Echo Pericardium, Tamponade and Constriction

September 1, 2009
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Pericardial Anatomy
Pericardium Anatomy and Physiology

- Visceral pericardium is serous membrane, single cell layer of mesothelial cells adherent to epicardium
- Parietal pericardium is fibrous, 2mm thick post mortem, largely acellular, more collagen and some elastin – attached to diaphragm, sternum and other anterior mediastinal structures; envelops the phrenic nerves
- Pericardial space normally contains up to 50 ml serous fluid
Figure 1. A, Pericardial specimen from patient with normal pericardial thickness. Pericardial dimension is \( \approx 1.5 \) mm in depth with 3 mm of fat on exterior surface of pericardium. B, Pericardial specimen from patient with increased pericardial thickness. Pericardial dimension is \( \approx 7 \) mm in depth with fat on exterior surface of pericardium (both specimens, hematoxylin and eosin, original magnification \( \times 10 \)).
**Pericardial Physiology**

**Visceral** pericardium is a serous membrane of single layer of epicardial mesothelial cells

**Parietal** pericardium (2 mm thick)
- fibrosa
- serosa

Fibrosa: mainly dense collagen (some elastin), anisotropic, with fibers oriented in three layers at equal angles

Normal human pericardium has about 50 ml fluid

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From **Hurst**, 9th ed CD-ROM

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Pericardial pressure-volume curve (canine).

(From Holt JP. The normal pericardium. *Am J Cardiol* 1970; 26:455. Reproduced with permission from the publisher and author.)
Anisotropy of Pericardium

Braunwald, 7th ed.

Relationship between stretch and tension in vitro in normal human pericardial tissue. The tissue has been stretched in two, mutually orthogonal directions (X, Y). Note the relatively abrupt transition from a relatively flat to a steep, inelastic relationship. In addition, the tissue is anisotropic; i.e., the relation between tension and stretch depends on the direction of stretch. (From Lee M-C, Fung YC, Shabetai R, LeWinter MM: Biaxial mechanical properties of the human pericardium and canine comparisons. Am J Physiol 22:H75, 1987.)
Adaptability/Remodeling of Pericardium

Pressure-volume relationship of the normal canine pericardium (left) and after 4 weeks of cardiac dilation caused by volume overload (right). Note the relatively abrupt transition to a steep relationship in normal pericardium and marked shift to the right and flattening after chronic volume overload. (From Freeman G, LeWinter M: Pericardial adaptations during chronic cardiac dilation in dogs. Circ Res 54:294, 1984.)

Braunwald, 7th ed.
Pericardial Anatomy

Oblique sinus
Transverse sinus

Clemente CD. Anatomy 3rd Ed, 1987, fig 212.
Pericardial Anatomy

Westberg’s space:
The space surrounding the origin of the aorta which is invested with the pericardium.
The pericardial reflections near the origins of the great vessels shown after removal of the heart. Note that portions of the caval vessels are within the pericardial space. (From Gabella G [sect ed]: The pericardium. In Gray H, Williams PL, Bannister LH [eds]: Gray’s Anatomy: The Anatomical Basis of Medicine and Surgery. New York, Churchill-Livingstone, 1995, p 1471.)
Pericardial Recess

Veins

Oblique pericardial sinus


Sobotta Atlas, 13th Ed, 2000, vol 2, p. 77
Base of Heart
Posterior View
Heart, Anterior View
Open RA
Case Presentation (fiction)

- 60 year old woman presents to ER with progressive dyspnea the last 3 days, associated with moderate constant vague chest discomfort; nonsmoker, no prior history of cardiac or respiratory disease
- PMH: DM, Htn, HLP; Meds: Glipizide, Metformin, Atenolol, Hctz, Lisinopril, Atorvastatin; NKDA
- VS: T 98.2, BP 100/80; P 110, R 28, BMI 27
- Exam: Moderately dyspneic, anxious, JVP difficult to appreciate (“no neck”), pulse weak, RRR no M or G, trace pretribial edema, lungs clear
- CXR – lungs clear, cardiomegaly
- ECG - pending
Causes of Cardiac Tamponade

- Acute pericarditis
- Neoplasm
  - bronchogenic
  - mammary
  - lymphoma
- Radiation
- Aortic Dissection
- Trauma
  - iatrogenic
  - non-iatrogenic
- AIDS
- Post myocardial infarction
- Collagen vascular
  - RA
  - SLE
- Drugs
  - Procainamide
  - Hydralazine
- Myxedema
Causes of Acute Pericarditis and Pericardial Effusion

• Myocardial infarction or postmyocardial infarction
• Drugs
  – Procainamide
  – Cromolyn sodium
  – Hydralazine
  – Dantrolene
  – Methylsergide
• Dissection aneurysm
• Infective endocarditis with ring abscess
• Uremia
• Hypothyroidism
• Thymic cyst
Intrapericardial Pressure

- Amount of pericardial fluid
- Rapidity of accumulation of fluid
  - Very rapid accumulation of 150 ml may cause tamponade
  - Slow accumulation of 1-2 L may be well tolerated
- Distensibility of pericardium
- Distensibility of ventricular chambers
Cardiac Tamponade

Echocardiography

- Pericardial effusion
- Right ventricular diastolic collapse
- Right atrial collapse
- IVC plethora, lack of inspiratory collapse
- Exaggeration of respiratory variation in RV and LV dimensions
- Pendular swinging of heart
Echo in Pericardial Disease

- Size of effusion
  - Small – less than 1 cm maximal size
  - Moderate – 1-2 cm maximal size
  - Large - >2 cm maximal size
- Pericardial thickness not possible unless concomitant pleural effusion
- Apical 4-chamber – differentiation of pleural from pericardial effusion may be problematic
- Stranding – marked inflammatory or possibly hemorrhagic etiology (uremic, infectious)
- Masses – metastatic disease from intrathoracic malignancy but often also marked inflammatory process

From Ch. 9 in Feigenbaum’s Echocardiography, 6th ed. 2005.
Echo in Pericardial Tamponade

- **Swinging heart**, early sign, marker of very large effusion, associated with electrical alternans
- **RV Collapse** – diastole, particularly early diastole, and RVOT is more compressible, usually best in PSLA and PSSA views; tamponade is worse if body of RV affected
- **RA Collapse** – exaggerated, best in subcostal or A4C views, occurs immediately after normal atrial systolic contraction
- **LA or LV collapse** occasional or isolated (in loculated effusion)

Ch. 9, Feigenbaum’s Echocardiography, 6th ed. 2005.
Doppler in Pericardial Tamponade

- Respiratory variation in LVIT is normally 15% or more, and RVIT 25% or more, and LVOT and RVOT TVI less than 10%
  - These are exaggerated in tamponade
- SVC (and hepatic vein) flow normally is S and D and nearly continuous – these findings are generally supportive but not diagnostic as a stand-alone finding
  - Tamponade, D is truncated
  - Hepatic flow may show exaggerated respiratory dependency

Ch. 9, Feigenbaum’s Echocardiography, 6th ed. 2005.
Doppler in Pericardial Tamponade

- Earliest: exaggerated RVIT respiratory variation
- Then LVIT
- RA collapse more sensitive than RVOT collapse, then RV free wall collapse
  - RVOT collapse may only be seen in expiration in early tamponade (collapse may be seen in large pleural effusions)
- False negative: RVH usually from pulmonary hypertension, RV MI, ASD
- Low pressure tamponade as in hypovolemic patients may have atypical findings

Ch. 9, Feigenbaum’s Echocardiography, 6th ed. 2005.
Echo-guided Pericardiocentesis

- "Two-dimensional echocardiography-guided pericardiocentesis has been the procedure of choice for all forms of diagnostic and therapeutic pericardiocentesis at the Mayo Clinic since 1980."
- 1000 procedures
- 1 death
- 8 serious complications
Comparison of RA and Peri pressure in 50 consecutive patients with RA or RV diastolic collapse on Echo.

Notice wide range of intrapericardial pressures, but usual occurrence of equalization.
Figure. Effusive-Constrictive Pericarditis.

As illustrated in Panel A, the presence of pericardial fluid causes tamponade, and a thickened visceral pericardium (epicardium) causes constriction. Pressure tracings (Panel B) show marked and equal elevations of the pericardial and right atrial pressures before the removal of fluid; after fluid removal, the pericardial pressure is normal (increasing and decreasing with respiration), but the right atrial pressure remains elevated, indicating that there is still constriction caused by the visceral pericardium.
**FIGURE 64-4** Beat-to-beat changes in pulmonary arterial and aortic stroke volume (as percentage of control) following abrupt production of cardiac tamponade (at arrow). Note that pulmonary arterial stroke volume decreases immediately, but there is a brief lag before aortic stroke volume decreases. Pulmonary arterial stroke volume is lower than aortic stroke volume until a new steady state is reached. (From Ditchey R, Engler R, LeWinter M, et al: The role of the right heart in acute cardiac tamponade in dogs. Circ Res 48:701, 1981.)
FIGURE 64-6A  A, Schematic illustration of leftward septal shift with encroachment of left ventricular volume during inspiration in cardiac tamponade. B, Respiration marker and aortic and right ventricular pressure tracings in cardiac tamponade. Note paradoxical pulse and marked, 180 degrees out of phase respiratory variation in right- and left-sided pressures. Ao = aortic pressure; ECG = electrocardiogram; Exp = expiration; Insp = inspiration; RV = right ventricular pressure. (From Shabetai R: The Pericardium. New York, Grune & Stratton, 1981, p 266.)
**Figure 1.** Angiographic diastolic pressures in eight patients (in mm Hg). RA a = right atrium a wave; RV dia = right ventricular diastolic pressure; PA dia = pulmonary artery diastolic pressure; PCWP mean = mean pulmonary capillary wedge pressure; LV dia = left ventricular diastolic pressure.
Chest X-ray in pericardial effusion:

Young patient with acute pericarditis with effusion. Water bottle-shaped cardiomegaly, clear lungs and normal pulmonary vascularity.

Panel B: 5 days later after therapy.
Chest X-ray in pericardial effusion:

Panel A: Hairlike normal pericardium sandwiched between 2 fat planes (epicardial fat stripe and mediastinal fat stripe). Normal is <2mm.

Panel B: 2 weeks later, moderate pericardial effusion, now >1cm.

From Hurst’s The Heart, 9th ed, 1998, p. 399
Anteroposterior chest radiograph of a patient with a large pericardial effusion (see text). (From Kabbani SS, LeWinter M: Cardiac constriction and restriction. In Crawford MH, DiMarco JP [eds]: Cardiology. St. Louis, Mosby, 2001, Sec. 5, Chap. 5, p 15.5.)

Braunwald, 7th ed.
Large Pericardial effusion

From Hurst, 9th ed CD-ROM, Ch 14
Epicardial fat has echogenicity between blood pool and myocardium; associated with metabolic syndrome

Figure 1: Transthoracic echocardiogram showing a large area of epicardial adipose tissue (white arrows) on free wall of right ventricle (RV).
Pleural effusion

From Hurst, 9th ed CD-ROM, Ch 14
FIGURE 64-9  Two-dimensional echocardiogram of a large, circumferential pericardial effusion. LA = left atrium; LV = left ventricle; PE = pericardial effusion; RA = right atrium; RV = right ventricle. (From Kabbani SS, LeWinter M: Cardiac constriction and restriction. In Crawford MH, DiMarco JP [eds]: Cardiology. St. Louis, Mosby, 2001, Sec. 5, Chap. 5, p 15.5.
Moderate Pericardial Effusion

Cardiac Tamponade

From Hurst, 9th ed CD-ROM, from Shabetai, The Pericardium, 1981
Two-dimensional echocardiogram illustrating diastolic collapse or indentation of the right ventricle in cardiac tamponade. **Top,** Systole; **middle,** early diastole with indentation indicated by arrow; **bottom,** late diastole with return of normal configuration. AV = aortic valve; LA = left atrium; LV = left ventricle; PE = pericardial effusion; RVOT = right ventricular outflow tract. (From Weyman AE: Principles and Practice of Echocardiography. Philadelphia, Lea & Febiger, 1994, p 1119.)
Subcostal view with RV compression

From Hurst, 9th ed CD-ROM
RV diastolic collapse

From Hurst, 9th ed CD-ROM, Ch 14
FIGURE 64-11 Two-dimensional echocardiogram illustrating right atrial collapse or indentation in cardiac tamponade. LA = left atrium; LV = left ventricle; PE = pericardial effusion; RA = right atrium; RAFW = right atrial free wall; RAI = right atrial indentation; RV = right ventricle. (From Gilliam LD: Hemodynamic compression of the right atrium: A new echocardiographic sign of cardiac tamponade. Circulation 68:294, 1983.)
Right atrial Collapse in Tamponade

From Hurst, 9th ed CD-ROM, Ch 14
Cardiac Tamponade

Four-chamber view with RA compression

From Hurst, 9th ed CD-ROM
RV Compression
Cardiac Tamponade

From *Hurst*, 9th ed CD-ROM, Ch 14
Malignant effusion with strands

From Hurst, 9th ed CD-ROM, Ch 14
Cardiac Tamponade

From Hurst, 9th ed CD-ROM, from Shabetai, The Pericardium, 1981
Exaggerated respiratory variation in LV inflow velocity

E=expiration, I=inspiration

From Hurst, 9th ed CD-ROM, Ch 14
Pericardial Constriction

- Can occur post pericardiectomy
- Can occur with normal (<2mm) pericardial thickness (18% of 143 pericardiectomy patients*)
- Effusive constrictive can occur in about 8% of patients with cath proved tamponade**
- Clinical features important
- Echo features
- CT features

Echo in Constriction

- Actual detection of a thickened pericardium is often difficult with TTE
- M-mode abrupt relaxation of the posterior wall with subsequent flattening of endocardial motion
- Abnormal septal motion (early systolic notch, paradoxic septal motion, diastolic dip)
- IVC dilation and decreased respiratory variation

Ch. 9, Feigenbaum’s Echocardiography, 6th ed. 2005.
Doppler in Constriction

- Exaggerated LVIT E/A ratio and short deceleration and exaggerated respiratory variation of the E wave (resp var’n is a relatively reliable sign of pericardial constriction, and elevated E/A is less specific)
- Resp variation is most marked in early inspiration
- Hepatic vein Doppler shows expiratory increase in diastolic flow reversal
- Effusive-constrictive pericarditis often has confusing combinations of findings

Ch. 9, Feigenbaum’s Echocardiography, 6th ed. 2005.
Doppler in Pericardial Constriction

- Inspiratory increase in IVRT > 25% in all CP and < 5% normals
- Inspiratory decrease in LVIT E wave of > 25% in all CP and < 5% normals (false neg in 12% in one study)
- Larger than normal expiratory decrease in RVIT than normal
- Inspiratory decrease in aortic velocity of 14%, compared with 4% in normal
- Inspiratory increase in pulmonary velocity of 16% compared with 5% in normal
- Changes in all measurements are greater in the first beat following inspiration, compared with changes 2-3 beats later in patients with pulmonary disease
- Hepatic vein flow has marked increase in flow reversal with atrial kick and decrease or reversal of flow in early diastole during expiration
- Pulmonary vein flow is less specific, but both S and D waves increase in expiration, particularly D wave

Ch. 29 in Otto’s The Practice of Clinical Echocardiography, 2nd ed. 2002.
Pericardial Calcification

From Hurst, 9th ed CD-ROM
FIGURE 64-14  Chest radiograph showing marked pericardial calcifications in a patient with constrictive pericarditis.
FIGURE 64-5A  Femoral arterial (FA), right atrial (RA), and pericardial pressure before (A) and after (B) pericardiocentesis in a patient with cardiac tamponade. Both RA and pericardial pressure are about 15 mm Hg before pericardiocentesis. In this case there was a negligible paradoxical pulse. Note presence of $x$ descent but absence of $y$ descent before pericardiocentesis. Pericardiocentesis results in a marked increase in FA pressure and marked decrease in RA pressure. During inspiration, pericardial pressure becomes negative, there is clear separation between RA and pericardial pressure, and $y$ descent is now evident and prominent, suggesting the possibility of an effusive-constrictive picture. (Adapted from Lorell BH, Grossman W: Profiles in constrictive pericarditis, restrictive cardiomyopathy and cardiac tamponade. In Baim DS, Grossman W [eds]: Grossman’s Cardiac Catheterization, Angiography, and Intervention. Philadelphia, Lippincott Williams & Wilkins, 2000, p 840.)
Constrictive Pericarditis, with dense mobile pericardial echo.
FIGURE 64-13  Schematic representation of transvalvular and central venous flow velocities in constrictive pericarditis. During inspiration the decrease in left ventricular filling results in a leftward septal shift allowing augmented flow into the right ventricle. The opposite occurs during expiration. D = diastolic venous flow; EA = mitral inflow; HV = hepatic vein; LA = left atrium; LV = left ventricle; PV = pulmonary venous flow; RA = right atrium; RV = right ventricle; S = systolic venous flow.  

Braunwald, 7th ed.
Supplementary Slides
Normal Pericardium enhanced by fat

Arrowheads indicate fat outside the pericardium, seen as a thin white line, normally 1-2 mm thick

From Hurst’s The Heart, 1998, p.636
MRI, transverse view using spin-echo (SE) and gradient-echo (GE) techniques
Pericardium in MRI is normally <3mm and is dark. 4-chamber (L) and 2-chamber (R) views. Pericardial effusion is white and pleural effusion is gray.

From Hurst’s *The Heart*, 1998, p. 651
Normal MRI Gradient-echo images: acquired in the 2-chamber, 4-chamber, and midventricular short axis tomographic planes.
Pericardial Calcification
By CT

Densely calcified pericardium at mid-heart level

From Hurst’s The Heart, 1998, p.636
FIGURE 64-16  Computed tomographic scan showing increased pericardial thickness and mild calcification in a patient with constrictive pericarditis.
Figure 2: MRI showing epicardial adipose tissue, as T1-emphasized high signal density, on free wall of right ventricle and around left ventricular apex. TSET1-weighted sequence with oblique axial orientation, 10-mm thickness section with 1-mm intersection gap, 370 FOV, 256 × 256 matrix.
Figure 2  CT section more inferiorly showing slight difference in attenuation between pleural fluid (white arrows) and pericardial mesothelioma (black arrows). Cardiac silhouette is enlarged, although the heart is small.
Figure 1A: Computed tomographic scan of the chest shows multiple hydatid cysts surrounding the heart inside the pericardial cavity.

Figure 1B: Computed tomographic scan of the chest shows multiple hydatid cysts surrounding the heart inside the pericardial cavity.
Fig. 1. Normal pericardium
The pericardium is visible as a very thin linear density surrounding the heart (arrows) but is not visualized over much of the left ventricle in an axial nonenhanced CT scan (A: 52-year-old male). An axial ECG-gated spin-echo (SE) T₁-weighted MR image (T₁WI) of a subject (B: 60-year-old male with mitral regurgitation) shows a pericardium with normal thickness (arrows).
Fig. 2. Normal transverse pericardial sinus
Axial nonenhanced CT scan (A: 65-year-old female) and ECG-gated SE T_{1}WI (B: 77-year-old male, C, D: 49-year-old male) show the superior aortic recesses (arrows) in a normal position, anterior or posterior to the ascending aorta.
Fig. 2. Normal transverse pericardial sinus
Axial nonenhanced CT scan (A: 65-year-old female) and ECG-gated SE T₁WI (B: 77-year-old male, C, D: 49-year-old male) show the superior aortic recesses (arrows) in a normal position, anterior or posterior to the ascending aorta.
Fig. 3. Pericardial cyst in an asymptomatic 39-year-old female
An axial enhanced CT scan shows a nonenhanced, low-attenuated, well-circumscribed, ovoid mass adjacent to the right side of the pericardium and consistent with a pericardial cyst.
Fig. 4. Pericardial cyst in an asymptomatic 65-year-old female.
A well-circumscribed, ovoid pericardial cyst (arrows) adjacent to the right side of the pericardium shows a low signal intensity in a T₁WI (A) and a high signal intensity in a short TI inversion recovery image (B).
Fig. 5. Pericardial cyst in an asymptomatic 44-year-old female
This subject was revealed to have an abnormality in a chest X-ray for health screening. The cyst (arrow) shows a low signal in an axial ECG-gated SE T1WI (A) and a high signal in a T2WI (B) in the sagittal plane. It appears to be pulmonary effusion. An axial nonenhanced CT scan taken with the subject in a supine position (C) show fluid collection (arrow) resembling pulmonary effusion. An axial nonenhanced CT scan taken with the subject in a prone position (D) reveals the fluid collection (arrow) appearing in the higher level and with a changed shape. Such changes are consistent with pericardial cyst.

Fig. 5. Pericardial cyst in an asymptomatic 44-year-old female. This subject was revealed to have an abnormality in a chest X-ray for health screening. The cyst (arrow) shows a low signal in an axial ECG-gated SE T1WI (A) and a high signal in a T2WI (B) in the sagittal plane. It appears to be pulmonary effusion. An axial nonenhanced CT scan taken with the subject in a supine position (C) show fluid collection (arrow) resembling pulmonary effusion. An axial nonenhanced CT scan taken with the subject in a prone position (D) reveals the fluid collection (arrow) appearing in the higher level and with a changed shape. Such changes are consistent with pericardial cyst.
Fig. 6. Constrictive pericarditis in a 41-year-old female with symptoms of heart failure

An axial enhanced CT scan (A) and ECG-gated SE $T_1$WI (B) demonstrate pericardial calcification and thickening (arrows). The dilated right atrium, in contrast to the normal-sized right and left ventricles, is suggestive of pericardial constriction.
Fig. 7. Constrictive pericarditis in a 77-year-old male with symptoms of heart failure. An axial enhanced CT scan shows diffuse pericardial thickening (arrows) without calcification and with tubular-shaped ventricles.
Fig. 8. Constrictive pericarditis in a 40-year-old male with symptoms of constriction
An enhanced paracoronal multiplanar reformatted (MPR) image (A) shows diffuse pericardial thickening (arrows). An MPR image after pericardectomy clearly shows that most of the pericardium, except for the bottom (arrow), has been resected (B).
Fig. 9. Pericardial effusion in a 60-year-old female with congestive heart failure
Axial ECG-gated SE $T_1$WI (A) and gradient echo (GRE) cine image (B) show diffuse pericardial effusion (arrows) and bilateral pulmonary effusion due to congestive heart failure. Note that pericardial effusion shows a higher signal than epicardial and subcutaneous fat in the GRE cine image (B).
Fig. 10. Pericardial hematoma in a 59-year-old male with recent mitral valve replacement. An axial nonenhanced CT scan shows localized fluid collection (arrow) within the pericardial space. The center of the fluid shows a slightly higher attenuation, which is compatible with hematoma.
Figure 2. Axial contrast material–enhanced CT scan obtained through the inferior aspect of the heart shows a fingerlike structure (arrow) projecting into the right atrium (RA). Note the coronary sinus emptying into the right atrium (arrowheads). The IVC is also seen entering the atrium at this level. The patient was referred for echocardiography, which revealed a prominent eustachian valve remnant. LV = left ventricle, RV = right ventricle.
Figure 3. Axial contrast-enhanced CT scan shows a filling defect (arrow) projecting from the lateral wall into the right atrium (RA). The filling defect is seen in the characteristic location of the crista terminalis. $A =$ aorta, $LA =$ left atrium, $RV =$ right ventricle.
Figure 4. Axial contrast-enhanced CT scan shows soft tissue projecting into the left atrium (LA) due to an infolding of the wall of the left atrium between the left atrial appendage (LAA) anteriorly and the left superior pulmonary vein (arrow) posteriorly. Note the bulbous tip of the projection (arrowhead). AA = ascending aorta, DA = descending aorta.
**Figure 5.** Contrast-enhanced CT scan shows areas of fat attenuation within the interatrial septum (arrowheads), findings that are consistent with LHIS. The fat spares the fossa ovalis (arrow), which should not be mistaken for an atrial septal defect. $LA =$ left atrium, $LV =$ left ventricle, $RA =$ right atrium, $RV =$ right ventricle.
Figure 6.  (a) Drawing illustrates a cut-away view of the right ventricle. Arrowheads indicate the anterior and posterior papillary muscles, open arrow indicates the moderator band. Note the smaller conal papillary muscle (solid arrow). (b, c) Axial contrast-enhanced CT scans show the anterior papillary muscle (arrowhead in b) and the moderator band (arrow in c).
Figure 7. (a) Contrast-enhanced CT scan of the heart shows a modified long-axis view of the anterior papillary muscle (A). Note the thin chordae tendineae (arrows) extending to the anterior and posterior mitral valve leaflets. (b) Contrast-enhanced CT scan of the heart shows a modified long-axis view of the posterior papillary muscle (P) and chordae tendineae (arrows). (c) Reformatted image of the left ventricle from CT data shows a modified short-axis view of the anterior (A) and posterior (P) papillary muscles.
Figure 8.  (a) Axial contrast-enhanced CT scan shows an outward bulge (arrow) along the expected location of the left aortic sinus. This finding should not be misinterpreted as an aneurysm in a sinus of Valsalva. (b) Coronal reformatted image from CT data better demonstrates the aortic anatomy. Note that the aortic valve plane (dashed line) is oriented oblique to the axial plane. (c) Oblique axial reformatted image of the aortic valve from CT data shows the normal appearance of the aortic sinuses (arrow).
Figure 9.  (a) Axial contrast-enhanced CT scan obtained in an 80-year-old woman with a history of a previously malpositioned Swan-Ganz catheter shows a bulge in the pulmonary artery (arrow), a finding that raised suspicion for a pseudoaneurysm.  
(b) Coronal oblique reformatted image from CT data again shows the bulge in the proximal pulmonary artery (arrow).  
(c) One of a series of oblique axial CT scans obtained along the longitudinal axis of the pulmonary artery helps confirm the absence of a pseudoaneurysm (arrow).
Figures 10, 11. (10) Drawing illustrates a cutaway view of the anterior aspect of the heart. Note how the pericardium extends superiorly to cover the great vessels (arrows). \( AA \) = ascending aorta, \( LV \) = left ventricle, \( PA \) = pulmonary artery, \( RA \) = right atrium, \( RV \) = right ventricle, \( SVC \) = superior vena cava. (11) Pericardial recesses and sinuses. (a) Drawing illustrates the pericardial sac with the heart removed. Note that the transverse sinus (\( T \)) and oblique sinus (\( \times \)) are separated by pericardial reflections. Note also the extension of the pericardium superiorly. The pulmonic vein recesses (arrows) lie between the superior and inferior pulmonary veins. (b, c) Axial contrast-enhanced cardiac-gated images of the heart obtained at (b) and inferior to (c) the level of the right inferior pulmonary vein show areas of fluid attenuation anterior, posterior, and inferior to the vein (arrowheads). (d, e) Sagittal (d) and coronal (e) reformatted images from CT data show the relationship between the fluid in the pulmonic vein recess (arrowhead) and the pulmonary vein.
Figures 10, 11. (10) Drawing illustrates a cutaway view of the anterior aspect of the heart. Note how the pericardium extends superiorly to cover the great vessels (arrows). AA = ascending aorta, LV = left ventricle, PA = pulmonary artery, RA = right atrium, RV = right ventricle, SVC = superior vena cava. (11) Pericardial recesses and sinuses. (a) Drawing illustrates the pericardial sac with the heart removed. Note that the transverse sinus (T) and oblique sinus (*) are separated by pericardial reflections. Note also the extension of the pericardium superiorly. The pulmonic vein recesses (arrows) lie between the superior and inferior pulmonary veins. (b, c) Axial contrast-enhanced cardiac-gated images of the heart obtained at (b) and inferior to (c) the level of the right inferior pulmonary vein show areas of fluid attenuation anterior, posterior, and inferior to the vein (arrowheads). (d, e) Sagittal (d) and coronal (e) reformatted images from CT data show the relationship between the fluid in the pulmonic vein recess (arrowhead) and the pulmonary vein.
Figure 12. (a) Axial contrast-enhanced CT scan shows fluid in the anterior portion of the superior aortic recess (arrow), a finding that can sometimes be mistaken for aortic dissection. A small amount of fluid is also seen posterior to the ascending aorta (A) in the posterior portion of the superior aortic recess (arrowhead). (b) Axial contrast-enhanced CT scan obtained cephalad to a shows the superior extension of the superior aortic recess (arrowhead), which now lies in a right paratracheal location. Fluid in this location can be mistaken for an enlarged lymph node. (c) Coronal reformatted image from CT data shows the superior extension of the superior aortic recess (arrowhead) lateral to the aorta (A). Fluid (arrow) is also identified in the anterior portion of the superior aortic recess between the aorta and the main pulmonary artery (PA). (d) Sagittal reformatted image from CT data again shows the superior extension of the superior aortic recess (arrowhead). SVC = superior vena cava, T = trachea.
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Figure 13.  (a) Axial contrast-enhanced CT scan shows a small amount of fluid in the transverse sinus (T) posterior to the ascending aorta (AA). The transverse sinus extends laterally, where it communicates with the left pulmonic recess (arrow) inferior to the left pulmonary artery. PA = main pulmonary artery. (b) Axial contrast-enhanced CT scan obtained slightly superior to a shows fluid in the left pulmonic recess (arrow). LSV = left superior pulmonary vein, PA = main pulmonary artery, RPA = right pulmonary artery.
Figure 14. (a) Axial contrast-enhanced CT scan shows a small collection of fluid (arrow) in the posterior pericardial recess located below the level of the carina. *B* = bronchus, *RPA* = right pulmonary artery. (b, c) Axial contrast-enhanced CT scans obtained inferior to a demonstrate the continuity between the posterior pericardial recess and the oblique sinus (arrow in c). *LA* = left atrium.
Figure 15. Fibrosing mediastinitis with obstruction of the medial left brachiocephalic vein and SVC in a 34-year-old woman. (a) Axial contrast-enhanced CT scan demonstrates occlusion of the SVC and left brachiocephalic vein. (b, c) Axial contrast-enhanced CT scans obtained at the level of the aortic root (b) and ventricles (c) show the pericardiophrenic vein (arrow) coursing along the lateral aspect of the heart. (d) On an axial CT scan, the pericardiophrenic vein is seen to connect with the inferior phrenic vein (arrowhead), at which point it drains into the IVC (arrow).
Figure 15. Fibrosing mediastinitis with obstruction of the medial left brachiocephalic vein and SVC in a 34-year-old woman. (a) Axial contrast-enhanced CT scan demonstrates occlusion of the SVC and left brachiocephalic vein. (b, c) Axial contrast-enhanced CT scans obtained at the level of the aortic root (b) and ventricles (c) show the pericardiophrenic vein (arrow) coursing along the lateral aspect of the heart. (d) On an axial CT scan, the pericardiophrenic vein is seen to connect with the inferior phrenic vein (arrowhead), at which point it drains into the IVC (arrow).
Figure 1. Initial chest CT shows generalized thickening of the pericardium (arrows) and a moderate-sized left pleural effusion (asterisk).

Figure 2. Repeat chest CT 2 months later shows interval decrease in pericardial thickness (arrows) and no residual pleural effusion.
Echocardiography of the Atria
Atrial Ins and Outs in Heart Failure


Atrial Function

• Poorly defined
• Atrial systolic wall motion
  – Afterload, preload, contractility
  – M-mode aortic root
  – Atrial ejection fraction
  – Atrial filling fraction of LV
• Inflow tract atrial velocity
  – P wave required for atrial systolic function
  – Peak A wave
  – A wave VTI

Atrial Function

- Atrial Active transport
- Atrial conduit

Figure 1 Left atrial pressure-volume loops from three variably loaded beats in a control dog (A) and in a dog with pacing induced heart failure (B). The A loop represents active atrial contraction. The V loop represents passive filling and emptying of the left atrium. Loops are computer smoothed for clarity.

Atrial Function

• In Humans
• LA area by echo

Atrial Inflows and Outflows
Comparing Atrial Inflow Patterns

- Hepatic vein
- IVC
- SVC
- Coronary sinus
- ASD
- Pulmonary vein

Pulmonary Venous Flow

From Rossvoll O et al. (Hatle) J Am Coll Cardiol 1993;21:1687
Pulmonary Venous Flow Pattern

- **LV preload and systolic and diastolic function**
  - Increased LA pressure - lower S if LV systolic dysfunction, (more S if LV systolic function is preserved)
  - Impaired relaxation – larger S and lower D, corresponding to lower MV E
  - Pseudonormal – lower S and dominant D wave and larger Ar wave (lower LV compliance)
  - Restrictive – low S and large D and rapid D deceleration, Ar is variable

- **Age** increases systolic dominance and maybe Ar
- **Mitral regurgitation*** reduces S wave, reverses if severe MR
- **Large ASD** causes single continuous antegrade wave and diminished AR wave**

Normal Pulmonary Vein PW Doppler Patterns

S - systolic
D - diastolic
$S_E$ - early systolic atrial relaxation
$S_L$ - late systolic descent of MV annulus
$A_r$ - atrial reversal
$S_i$ - systolic integral
$D_i$ - diastolic integral

Smith, MD, in Otto, 2002, p. 123
Comparing Atrial Outflow Patterns

- Mitral flow
- Tricuspid flow
Interatrial Septum

- Atrial septal defect – secundum, primum, sinus venosus (absent septum – single atrium)
- Patent foramen ovale
- Atrial septal aneurysm
- Lipomatous hypertrophy of the interatrial septum
- Myxomas usually arise from the interatrial septum
- ATRIAL SEPTAL CURVATURE as a pressure indicator
LA Anomalous Band Causing MR

43 yo woman with dyspnea.

TEE view showing the anomalous band in the LA, connecting the atrial side of the anterior MV leaflet and atrial septum.

TEE view showing the anomalous band in the left atrium, causing prolapse and moderate-to-severe mitral regurgitation.

LA Anomalous Band Causing MR

43 yo woman with dyspnea.
Pathological specimen of the removed band.
TEE view showing improvement in the regurgitation after resection of the band.