wall (Figure 6-28).

Ultrasound findings of chronic left ventricular infarction include a dilated left ventricle, global wall motion abnormalities with thinning of the ventricular wall (<7 mm or 30% less than the adjacent normal wall) and increased echogenicity of the segment due to fibrotic changes. (Figure 6-30A and 6-30B). Chronic left ventricular dysfunction will lead to a dilated cardiomyopathy and an immobile aortic root (Figure 6-31). The left atrium may be dilated and there may be thrombus at the apex.

Right ventricular dysfunction or dilatation may be the only sign of severe pulmonary disease, pericardial disease, or right-sided ischemia. The right ventricle is a thin and narrow chamber that is generally two thirds of the size of the left ventricle. While a dilated right ventricle may be seen in any view, the four-chamber apical view may be particularly useful.

MASSIVE PULMONARY EMBOLISM

While direct visualization of a thrombus may occasionally be seen in the right heart, most echocardiographically detectable changes are indirect indices of right heart strain caused by pumping against a fixed blood clot in the lung. These changes include right ventricular dilatation, right ventricular hypokinesis, tricuspid regurgitation, and abnormal septal motion.

The normal right ventricular end diastolic diameter is 21 ± 1 mm in a parasternal long axis view. Abnormal values have been described as being greater than 25–30 mm. The normal right to left ventricle ratio, obtained in

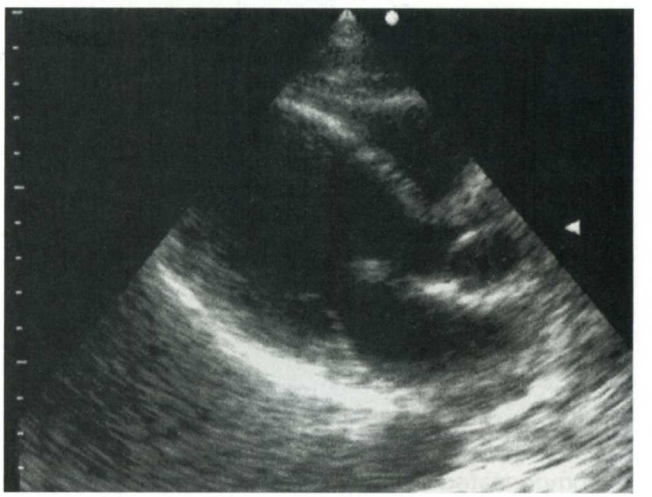


Figure 6-31. Dilated left ventricle in parasternal long-axis view.

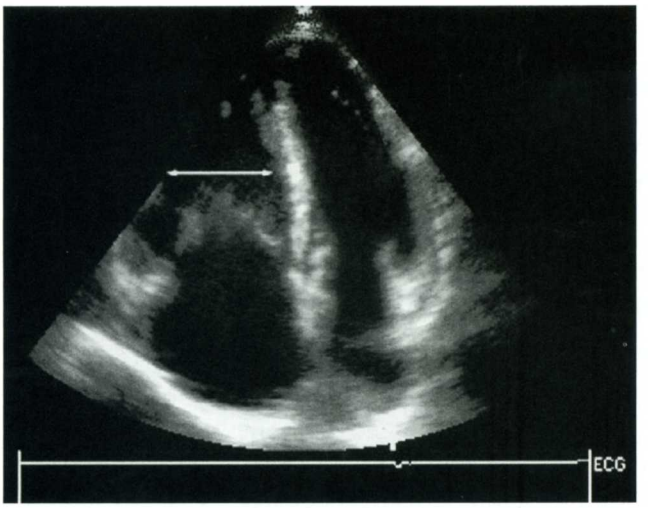


Figure 6-32. RV enlargement. Apical four-chamber transverse diameter exceeds 2.5 cm.

the apical four-chamber view, is less than 0.5. Abnormal ratios vary by author but have been described as being greater than 0.5 or as high as greater than 1 (Figure 6-32). With massive PE the right ventricle will be round in shape and larger than the left ventricle (Figure 6-33).

Tricuspid regurgitation may occur when pulmonary artery pressures exceed right ventricular end diastolic (right atrial) pressures. Measurement of tricuspid regurgitation requires spectral Doppler velocity measurement and is usually obtained on the apical four-chamber view. While many healthy persons have a trivial degree of tricuspid regurgitation, up to 90% of patients with PE will have measurable tricuspid regurgitation.[103] Normal pulmonary artery systolic pressure is approximately 25 mmHg in a healthy person, corresponding to a regurgitant jet of less than 2 m/s. Over 3 m/s would correspond to a pulmonary artery pressure of 46 mmHg. Studies using cutoff values for diagnosis of PE typically cite velocities over 2.5–2.7 m/s as being elevated.

In addition to right-sided heart strain, a blood clot in the lung may cause decreased venous return to the left heart. This may result in decreased left ventricular end diastolic diameter as well as "paradoxical septal motion." The normal interventricular septum relaxes outward (toward the right ventricle) in diastole. With increased right end diastolic pressures and decreased left-sided pressures, abnormal motion of the septum in diastole may be visualized. While this septal deviation toward the left ventricle (also described as "septal flattening") may also be observed in systole, its presence is more pronounced in diastole and is especially prominent in the acute phase of massive PE.[104]

All of the indirect indicators of right-sided heart strain may occur in conditions other than PE. These conditions include right ventricular infarct, emphysema, and primary pulmonary hypertension. It is worthwhile noting that the acutely strained right-sided heart rarely has the muscle mass to elevate pulmonary artery pressure into an extremely high range and values well over 40 mmHg should suggest a chronic elevation.[103] An increase in muscle mass on measurement of the right ventricular free wall may also indicate a more chronic etiology for right ventricular strain as opposed to a thin, acutely

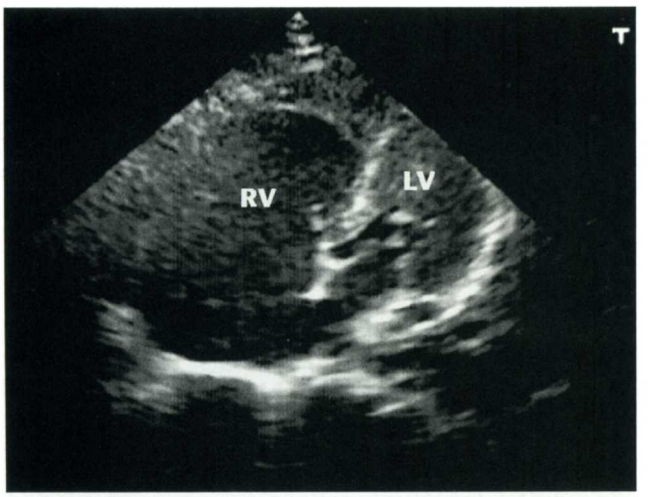


Figure 6-33. Massive pulmonary embolism. Apical view (centered over RV apex) shows severely decompensated right ventricle that is round in shape and much larger than the left ventricle. RV = right ventricle, LV = left ventricle. (Courtesy of Hennepin County Medical Center)

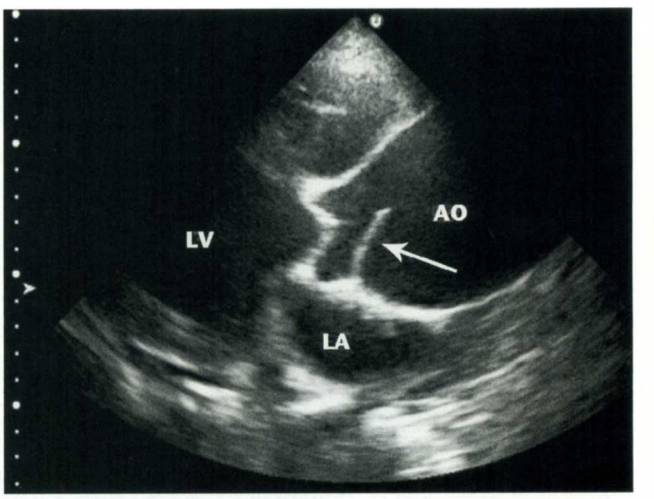


Figure 6-34. Ascending aortic aneurysm with dissection. Parasternal long-axis view shows dilated aortic root and proximal flap (arrow). Ao = aorta, LV = left ventricle, LA = left atrium. (Courtesy of Hennepin County Medical Center)

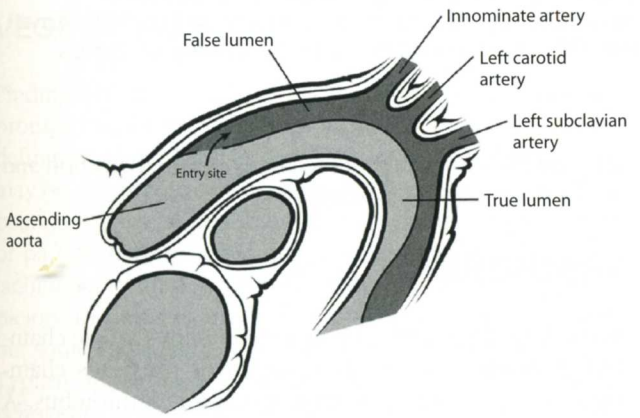
dilated right ventricle. The normal right ventricular free wall is 2.4 ± 0.5 mm, and is generally considered hypertrophied at measurements of 5 mm and greater.[105]

PROXIMAL AORTIC DISSECTION

On transthoracic echocardiography, patients with a proximal thoracic aortic dissection often have a dilated aortic root (<3.8 cm), which is easily visualized on the parasternal long axis view. An intimal flap can sometimes be seen within the dilated aortic root (Figure 6-34). The descending aorta may also be seen on the parasternal long axis view in cross section posterior to the mitral valve. The arch of the aorta may be seen with transthoracic echocardiography using a suprasternal window in a minority of patients but is more easily visualized when dilated. A dissection within the aortic arch or descending thoracic aorta may be seen in this view (Figure 6-35A and 6-35B). In addition to the linear flap, aortic dissection is characterized on echocardiography as having two lumens, true and false, with different flow patterns. This may be best demonstrated using transesophageal views (Figure 6-36A and 6-36B).

ASYSTOLE

Asystole is seen as a lack of myocardial contraction on echocardiography. Pooling of blood may be seen and echogenic clots may form with onset of asystole. As severe hypokinesis progresses toward asystole, there is progressive decreased left ventricular diastolic and systolic volumes associated with rising left ventricular



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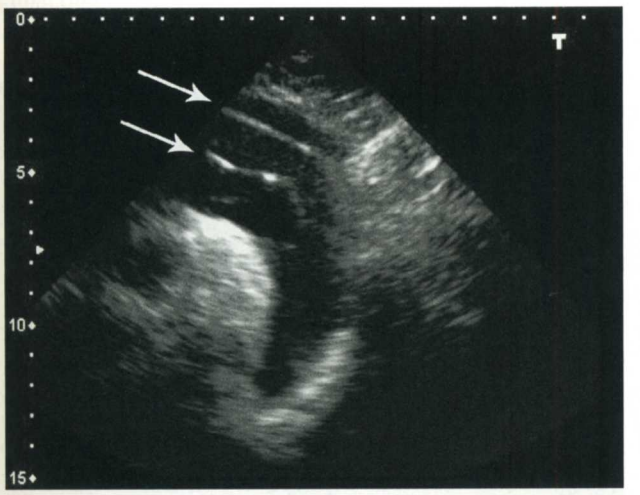
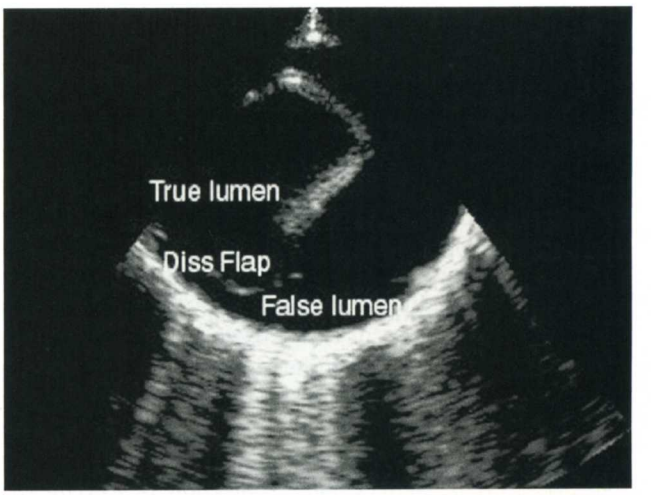
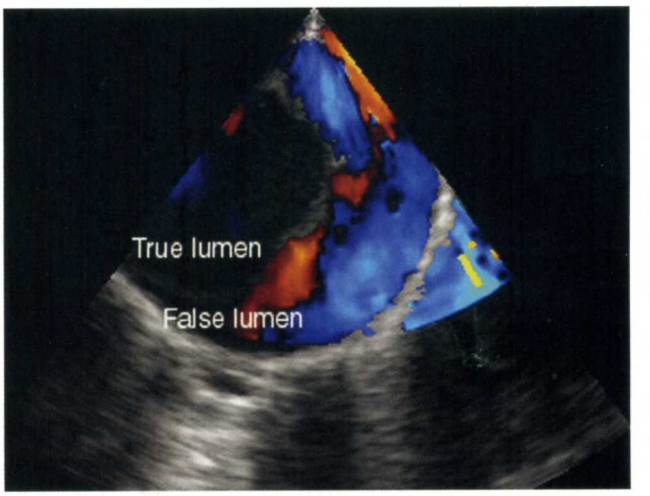


B

Figure 6-35. Type A aortic dissection diagram (A). Suprasternal ultrasound view of the aortic arch (B). The imaging plane crosses the intimal flap in two locations (arrows). (Courtesy of Hennepin County Medical Center for (B))

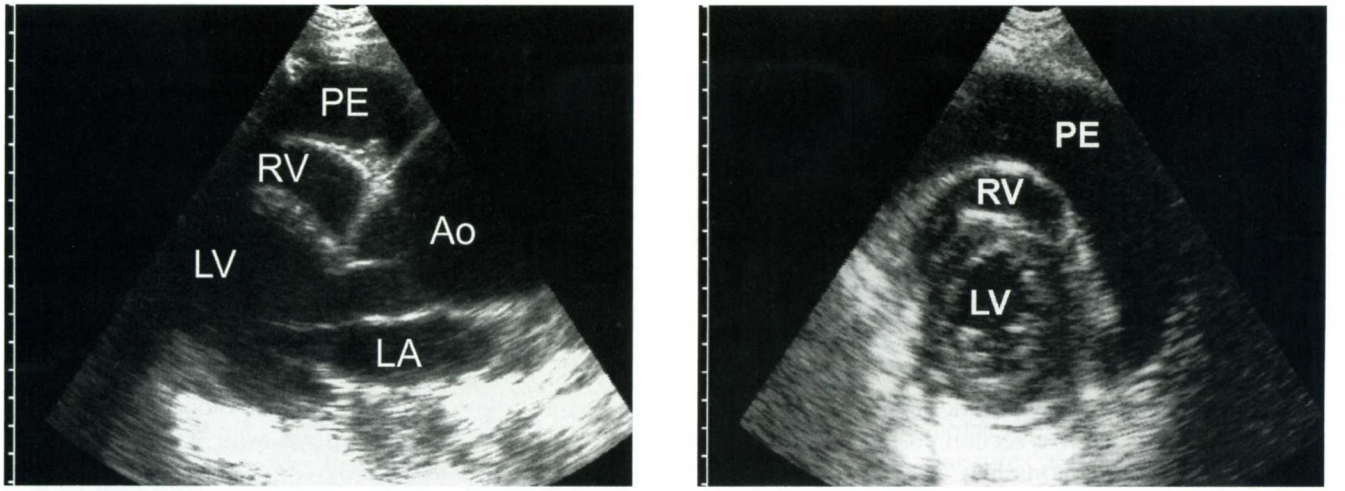


A



B

Figure 6-36. TEE transverse view of an aortic dissection with flap (A). TEE aortic dissection, color Doppler with true-and-false lumens (B).



A B

Figure 6-37. Aortic aneurism. Parasternal long-axis view shows a 6-cm aneurysm in the ascending aorta (A). The pericardial fluid collected anteriorly. The enlarged aorta may be pushing the LV against the posterior pericardial sac. This is best seen on the parasternal short axis view (B). No intimal flap was found on TEE. Ao = Aorta, LA = left atrium, LV = left ventricle, RV = right ventricle, PE = pericardial effusion. (Courtesy of James Mateer, MD)

pressures. Ventricular wall thickness progressively increases until the heart becomes motionless.[106] Movement of the valves can be seen just with positive pressure ventilation and should not be taken for spontaneous circulation in the absence of myocardial contraction.

▶ COMMON VARIANTS AND SELECTED ABNORMALITIES

ASCENDING AORTIC ANEURYSM

Dilation of the ascending aorta over 1.5 times the normal segment may reflect an aneurysmal change (Figure 6-37A and B). A true aneurysm of the ascending aorta involves all layers of the vessel wall. A false aneurysm, or pseudoaneurysm, involves a penetration of the intima and media layers only. Most thoracic aneurysms are fusiform but may be saccular. Concomitant aortic dissection may occur as well.

On echocardiography, the aorta is usually measured at several locations: aortic annulus, aortic leaflet tip, ascending aorta, aortic arch, and descending aorta. The length and levels of dilatation should be noted. As with the abdominal aorta, if the thoracic aorta diameter is measured at 5–6 cm, then the patient should be referred to a cardiothoracic surgery consultant.

The role of transthoracic echocardiography is limited as the aortic arch and descending aorta cannot be fully visualized because of the depth of the aorta in many views. There is also difficulty in viewing the endothelium and poor windows due to intervening bone and air. TEE, CT, and MRI are similar in accuracy for the detection and evaluation of aortic aneurysm.

THROMBUS

While a thrombus may be detected in any cardiac chamber, slow-moving chambers or lower pressures chambers are at greater risk for developing a thrombus. A thrombus may be hyperechoic, isoechoic, and even hypoechoic in appearance (Figure 6-38). It is usually laminated, with the layers paralleling the chamber wall. A

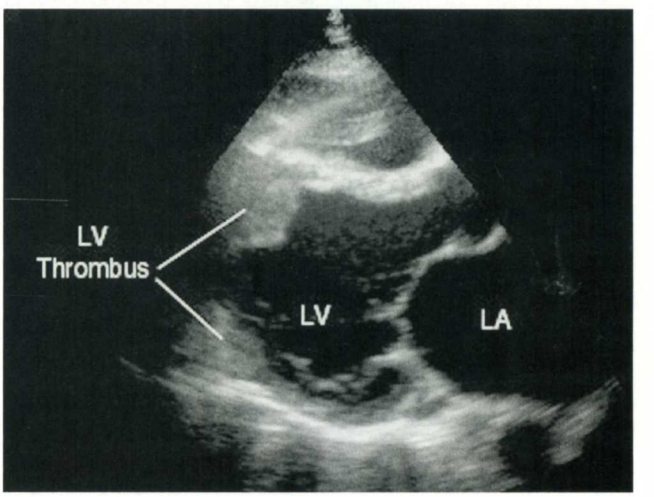


Figure 6-38. A left ventricular thrombus is located near the apex (parasternal long-axis view). LV = left ventricle, LA = left atrium.

thrombus is typically homogeneous with irregular borders, and may fill in the apex of a ventricle or attach itself to a chamber wall or valves of the atria. Near-field or time-gain compensation may have to be adjusted to visualize suspected areas. A thrombus may make differentiating the pericardial layers difficult.

High-frequency transducers that utilize cardiac scanning windows close to the cardiac chamber in question provide the best imaging. While transesophageal transducers are required for thrombus detection in atria, transthoracic scanning is adequate for thrombus detection within the ventricles in many cases. If color Doppler is available, the swirling vortices of flow may indicate the presence of a thrombus. Normal structures, such as the left atrial appendages, right atrial Chiari network, and right ventricular moderator bands must be distinguished from thrombus.

VEGETATIONS

Findings of irregularities on valvular surfaces should prompt further investigation and consultation for more definitive diagnosis (Figures 6-39 and 6-40). Vegetations may be echogenic or isoechoic and have an irregular appearance. Vegetations may be seen on any valve leaflet or part of the apparatus. Laminated or pedunculated attachments to the leaflet of the valve should prompt suspicion. In general, they do not restrict valvular motion but some valve leaflets may not coapt together correctly. Typical appearance of normal valves includes smooth echogenic leaflets. All suspected cases should be referred for transesophageal imaging and cardiology consultation.

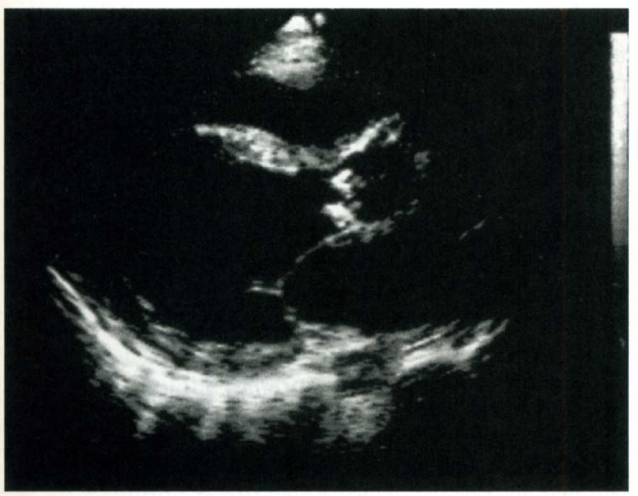


Figure 6-39. Endocarditis. Parasternal long-axis view reveals echogenic mobile vegetations on the aortic valve leaflets. (Courtesy of Lori Sens and Lori Green, Gulfcoast Ultrasound)

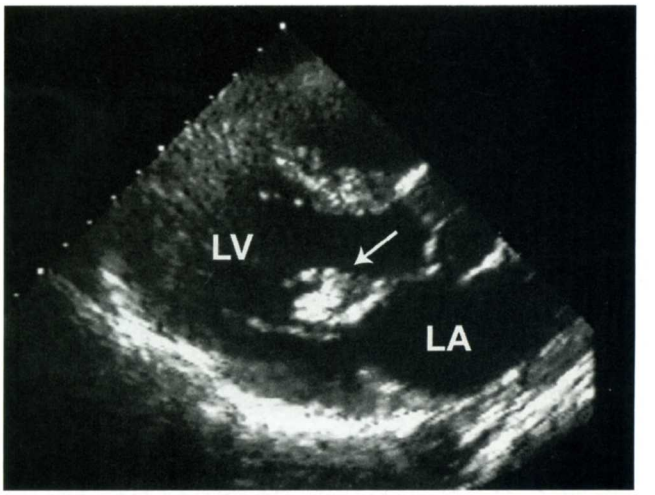


Figure 6-40. Endocarditis. Parasternal long-axis view with echogenic mobile vegetations on the mitral valve leaflets (arrow). LV = left ventricle, LA = left atrium. (Courtesy of Lori Sens and Lori Green, Gulfcoast Ultrasound)

VALVULAR ABNORMALITIES

Valvular abnormalities may present as an incidental finding and should be recognized for appropriate referral (Figure 6-41A and B). Most hemodynamically significant valvular abnormalities will eventually cause cardiac chamber enlargement and this may lead the sonologist to the diagnosis. In the setting of acute myocardial infarction, a new onset murmur can be caused by mitral regurgitation associated with papillary muscle dysfunction or rupture. This may be recognized on ultrasound as prolapse of the mitral valve leaflets or by abnormal color flow Doppler of the mitral valve (Figure 6-42). Another consideration is acute ventricular septal defect that may be seen with color flow imaging.

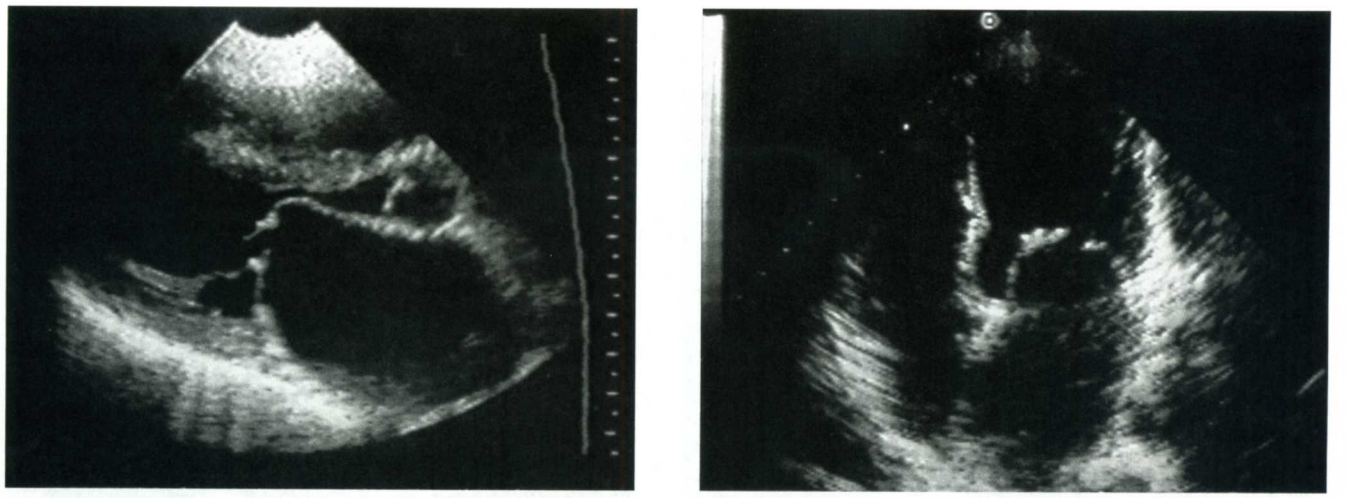
Tricuspid and pulmonary valve abnormalities usually are not emergencies unless large masses or clots are obstructing the valves. In acute ischemic or traumatic events, the mitral valve may provide a clue to injury. Aortic valve involvement may be associated with ascending aortic abnormalities.

VENTRICULAR HYPERTROPHY

Normal left ventricular wall thickness is 0.6–1.2 cm measured at end diastole. Left ventricular hypertrophy may be concentric (Figure 6-43) or asymmetric (Figure 6-44).

MYXOMA

Myxomas, which are uncommon benign fibrous tumors, are usually attached to a septal wall. Myxomas are



A B

Figure 6-41. Mitral stenosis. Parasternal long-axis view (A) shows the typical features: LA enlargement, ballooning of the valve and a "hockey stick" appearance of the anterior leaflet. Apical four-chamber example (B) of this condition. (Courtesy of Lori Sens and Lori Green, Gulfcoast Ultrasound)

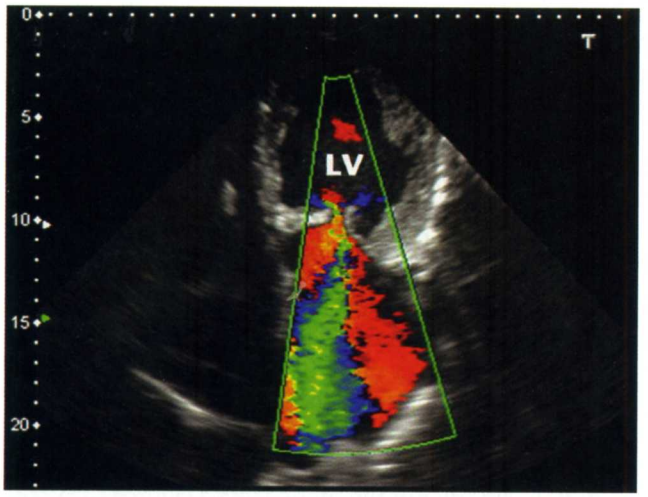


Figure 6-42. Mitral insufficiency. A shortened apical view with color Doppler demonstrates severe mitral regurgitation with turbulent flow. LV = left ventricle. (Courtesy of Hennepin County Medical Center)

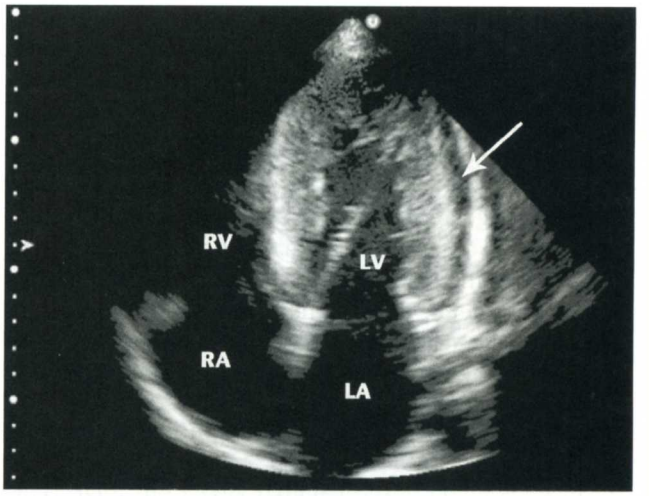


Figure 6-43. Concentric hypertrophy. Apical four-chamber view demonstrates symmetrical thickening of the left ventricular wall. A small pericardial effusion is noted adjacent to the left ventricle (arrow). LA = left atrium, LV = left ventricle, RV = right ventricle, RA = right atrium. (Courtesy of Hennepin County Medical Center)

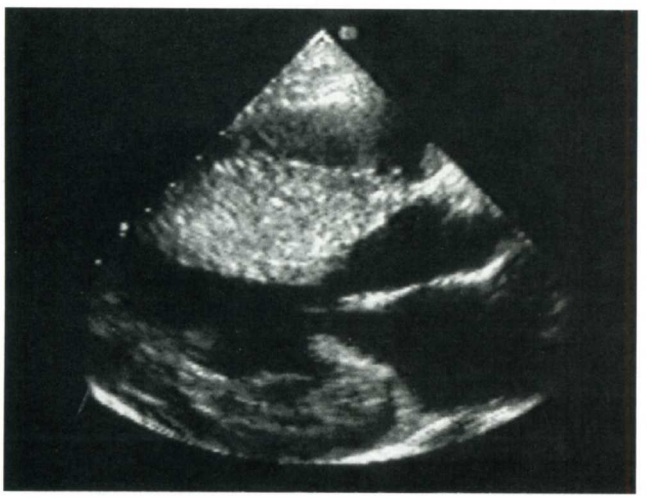
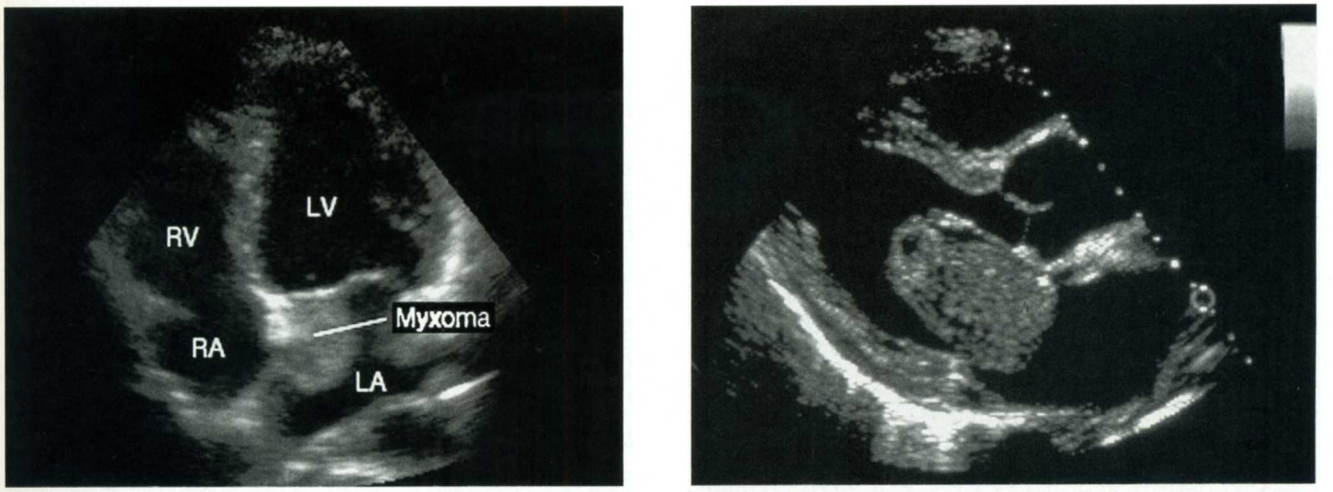


Figure 6-44. Assymetric septal hypertrophy. A thickened, echogenic LV septum is noted in PSL view in a patient with this condition (also known as IHSS). (Courtesy of Lori Sens and Lori Green, Gulfcoast Ultrasound)



A B

Figure 6-45. Left atrial myxoma shown on apical four-chamber view (A). Left atrial myxoma shown on a parasternal long-axis view (B). The mass was mobile and prolapsing into the LV on the real-time exam. (Courtesy of Lori Sens and Lori Green, Gulfcoast Ultrasound)

usually echogenic, globular, and smooth. They are pedunculated with a stalk on one wall that may or may not be visualized. They are usually seen attached to an atrial wall, most often the left atrium (Figure 6-45A and 6-45B).

▶ PITFALLS

1. **Contraindications.** No contraindications exist for transthoracic echocardiography unless its use is interfering with life-saving procedures and treatments.
2. **Inability to obtain adequate views.** Some patients cannot be imaged well by transthoracic echocardiography. These include patients with subcutaneous emphysema, pneumopericardium, large anterior–posterior girth, and chest wall deformities. Suggestions for improving image acquisition include maintaining transducer contact with the chest wall, use of an adequate amount of conduction gel, use of adjacent cardiac windows, and angling, rotating, and tilting the transducer, as necessary. The patient may be turned in the left lateral decubitus position to bring the heart closer to the anterior chest wall.
   a. The subxiphoid window is a mainstay of the emergency cardiac ultrasound examination during resuscitation of a critically ill patient. Suggestions for improving image acquisition for this view include ensuring the transducer is at a shallow angle to the plane of the body (15° in general) and moving the transducer to the patient's right in the subxiphoid space instead of the more intuitive left side. This helps to avoid the air-filled stomach and uses the left lobe of the liver as a soft-tissue window. Also, asking the patient to take a deep inspiration or, if the patient is intubated, providing a large tidal volume will help push the heart toward the subxiphoid space.
   b. The parasternal view is limited by retrosternal air or altered anatomy. Moving the transducer to the left, and then up and down along the anterior–posterior axis may help with obtaining a better view.
   c. The apical view may be improved by changing the angle and aiming the transducer toward the head or right elbow instead of the right shoulder.
3. **Reversed orientation.** Proper imaging requires knowledge of the orientation of the transducer. Reverse orientation may lead the sonologist to mistaken ventricular hypertrophy for normal and vice versa. For example, a dilated right ventricle is an important clue for massive PE, but may be falsely identified as normal if a normal left ventricle is viewed on the reversed side of the monitor screen. When ventricle sizes are similar, the right ventricle can be identified on apical four-chamber view by recognizing that the tricuspid valve is positioned closer to the apex than the mitral valve.
4. **Fluid versus blood clot or fat.** Fluid (serous pericardial fluid, or defibrinated blood) will appear as anechoic. However, a blood clot may

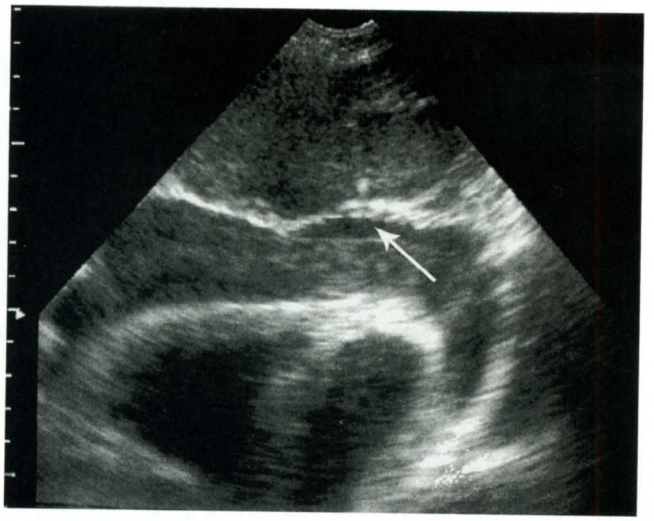


Figure 6-46. Hemopericardium. Echogenic clotted blood with a thin stripe of liquid blood above (arrow) is shown in this subcostal view.

be echogenic initially (Figure 6-46). The borders of clot usually have a thin anechoic stripe. Viewing other windows may assist with identifying free fluid in other aspects of the pericardium. Fat is commonly located in the anterior precordial space. In some patients this appears hypoechoic and can be mistaken for fluid or hematoma. Clues to identification are mildly echogenic septations characteristic of fat and the lack of any dependent pooling of fluid within the posterior pericardial space.

5. **Gain issues.** Gain should be adjusted to allow the posterior aspect of the heart to have the highest time-gain compensation. Cardiac chambers should be anechoic and cardiac structures should be echogenic.
6. **Depth.** Depth should be adjusted to visualize posterior to the cardiac structure in question. The focus, if adjustable, should be placed at the structure of interest. Too much magnification can alter proper interpretation and too shallow depth can minimize pathologic findings. A large pericardial effusion may occasionally be missed by a novice sonologist if the depth is not adequate to capture the entire heart in view and the large fluid stripe between the right ventricular wall and diaphragm in the subxiphoid view is mistaken for the right ventricle.
7. **Dynamic range.** Many machines used for emergency ultrasound applications are preset for abdominal applications; this includes the dynamic range setting. In cardiac ultrasound, the image is more black and white. The dynamic range should be lower than the settings used in abdominal or pelvic imaging.

▶ CASE STUDIES

CASE 1

Patient Presentation

A 64-year-old woman presented to the emergency department by ambulance in severe respiratory distress. A nebulized albuterol treatment was in progress. She told paramedics that her shortness of breath had become progressively worse over the last several hours. She denied chest pain and any history of cardiac or pulmonary disease. The paramedics communicated that her respiratory distress was worsening.

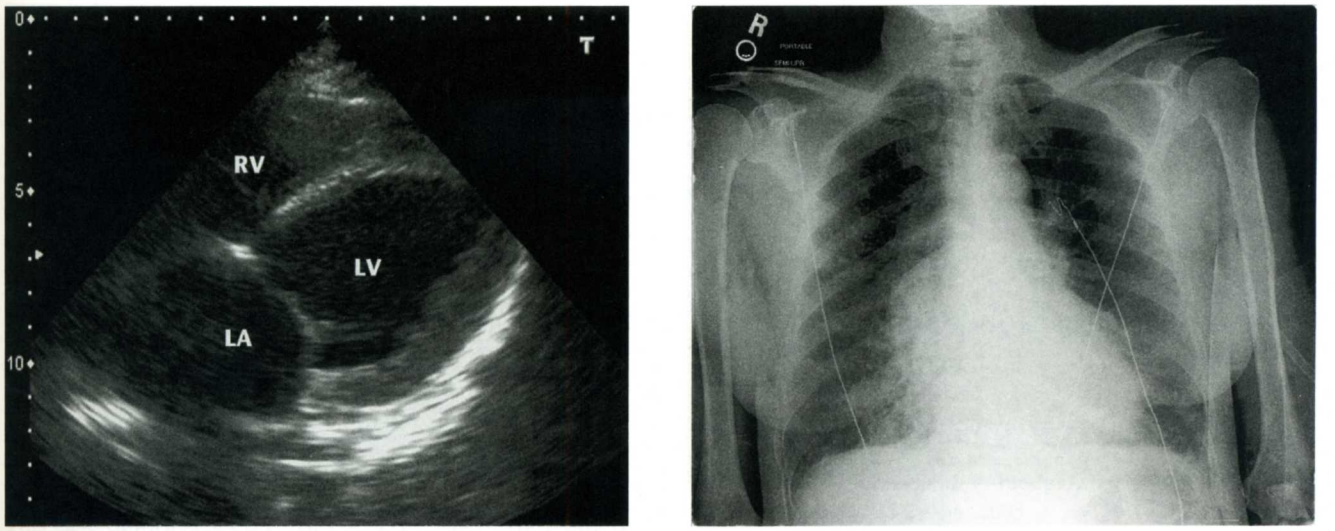
On physical examination, her respiratory rate was 50–60 breaths per minute and she was using all accessory muscles. She could only speak in two or three word sentences due to her dyspnea. Her blood pressure was 161/101 mmHg; heart rate, 136 beats per minute; and oxygen saturation 94% despite receiving 100% supplemental oxygen by nonrebreather mask. Her temperature was normal. Auscultation of her chest revealed diffuse expiratory wheezes, decreased aeration, and a prolonged expiratory phase. There were no crackles appreciated. Cardiovascular examination revealed tachycardia without murmurs and strong, equal peripheral pulses. Neck examination was without any noticeable jugular venous distention. Lower extremity edema was absent. The remainder of her examination was unremarkable.

Management Course

Two minutes after arrival, a bedside echocardiogram was performed and interpreted by the emergency physician. Notable findings were a dilated left ventricle with obvious severe left ventricular failure and a relatively small right ventricle (Figure 6-47A). The nebulization treatment was stopped and she was given high-flow oxygen and sublingual nitroglycerin. The patient received an intravenous bolus of furosemide and an intravenous nitroglycerin infusion was started. By the time her portable chest radiograph was available for viewing (Figure 6-47B) about 15 minutes after arrival, the patient was markedly improved. The chest radiograph confirmed the diagnosis of acute pulmonary edema. An ECG showed sinus tachycardia with nonspecific changes. She was admitted to the cardiac ICU and eventually diagnosed with severe diffuse ischemic cardiomyopathy.

Commentary

Bedside echocardiography was an important tool in the evaluation of this patient's undifferentiated respiratory distress. The course of treatment delivered to this critically ill patient in the emergency department was significantly altered by the information provided by bedside echocardiography. Even though the patient could



A B

Figure 6-47. Case 1: Subcostal four-chamber ultrasound view. (A). Portable chest radiograph (B). (Courtesy of Hennepin County Medical Center)

not tolerate lying flat, subxiphoid probe positioning proved adequate visualization of her left ventricular dysfunction. This vital piece of information would not have been detectable by any other diagnostic modality within 2 minutes of the patient's arrival.

CASE 2

Patient Presentation

A 52-year-old man presented to the emergency department with vague, nonradiating chest pain for the past 2–3 hours that was gradual in onset over about 30 minutes. The patient had not experienced any significant shortness of breath, nausea, or palpitations. He acknowledged a history of inconsistently controlled hypertension over the past 20 years and a 40 pack-year history of smoking cigarettes.

On physical examination, blood pressure was noted at 182/100 mmHg; heart rate, 87 beats per minute; respiratory rate, 15 breaths per minute; and oxygen saturation 98% on room air. The patient was afebrile. Head, neck, pulmonary, abdominal, and back examinations were unremarkable. Cardiovascular examination revealed normal heart sounds without murmurs. Normal and equal peripheral pulses were palpable in the upper and lower extremities.

Management Course

The patient was given an aspirin and sublingual nitroglycerin without improvement. Morphine sulfate provided some relief. His ECG showed a normal sinus rhythm and nonspecific ST changes. Chest radiograph was negative for pneumothorax and showed a normal appearing mediastinum. Laboratory studies, including the initial cardiac enzyme, were unremarkable. The patient was considered to have nonspecific, but concerning chest pain and plans were made for hospital admission, serial cardiac enzymes, cardiac monitoring, and further cardiac workup. As part of a routine chest pain evaluation the emergency physician performed bedside echocardiography and noted a dilated aortic root with a diameter of 4.2 cm (Figure 6-48). Contrast-enhanced CT of the thoracic aorta confirmed suspicions of a proximal aortic dissection. Cardiovascular surgery was contacted and performed a timely repair without incident.

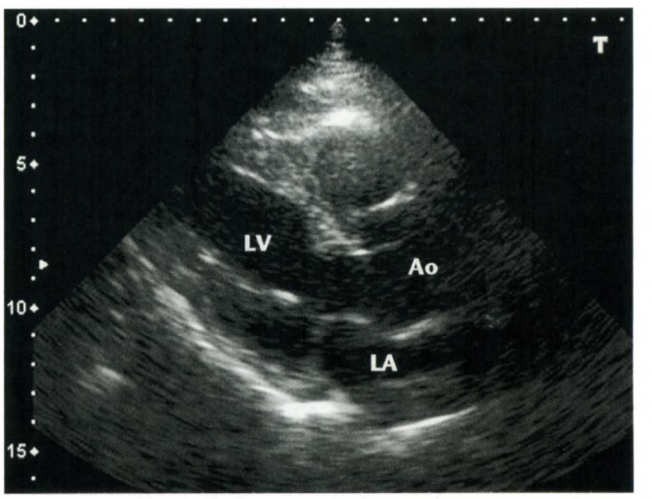
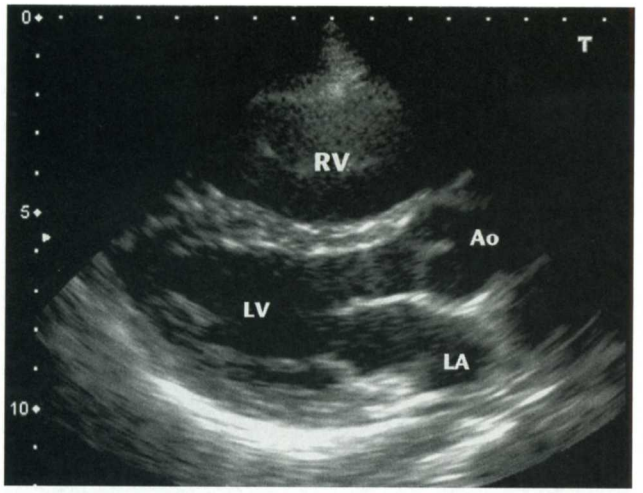


Figure 6-48. Case 2: Parasternal long-axis ultrasound view. (Courtesy of Hennepin County Medical Center)



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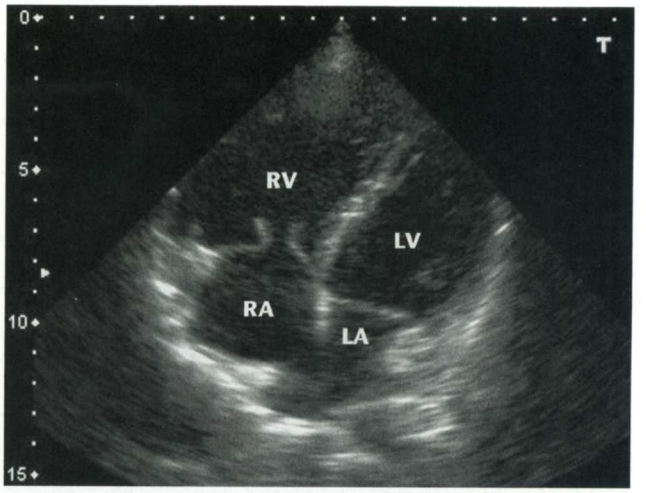

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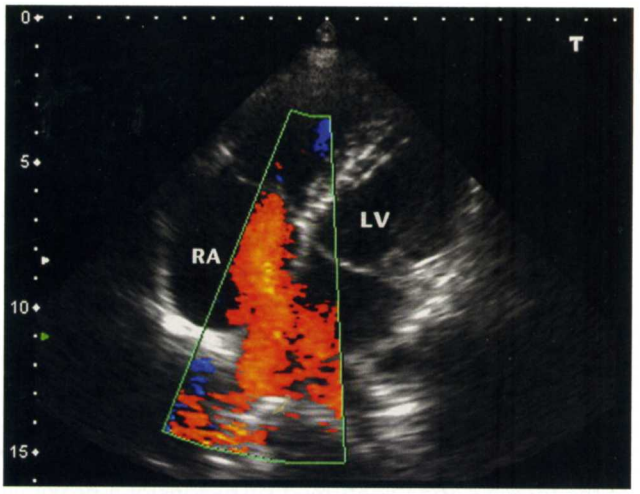

C

Figure 6-49. Case 3: Parasternal long-axis ultrasound view (A). Apical four-chamber ultrasound view (B). Color Doppler activated (C). (Courtesy of Hennepin County Medical Center)

Commentary

Case 2 exemplified the utility of routine bedside echocardiography by emergency physicians during the evaluation of nonspecific chest pain. This patient's aortic dissection may have caused a myocardial infarction, aortic valve failure, cardiac tamponade, or death had it not been identified in the emergency department. Patients with aortic dissection often present without the classic red flags. Bedside echocardiography performed by emergency physicians can provide essential information to help expedite the disposition.

CASE 3

Patient Presentation

A 21-year-old woman presented to the emergency department with 3 days of cough, sore throat, and chest pain. She had mild dyspnea on exertion while working in a restaurant. The chest pain was sharp, bilateral, and increased with coughing. Further review of systems was negative. She denied any past medical history. Oral contraceptives were her only medication. She smoked one to two cigarettes per day.

On physical examination, blood pressure was 110/67 mmHg; heart rate, 96 beats per minute; respiratory rate, 20 breaths per minute; temperature, 97°F; and oxygen saturation 97% on room air. The patient was in no distress. Head, neck, pulmonary, abdominal, and back examinations were unremarkable. There was no murmur appreciated on the initial cardiac examination.

Management Course

Laboratory and imaging studies ensued, with particular concern for possible PE. ECG showed a normal sinus rhythm at 78 beats per minute with a small R wave in lead V_1. Initial cardiac enzymes and a D-dimer were in the normal range. The patient's chest radiograph was

unremarkable. The tentative diagnosis was chest wall pain with a viral upper respiratory infection. Bedside echocardiography by the emergency physician showed a markedly enlarged right ventricle on the parasternal long axis view (Figure 6-49A). The apical four-chamber view revealed a dilated right side and an obvious large atrial-septal defect (Figure 6-49B). Color Doppler showed significant left-to-right flow through the defect (Figure 6-49C). The patient was admitted to the cardiology service and had a comprehensive echocardiographic study that was consistent with the emergency physician's findings. She underwent elective surgical repair of the atrial septal defect via thoracotomy and recovered well.

Commentary

This case demonstrated a common finding of right-sided chamber enlargement and a rare incidental finding of an atrial septal defect. The right-sided enlargement was straightforward to detect and apparent on two different views. Right-sided enlargement can be caused by PE, chronic pulmonary disease, and structural heart defects. If only the right-sided enlargement had been detected, it would have prompted further testing and the correct diagnosis would have eventually been made.

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