The conventional upright posteroanterior (PA) and lateral x-ray projections of the chest are obtained with high kilovoltage technique at maximal inspiration to permit short exposure times, which freeze cardiac motion. Interstitial markings are accentuated on a poor inspiratory effort film. A tube-to-film distance of at least 6 feet minimizes distortion and magnification.

Chest radiographs that show cardiac abnormality are a very important part of cardiology examinations.

Always take a systematic approach to reading chest radiographs. Always identify the border-forming structures of the heart on both the frontal and lateral views. Use the pulmonary blood vessels to help explain all abnormal contours.

Always try to compare a chest radiograph with any available previous study.

In a postoperative patient, suspect new abnormalities on chest films to be related to the surgical procedure.

If a CT or MRI scan is shown on the Cardiology Boards, look carefully for pericardial or aortic disease.

Cardiac Silhouette/Chambers

The image of the heart and great vessels on the chest radiograph is a two-dimensional display of dynamic three-dimensional structures (Fig. 1-10). The cardiovascular silhouette varies not only with the abnormality but also with body habitus, age, respiratory depth, cardiac cycle, and position of the patient.

Posteroanterior (PA) Projection

The right mediastinal contour consists of a straight upper vertical border formed by the superior vena cava and a smooth convex lower cardiac contour formed by the right atrium. Occasionally, a short segment of inferior vena cava may be seen where the right atrium meets the diaphragm.

The normal left mediastinal contour is formed by a series of convexities: from superior to inferior, the aortic knob, the pulmonary trunk, and the left ventricle abutting the diaphragm. Rarely, the left atrial appendage can be projected between the pulmonary trunk and the left ventricle in the normal heart, primarily in young females. The shape of the pulmonary trunk segment varies with age and body habitus. Most frequently, this segment is only slightly convex; however, it can be prominent in women 20 to 40 years old and straight or even concave in older patients and still be within normal limits. Occasionally, the cardiophrenic junction of the cardiac silhouette is not formed by the left ventricle but by a fat pad. Less common is a border-forming fat pad in the right cardiophrenic angle which should not be confused with a cardiac mass.
Fig. 1. PA projection of the heart. SVC, superior vena cava.

Fig. 2. Lateral projection of the heart.

Fig. 3. Magnetic resonance imaging of the heart in the frontal plane. AO, aorta; LV, left ventricle; PA, pulmonary artery; RA, right atrium.

Fig. 5. Angiogram demonstrating the relative positions of the right heart chambers on the PA projection. *LPA*, left pulmonary artery; *RA*, right atrium; *RPA*, right pulmonary artery; *RV*, right ventricle.

Fig. 6. Angiogram demonstrating the drainage of the pulmonary veins into the left atrium. *LA*, left atrium; *PV*, pulmonary vein.

Fig. 7. Angiogram demonstrating the relative positions of the right heart chambers on the lateral projection. *LPA*, left pulmonary artery; *MPA*, main pulmonary artery; *RA*, right atrium; *RPA*, right pulmonary artery; *RV*, right ventricle.

Fig. 8. Magnetic resonance image of the heart demonstrating the close anatomical relationship of right and left atria (*RA* and *LA*), aorta (*AO*), and pulmonary artery (*PA*). *IVC*, inferior vena cava.
Fig. 9. Angiograms demonstrating the relative position of the left heart chambers on frontal (A) and lateral (B) projections. *AO*, aorta; *LA*, left atrium; *LV*, left ventricle.

Fig. 10. Prosthetic Starr-Edwards valves as seen on frontal (A) and lateral (B) projections. *AVR*, aortic valve prosthesis; *MVR*, mitral valve prosthesis; *TVR*, tricuspid valve prosthesis.
A normal left atrial appendage may be seen projecting between the pulmonary trunk and left ventricle, especially in young females.

The left cardiophrenic junction may be formed by a fat pad and give a false impression of cardiomegaly.

**Lateral Projection**

It is routine that the patient's left side is positioned against the film cassette to minimize distortion of the heart due to geometric magnification. Superiorly, the anterior border is formed by the ascending aorta posterior to the retrosternal air space; inferiorly, the right ventricle and right ventricular outflow tract abut the sternum and blend into the main pulmonary artery, which then courses posteriorly to its bifurcation. The posterior cardiac contour is formed by the left atrium superiorly beneath the carina and the left ventricle curving inferiorly to the diaphragm, where the straight vertical edge of the inferior vena cava is often apparent within the thorax as it enters the right atrium.

**Heart Size on Chest Radiographs**

The cardiothoracic ratio (CTR)—the ratio of the transverse cardiac diameter to the maximal internal diameter of the thorax at the level of the diaphragm on an upright PA chest radiograph—corrects for body size and magnification produced by slight differences in radiographic techniques. In adults, a CTR greater than 0.5 is considered to represent cardiomegaly. In aortic regurgitation, the left ventricle is often enlarged downward rather than horizontally. A high diaphragm position, as seen with obesity or shallow inspiration, will produce an erroneous CTR greater than 0.5. Pectus excavatum and the absence of pericardium displace the heart posteriorly and rotate the apex laterally, resulting in a CTR greater than 0.5 in the presence of a normal-sized heart. Large pericardial fat pads may give a falsely increased CTR. Because of these factors, one can be misled if relying on the CTR alone to diagnose cardiomegaly; however, it does serve as a baseline for future comparisons.

A CTR > 0.5 with a normal heart size occurs with
- Absent pericardium
- Pectus excavatum
- Obesity
- Poor inspiration

**Generalized Cardiac Enlargement**

Global heart enlargement, with maintenance of an otherwise normal cardiac contour, usually is due to diffuse myocardial disease, abnormal volume or pressure overload as a consequence of valvular heart disease, hyperthyroidism, hypothyroidism, or anemia. Pericardial effusions also produce generalized enlargement of the cardiac silhouette (Fig. 11). Asymmetric enlargement with left ventricular prominence can be seen in the late stages of essential hypertension and other left-sided obstructive lesions with secondary left ventricular failure or in left-sided regurgitant valvular lesions (Fig. 12).

**Left Atrial Enlargement**

The left atrium sits just below the angle of the carina, in proximity with the left bronchus and esophagus; thus, enlargement is readily reflected by the displacement of these neighboring structures. Enlargement usually produces a double density behind the right atrial margin on a frontal projection as the left atrium bulges out from the mediastinum into the right lung. Occasionally, a double density can be seen in the presence of a normal-sized left atrium in patients with a prominent right pulmonary venous confluence.

Additional signs of left atrial enlargement on the PA projection include upward and posterior displacement of the left main bronchus, resulting in a less acute carinal angle. Enlargement of the left atrial appendage initially causes straightening and, subsequently, a convexity in the upper left cardiac contour. In the presence of a giant left atrium, the left atrium itself may project beyond the right atrium and form a portion of the right cardiac contour. On the
Isolated left atrial enlargement most commonly is due to mitral valve stenosis caused by rheumatic heart disease (Fig. 13 and 14). Left atrial myxoma and cor triatriatum can also cause isolated left atrial enlargement. Isolated enlargement of the left atrial appendage or apparent enlargement due to a pericardial defect and focal herniation of the appendage may cause a localized bulge in the upper left cardiac contour without other signs of left atrial dilatation. Left atrial enlargement in combination with additional chamber involvement may be produced by various conditions, such as left ventricular failure, left-sided obstructive lesions, and certain shunts (e.g., ventricular septal defect, patent ductus arteriosus, and aortopulmonary window). However, left atrial enlargement is not seen with simple atrial septal defects. When left atrial enlargement is marked, it most often is due to rheumatic valvular disease.

- Isolated left atrial enlargement—
  - Mitral valve disease
  - Occasionally, cor triatriatum or left atrial myxoma
- Left atrial enlargement does not occur with simple atrial septal defects.

**Left Ventricular Enlargement**

Left ventricular enlargement can be due to dilatation or hypertrophy or both. Considerable hypertrophy must be present to cause the cardiac shadow to enlarge appreciably. The classic appearance of left ventricular hypertrophy...
on the PA projection is rounding of the cardiac apex, with downward and lateral displacement without cardiac enlargement. Left ventricular dilatation causes an increase in the transverse diameter of the heart and CTR, together with an apparent increase in the length of the left heart border. The cardiac apex may be displaced to the extent that it projects below the diaphragm. On the lateral projection, dilatation increases the posterior convexity of the left ventricular contour, which will project behind the edge of the vertical inferior vena cava. Obstruction to left ventricular emptying or increased afterload, as caused by systemic hypertension, aortic coarctation, or aortic valve stenosis, leads to hypertrophy initially, with rounding of the cardiac apex (Fig. 15). Left ventricular dilatation with cardiac failure may follow. Dilated cardiomyopathy, especially ischemic cardiomyopathy, primarily enlarges the left ventricle. Aortic valve regurgitation and mitral valve regurgitation enlarge the left ventricle and are associated with dilatation of the aorta and left atrium, respectively. Left ventricular aneurysms, usually the result of a previous myocardial infarction, occasionally result in a localized bulge that projects beyond the normal ventricular contour or an angulation of the left ventricular contour (Fig. 16). A large apical aneurysm can appear similar to simple left ventricular chamber dilatation. Sometimes with true aneurysms of the left ventricle, the heart appears normal in size and contour. False aneurysms often are paracardiac in location, posterior and inferior to the left ventricle. All cardiac chambers have been reported to be involved with aneurysm formation, although atrial aneurysms are extremely rare.

- In the absence of heart failure, left ventricular hypertrophy must be massive before the heart shadow enlarges.

**Right Atrial Enlargement**

Isolated right atrial enlargement is detected best on a frontal film. Enlargement is to the right and causes increased fullness and convexity of the right cardiac contour and angulation of the junction of the superior vena cava and right atrium. There may be associated dilatation of the superior and inferior venae cavae that causes widening of the right superior mediastinum and an additional border in the right cardiophrenic angle. On the lateral projection, right atrial dilatation is often difficult to appreciate. It causes a “filling-in” of the retrosternal clear space anteriorly and superiorly, with the cardiac silhouette extending behind the sternum more than one-third the way above the cardiophrenic angle, similar to that seen with right ventricular enlargement. There may be a double density that merges with the inferior vena caval shadow, which may be a slightly convex structure. Left atrial enlargement can be simulated by marked right atrial dilatation.
Fig. 15. A, PA and, B, lateral projections of an enlarged left ventricle with dilatation of the ascending aorta due to combined aortic insufficiency and aortic stenosis. The aortic valve is calcified (arrows) and the pulmonary arteries are enlarged in this patient, who also has chronic obstructive pulmonary disease.

Fig. 16. A, PA projection showing marked enlargement of the left ventricle due to left ventricular aneurysm. B, Curvilinear calcification outlines the aneurysm (arrows).
Right atrial enlargement “fills in” the retrosternal clear space on the lateral projection.

Isolated right atrial enlargement is uncommon and usually is due to tricuspid stenosis or right atrial tumor. Right atrial dilatation associated with other chamber enlargement, primarily right ventricular enlargement, can be seen in several conditions, such as tricuspid regurgitation, pulmonary arterial hypertension, shunts to the right atrium, and cardiomyopathies (Fig. 17 and 18). Marked isolated right atrial enlargement resulting in a “box-shaped” heart is seen in Ebstein’s malformation of the tricuspid valve (Fig. 19). This configuration of the heart is the result of marked angulation at the superior vena caval-right atrial junction as the right atrium enlarges.

- Ebstein’s anomaly causes a “box-shaped” heart.

**Right Ventricular Enlargement**

The right ventricle enlarges by broadening its triangular shape in the superior and leftward direction. With increasing right ventricular enlargement, the entire heart rotates to the left around its long axis and displaces the left ventricle posteriorly. This displacement causes increased convexity of the left upper heart border and elevation of the cardiac apex. The rotation also makes the pulmonary trunk appear relatively small. With marked dilatation, the right ventricle may form the left heart border on the PA projection.

On the lateral projection, the right ventricle extendscranially behind the sternum, with increased bulk anteriorly. Normally, the heart does not extend more than one-third of the distance from the cardiophrenic angle to the sternal angle or the level of the carina; however, normal extension can vary with body habitus. Isolated right ventricular enlargement

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Fig. 17. Marked right atrial dilatation and right ventricular dilatation due to severe tricuspid regurgitation related to traumatic injury of the tricuspid valve.

Fig. 18. A, PA and, B, lateral projections showing combined mitral stenosis and mitral insufficiency resulting in left atrial enlargement, marked right ventricular enlargement, and slight left ventricular enlargement. The dilated ventricles (arrows) are appreciated best on the lateral view.
is very unusual. More typically, there is associated prominence of the right atrium and pulmonary trunk.

**Pulmonary Vasculature**

Because the pulmonary vasculature reflects the physiologic effects of a cardiac lesion, it provides important clues to the diagnosis. Radiographic abnormalities are primarily the result of an increase in pulmonary blood flow or an obstruction to flow somewhere in the pulmonary circuit.

**Normal Pulmonary Blood Flow**

The pulmonary arteries and veins extend outward from each hilum in an orderly branching fashion, with gradual tapering peripherally. The hilar density is composed of the proximal pulmonary arteries, with the left hilum normally projecting more cranially than the right one because of the course of the left pulmonary artery over the left main bronchus. In the upper lobes, the veins and arteries are essentially parallel, with the veins lying lateral to their corresponding arteries. The major arteries and veins in the lower lung fields cross each other, with the veins taking a more horizontal course toward the left atrium. In the upright position, there is increased flow to the base of the lungs (largely due to the effects of gravity), which causes the lower-lobe vessels to increase in size. It may be difficult to identify the apical vessels clearly because pulmonary flow to the apices is negligible in the upright position. Therefore, position has a marked effect on flow distribution.

**Increased Pulmonary Blood Flow**

As pulmonary flow increases, the pulmonary vessels, both arteries and veins, become enlarged. These enlarged vessels become apparent when pulmonary flow is approximately twice normal. The “over-circulation” pattern may be symmetric or asymmetric. High-output states with increased circulating blood volume, such as anemia, pregnancy, thyrotoxicosis, overhydration, and fever, result in a symmetric increase in vascularity, as do various congenital defects characterized by left-to-right shunts (Fig. 20 and 21). An asymmetric increase in pulmonary flow may be congenital in origin (e.g., pulmonary arteriovenous malformation, anomalous origin of a pulmonary artery) but is more commonly the result of surgical intervention to create a systemic-to-pulmonary shunt to improve pulmonary blood flow in the presence of severe pulmonary stenosis or atresia (e.g., a Blalock-Taussig shunt).

**Decreased Pulmonary Blood Flow**

Essentially all the linear shadows in the normal lung fields are due to pulmonary vasculature. When flow and, therefore, vessel size are diminished, the lung fields appear abnormally radiolucent. Both symmetric and asymmetric patterns of abnormal vascularity can be observed. Generalized undercirculation can be due to an obstructive lesion in the right heart, as in tetralogy of Fallot, pulmonary atresia, right ventricular tumor, or tricuspid valve atresia. Small-caliber pulmonary vessels with relatively hyperlucent lungs and a small heart are evidence of a marked decrease in the circulating blood volume (e.g., in Addison disease, hemorrhage). Chronic obstructive pulmonary disease (COPD) may result in generalized lung destruction or, more commonly, a patchy distribution of decreased vascularity. Segmental and asymmetric decreases in pulmonary vascularity are seen with pulmonary embolic disease (Westermark sign), segmental COPD, partial pneumonectomy, and branch pulmonary artery stenoses (Fig. 22). Rarely, postinflammatory changes (e.g., granulomatous mediastinitis), extrinsic compression (e.g., aortic aneurysm), and congenital hypoplasia as seen in the scimitar syndrome result in areas of decreased pulmonary flow. Bronchial collateral circulation may become prominent, with a somewhat disordered pattern, when there is a decrease in
pulmonary artery blood flow, and occasionally, it gives the
illusion that the overall vascularity is actually normal or even
increased. Small hila in tetralogy of Fallot or pulmonary atresia
and loss of the normal branching pattern of pulmonary
vasculature should be evident on the chest radiograph.

If the lung hila are small, consider tetralogy of Fallot or
pulmonary atresia.

**Increased Resistance to Pulmonary Blood Flow**
Pulmonary hypertension with redistribution of flow is the
result of increased resistance in the pulmonary circuit.
Recognition of the various redistribution patterns seen on
chest radiographs often allows the level of the increased
resistance and the possible underlying abnormality to be
identified.

**Pulmonary Venous Hypertension**
Lesions acting beyond the pulmonary capillary level result
in elevation of the pulmonary venous pressure. Left ventricular
dysfunction and mitral valve disease are the most common
causes of pulmonary venous hypertension; other obstructive
lesions at the left atrial level (e.g., atrial myxoma, cor triatriatum, thrombus) or pulmonary vein level (e.g., stenosis,
veno-occlusive disease, or thrombosis) are relatively rare.

Initially, because of the increase in venous pressure,
venous dilatation occurs throughout the lungs. However,
the radiographic pattern typically seen is that of prominent upper lung vessels, both arteries and veins. This phenomenon is thought to be due to a localized segmental reflex initiated by the increase in pulmonary venous pressure above a critical level of about 10 to 15 mm Hg. An additional factor is the accumulation of fluid around compressible small vessels when plasma oncotic pressure is exceeded by pulmonary venous pressure. When a person is in the upright position, the pressure in the lower lung is greater because of hydrostatic forces; therefore, vasoconstriction of both arteries and veins occurs here first and increases resistance to flow, thereby reducing the circulatory volume through these vessels. To overcome the increased resistance and to maintain a gradient in the presence of increased pulmonary venous pressure, the pulmonary artery pressure must increase, resulting in increased flow to the apices. The diverted pulmonary flow increases the size and visibility of the upper-lobe vessels (Fig. 23). As pulmonary venous hypertension increases to the order of 25 mm Hg, there is increased transudation of plasma from the lower lung capillaries that results in interstitial edema. In addition to obscuring further the now smaller and crowded lower-lobe vessels, this transudation results in the radiographic appearance of septal lines (Kerley lines), which are due to fluid within the interlobular septa (Fig. 24). Still further increase in pulmonary venous pressure results in transudation of plasma into the alveoli, producing classic alveolar edema when the pressure exceeds 30 mm Hg.

Fig. 22. A, PA projection showing decreased vascular markings in both lungs, most marked in the right upper lobe. B, The angiogram demonstrates large bilateral emboli, resulting in little flow to the right upper lobe and left lower lobe.

Fig. 23. A-D, Serial radiographs demonstrating development of pulmonary venous hypertension continuing on to florid pulmonary edema in a patient with a large myocardial infarction. Note the progressive redistribution of the prominence of the pulmonary vessels to the right upper lobe. The hilar vessels become much less distinct as the edema develops.
Pulmonary Arterial Hypertension

Increased resistance at the pulmonary capillary or arteriolar level increases pulmonary artery pressure. The causes of pulmonary arterial hypertension include 1) obstructive processes (e.g., chronic pulmonary emboli, idiopathic or primary pulmonary arterial hypertension, pulmonary schistosomiasis), 2) obliterative processes (e.g., pulmonary fibrosis, COPD), 3) constrictive processes (e.g., chronic hypoxia), and 4) increased flow as seen in large left-to-right shunts with development of Eisenmenger syndrome (Fig. 25 and 26). Radiographically, pulmonary arteries are dilated centrally, with an abrupt disparity in the caliber of the central and intrapulmonary arteries or “pruning” of the intrapulmonary branches. This uneven response is thought to be due to constriction of the muscular intrapulmonary branches in response to the increased intraluminal pressures, with dilatation of the more elastic central arteries.

- The classic chest radiographic findings in pulmonary arterial hypertension are dilated distal proximal pulmonary arteries and “pruning” of intrapulmonary branches.

Pericardial Disease

Normal pericardium is seldom identified on plain chest radiographs. It may be visible as a sharp line at the cardiac apex, outlined by epicardial and mediastinal fat.

Pericardial Effusion

A pericardial stripe wider than 2 mm that parallels the lower heart border, usually in the lateral projection and best identified in the sternophrenic angle, is diagnostic of a pericardial effusion. The only clue to a relatively small effusion may be a noticeable change in heart size compared with that on previous films. The classic “water flask” configuration of a large effusion may not be present, and the appearance of the cardiac silhouette may be identical to that in dilated cardiomyopathy with no significant distortion other than enlargement. A large heart with a prominent superior vena cava and azygos vein in combination with decreased pulmonary vasculature should raise the question of cardiac tamponade. Acutely, a relatively small effusion can cause tamponade with minimal enlargement of the cardiac shadow.

Pericardial Calcification

Constrictive pericarditis may occur as the end result of pericarditis and pericardial effusion of any cause. Calcification of the pericardium is highly suggestive but not pathognomonic of constrictive pericarditis. More than 50% of patients with constrictive pericarditis do not show calcifications on the plain chest film. Calcifications are found frequently on the anterior and diaphragmatic surfaces, but they may be over any part of the heart. Linear or plaque-like calcifications, often best seen on the lateral view, are typically projected over the right ventricle or the atrioventricular groove (Fig. 27). The entire heart may appear encased in a shell. The calcification may be quite dense and thick.

- More than 50% of patients with constrictive pericarditis do not show pericardial calcification.

Pericardial Defects

Congenital or surgical absence of the pericardium may result in changes in the cardiac contours. Congenital absence is more commonly left-sided and rarely right-sided. Partial defects may allow a portion of the heart (usually the left atrial appendage in congenital defects) to herniate outside the pericardial sac, with the herniated portion producing a bulge in the contour of the heart. “Complete” absence of the
Fig. 25. Pulmonary hypertension due to chronic pulmonary emboli. Note the central pulmonary artery enlargement (arrows) (A) and right ventricular dilatation seen best in the lateral projection (B). The angiogram demonstrates classic arterial occlusions and stenoses (arrowheads) (C).

Fig. 26. Marked enlargement of the pulmonary arteries centrally secondary to Eisenmenger syndrome caused by long-standing atrial septal defect.
Fig. 27. A and B, Plain films showing constrictive pericarditis with circumferential pericardial calcifications (arrows). Note the pulmonary venous hypertension and right pleural effusion. C, Computed tomographic image through the mid ventricles better demonstrates the circumferential nature of the relatively coarse calcifications.
pericardium is actually a unilateral defect and nearly always left-sided. The heart appears shifted to the left without a shift in the trachea (Fig. 28). The left cardiac contour has an elongated appearance. The pulmonary artery often appears prominent and sharply defined. A somewhat similar appearance is seen on the frontal projection when the heart is rotated because of compression of the chest wall in patients with pectus excavatum deformity.

- Partial or “complete” absence of the pericardium is usually left-sided.

**Cardiac Masses**

The role of plain chest radiographs in the identification of cardiac masses is often limited. Radiologic manifestations are dependent on tumor size and location as well as type. With many intracavitary and intramural tumors of even moderate size, no changes are seen on plain films unless hemodynamic alterations are produced, such as the mimicking of mitral stenosis by a left atrial myxoma. Left ventricular aneurysms, pericardial cysts, extracardiac mediastinal masses, loculated pericardial cysts, and loculated pericardial effusions are all causes of abnormal contours that can be indistinguishable from neoplasms (Fig. 29 and 30). The presence of calcification may help in the detection of a mass, but calcification patterns are not specific, and differentiation from calcification of thrombus or normal structures usually requires additional imaging modalities.

**Aortic Disease**

The aortic knob, representing the foreshortened transverse aortic arch, is the only border-forming portion of the normal thoracic aorta that is otherwise hidden within the mediastinum. The descending thoracic aorta parallels the thoracic spine on the left. With the development of atherosclerotic aortic disease, unfolding and ectasia (dilatation and elongation) of the aorta occur. As the descending aorta swings into the left chest, more and more of the contour becomes silhouetted by lung; on the lateral projection, a portion of the descending aorta may be demonstrated, and only then is a clue to the presence of an aneurysm obtained. Unfolding or ectasia of the ascending aorta produces a convexity of the right superior mediastinum. These findings may be indistinguishable from those present with an aortic aneurysm. The most common finding of an aortic aneurysm on a frontal chest radiograph is widening of the superior mediastinum (Fig. 31). Other chest film findings suggestive of a thoracic aortic aneurysm, whether atherosclerotic, luetic, dissecting, or traumatic, include displacement or compression (or both) of the trachea and esophagus either to the left and posteriorly by an aneurysm of the ascending aorta or to the right and anteriorly by an aneurysm of the descending aorta. Calcification in the aorta is a common finding in atherosclerotic aortic disease. Because the aorta is largely hidden by the mediastinal silhouette, the cross-sectional modalities, such as computed tomography and magnetic resonance imaging, provide greater detail in the evaluation and follow-up of aortic disease (Fig. 32-34).
Fig. 29. A, Mass (arrows) in right cardiophrenic angle consistent with a prominent cardiac fat pad. B, The high signal of adipose tissue (arrows) is demonstrated with magnetic resonance imaging of this region.

Fig. 30. A, PA and, B, lateral projections showing a well-defined rounded mass projected adjacent to the right atrium. The heart and pulmonary vasculature are otherwise normal. Appearance is typical of a pericardial cyst.
Fig. 31. A, PA and, B, lateral projections showing marked enlargement of the aortic knob and widening of the mediastinum due to a large ascending aortic aneurysm. The size of the ascending aorta is appreciated better on the lateral view. The patient has significant aortic regurgitation with bilateral pleural effusions.

Fig. 32. A, PA projection showing marked enlargement of the ascending aorta caused by a dissecting aneurysm. B, This is appreciated better on a computed tomographic scan. Note discrepancy in size between the ascending aorta (arrows) and the main pulmonary artery (arrowheads).
Fig. 33. A, Enlargement of the aortic arch containing curvilinear calcification (arrow). B, Computed tomographic scan demonstrates better the small pseudoaneurysm containing peripheral calcification (arrows), the result of remote trauma.

Fig. 34. A, Double contour to the aortic arch typical of coarctation. B, A magnetic resonance image nicely demonstrates the focal narrowing and resulting kinking (arrows) of the proximal descending aorta.
Questions

Multiple Choice (choose the one best answer)

1. These two patients have the same condition. What is it?
   a. Left ventricular aneurysms
   b. Normal chest radiographs, but the patients are poorly positioned for the studies
   c. Absent pericardium
   d. Dilated cardiomyopathy
   e. Pulmonary hypertension

2. What is the diagnosis?
   a. Calcified left ventricular aneurysms
   b. Left atrial calcification due to mitral stenosis
   c. Dense mitral annulus calcification
   d. Pericardial calcification
   e. Calcified pleural plaques
3. These four patients all have the same condition. What is it?
   a. Ventricular septal defect
   b. Atrial septal defect
c. Patent ductus arteriosus
d. Chronic pulmonary emboli
e. Left ventricular failure with pulmonary venous hypertension
4. What is the diagnosis?
   a. Type A dissection
   b. Type B dissection

5. Which chamber is massively enlarged?
   a. Left ventricle
   b. Right ventricle
   c. Left atrium
   d. Right atrium

   c. Traumatic aortic transection
d. Syphilis
e. Penetrating aortic ulcer
6. This patient has radiographic findings of which of the following conditions?
   a. Mitral stenosis
   b. Aneurysm of the left atrial appendage
   c. Left atrial myxoma
   d. Cor triatriatum

7. What is the diagnosis?
   a. Normal findings
   b. Partial anomalous return of the right upper lobe pulmonary vein
   c. Scimitar syndrome
   d. Arteriovenous fistula
   e. Total anomalous venous return below the diaphragm
8. What is the diagnosis?
   a. Coarctation of the aorta
   b. Pseudocoarctation of the aorta
   c. Type IV truncus arteriosus
   d. Chronic aortic dissection
   e. Arteriovenous malformation of the aorta

9. What is the diagnosis?
   a. Ventricular septal defect
   b. Atrial septal defect
   c. Pulmonary stenosis
   d. Coarctation of the aorta
   e. Patent ductus arteriosus
10. What is the diagnosis?
   a. Ventricular septal defect
   b. Patent ductus arteriosus
   c. Pulmonary stenosis
   d. Coarctation of the aorta
   e. Truncus arteriosus

Questions

**Question 10**

**Answers**

1. **Answer c**
   The heart is shifted to the left but is normal size. The patient is properly positioned. Pectus excavatum also causes this appearance.

2. **Answer d**
   Dense coarse calcification is seen surrounding the heart.

3. **Answer b**
   Four classic examples of patients with atrial septal defect of different severity and duration.

4. **Answer b**
   The dissection flap is not present in the ascending aorta.

5. **Answer b**
   Note how unimpressive the chest radiograph is compared with this patient’s computed tomographic scan. The left ventricle is compressed, with flattening of the interventricular septum.

6. **Answer b**
   This patient has an aneurysm of the left atrial appendage. There is no enlargement of the body of the left atrium and no evidence of pulmonary venous or arterial hypertension. The chambers on the right side are not enlarged. Partial absence of the pericardium with a herniated left atrial appendage can also look like this and would be in the differential diagnosis for this patient.
7. **Answer c**
   The chest radiograph and pulmonary angiograms show the classic findings of scimitar syndrome, namely, anomalous connection of the right pulmonary vein(s) to the inferior vena cava, hypoplasia of the right lung, anomalous systemic pulmonary arterial blood supply to the right lung from the aorta, and bronchial abnormalities.

8. **Answer a**
   Coarctation of the aorta. The magnetic resonance image on the right shows the marked narrowing of the descending aorta, with the development of large intercostal collateral vessels. The middle image shows rib notching, a classic radiographic feature of coarctation of the aorta caused by inferior rib erosion due to large collateral intercostal arteries.

9. **Answer e**
   The aorta is prominent along with dilated pulmonary arteries, shunt vascularity, and an enlarged left ventricle.

10. **Answer d**
    The chest radiograph shows an abnormal contour of the aortic arch with a localized indentation at the site of the coarctation to give the “3” sign. Rib notching is absent in this case. The upper curve of the “3” is formed by the left subclavian artery, and the lower half of the curve of the “3” is formed by the post-stenotic dilatation of the aorta.