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Au3 **TABLE 139.2** Protocols and Findings for the Multimodality Imaging Modalities in the Evaluation of Pericardial Diseases—cont'd

Echocardiography	Prospective or Retrospective, ECG-Gated Multidetector CT	CMR
	Cine Imaging (Retrospective Gated Study Only)	**Bright Blood Cine Images[4] (SSFP)**
	☐ Functional evaluation (septal bounce, pericardial tethering)	☐ Atrial/ventricular size and function ☐ Diastolic restraint ☐ Conical deformity of the ventricles ☐ Myocardial tethering ☐ Diastolic septal bounce ☐ Pericardial thickening and/or effusion
		Late Gadolinium Enhancement Images[5] (Phase-Sensitive Inversion Recovery Sequence)
		☐ Detection of pericardial inflammation
		Real-Time Gradient Echo Cine Image[6]
		☐ Monitor respiratory variation of ventricular septal motion

2D, Two-dimensional; *CMR*, cardiac magnetic resonance; *COPD*, chronic obstructive pulmonary disease; *CT*, computed tomography; *ECG*, electrocardiogram; *ICD*, implantable cardioverter-defibrillator; *IVC*, inferior vena cava; *RA*, right atrium; *SSFP*, steady state free precession; *STIR*, short T1 Au4 inversion recovery; *TEE*, transesophageal echocardiography.
Orientation: axial.
Orientation: 2-, 3- and 4-chamber views + 3 short-axis LV slices (base, mid, and distal).
Orientation: 3 short-axis LV slices (basal, mid, and distal), *optional:* 2-, 3-, and 4-chamber views.
Orientation: 2-, 3-, and 4-chamber views + 3 short-axis LV slices.
Orientation: 2-, 3-, and 4-chamber views + short-axis stack.
Orientation: basal and mid short-axis slice with diaphragm in view.
*Echo Doppler measurements should be repeated in the sitting position (reducing preload) in case of nondiagnostic findings and suspicion for constriction.
From Klein AL, Abbara S, Agler DA, et al. American Society of Echocardiography clinical recommendations for multimodality cardiovascular imaging of patients with pericardial disease. J Am Soc Echocardiogr *2013;26:965-1012; and Verhaert D, Gabriel RS, Johnston D, et al. The role of multimodality imaging in the management of pericardial diseases.* Circ Cardiovasc Imaging *2010;3:333-343.*

Feigenbaum.[8] This seminal work led to the clinical use of echocardiography in the United States, and later elsewhere.

Au7 p0480 Over the next five decades, echocardiography made great progress, and it now includes two-dimensional and three-dimensional imaging, transesophageal echocardiography, spectral and color Doppler, tissue Doppler, and speckle tracking. Each of these ultrasound modalities improves the ability to accurately and noninvasively diagnose the entire spectrum of pericardial disorders. Although for most conditions echocardiography alone is sufficient for diagnosis and initiation of treatment, other modern imaging modalities are sometimes needed to further refine the diagnosis and better initiate and tailor the treatment. These imaging modalities include cardiac computed tomography and cardiac magnetic resonance. Each of these can be useful in the evaluation of the structures, hemodynamics, and functional abnormalities of pericardial disease.

p0485 Table 139.1 describes and compares the strength and weakness of each of those modalities. Table 139.2 summarizes protocols and findings for the multimodality imaging modalities in the evaluation of pericardial diseases.

p0490 Invasive hemodynamic studies may still be needed to further assess clinical issues not resolved by the noninvasive armamentarium. The chapters in this section concentrate mainly on the echocardiographic findings of common and not-so-common pericardial disorders.

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Au9 sc0015 # 140 Normal Pericardial Anatomy

Au10 *Steven Giovannone, MD, Robert Donnino, MD, Muhamed Saric, MD, PhD*

p0495 The pericardium is a membranous sac that envelops almost the entire heart (with the exception of the region of the left atrium around the pulmonary venous ostia) as well as the origins of the great cardiac vessels (the ascending aorta, the main pulmonary artery and the venae cavae). The term "pericardium" is a Latinized version of the Greek word περικάρδιον, which literally means "that

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which is around the heart." As an anatomic term, the word has been used at least since the time of the Greco-Roman physician Galen; for instance, in around AD 160 he used it in describing stab wounds of gladiators resulting in pericardial effusions.[1] In English, the word "pericardium" first appears in print around 1425 in a Middle English translation of *Chirurgia Magna*, a surgical treatise written in Latin by the French physician Guy de Chauliac (c. 1300-1368).[2]

PHYLOGENY AND EMBRYOLOGY

The pericardium envelops the heart of all vertebrates including fishes, amphibians, reptiles, birds, and mammals. As such a phylogenetically ancient structure, it forms very early during embryologic development in humans (starting around 5 weeks' gestation) by the division of the coelom—the original visceral cavity—into pericardial, pleural, and peritoneal spaces. Through incompletely understood mechanisms, embryologic mishaps may result in congenitally absent pericardium or pericardial cysts.

BASIC ANATOMY

The normal pericardium (Fig. 140.1) consists of a double-layered sac: an outer fibrous envelope (fibrous pericardium) and an inner serous sac (serous pericardium). The serious pericardium can be divided into an outer (parietal) and an inner (visceral) layer. The parietal layer normally fuses with the fibrous pericardium to create an inseparable outer layer of the pericardium. The fibrous pericardium is contiguous with the adventitia of the great arteries.

The visceral layer of the serious pericardium is synonymous with the epicardium.[3] Between these two layers there is a virtual space that contains a very small amount of clear serous fluid, as discussed later.[4]

The pericardium spans the space between the third and the seventh rib. Strong superior and inferior sternopericardial ligaments anchor the pericardial sac to the posterior aspect of the sternum. In addition, loose fibrous tissue binds the pericardium to the diaphragm and surrounding thoracic structures, including pleurae. The right and left phrenic nerves travel in this loose tissue between the fibrous pericardium and the pleurae. The arterial supply to the pericardium is provided by the branches of the internal mammary arteries (especially the pericardiophrenic artery) and the descending thoracic aorta. The pericardiophrenic vein, ultimately draining into the brachiocephalic vein, provides the principal venous drainage of the pericardium. The nerves of the pericardium are derived from the sympathetic trunks as well as the vagus and phrenic nerves.

PERICARDIAL THICKNESS

Normal pericardial wall thickness is approximately 1 to 2 mm. It is important to emphasize that transthoracic echocardiography (TTE) does not delineate the pericardial wall boundaries well enough, and therefore TTE is not recommended for measurements of pericardial thickness by either the American Society of Echocardiography[5] or the European Society of Cardiology[6] guidelines on proper use of echocardiography in pericardial disorders. In contrast, pericardial thickness can be obtained by transesophageal echocardiography (TEE).[7] TEE measurements approach the gold standard of computed tomography (CT) and cardiac magnetic resonance (CMR) imaging (Fig. 140.2). Increased pericardial thickness due to fibrosis and calcification is the hallmark of constrictive pericarditis.

PERICARDIAL FLUID

Under physiologic conditions, there is only a very small amount of clear straw-colored pericardial fluid (typically <50 mL) representing an ultrafiltrate of plasma. On echocardiography, the separation between parietal and visceral layers of the serous pericardium

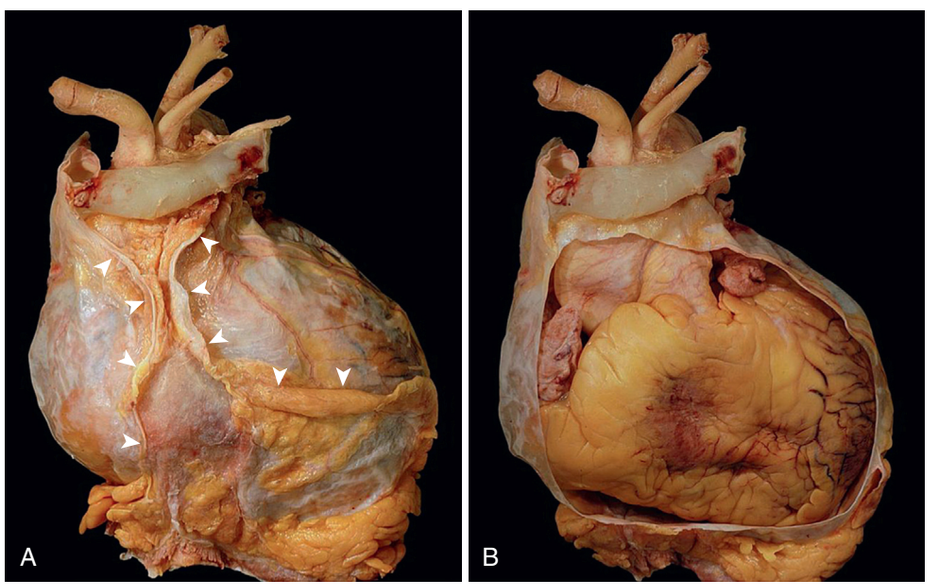

Figure 140.1. Gross anatomy of normal human pericardium. **A**, Anterior view of the intact parietal pericardial sac. The attachment of the fibrous sac to the diaphragm is seen at the base. Abundant epipericardial fat is conspicuously present at the pericardium-diaphragm junction. The mediastinal pleura invest the lateral portion of fibrous pericardium. The anterior reflections of the mediastinal pleura are indicated by the *white arrowheads*. The space between the arrowheads corresponds to the attachment of the pericardium to the posterior surface of the sternum. Superiorly, the left innominate vein is seen merging with the superior vena cava. The arterial branches of the aortic arch are just dorsal to the innominate vein. **B**, The anterior portion of the pericardial sac has been removed to show the heart and great vessels in anatomic position. It distinctly shows how the proximal segments of the great arteries are intrapericardial. At that point, there is fusion of the adventitia of the great vessels with the fibrous pericardium. *(From Klein AL, Abbara S, Agler DA, et al. American Society of Echocardiography clinical recommendations for multimodality cardiovascular imaging of patients with pericardial disease.* J Am Soc Echocardiogr *2013;26(9):965-1012.e15.)*

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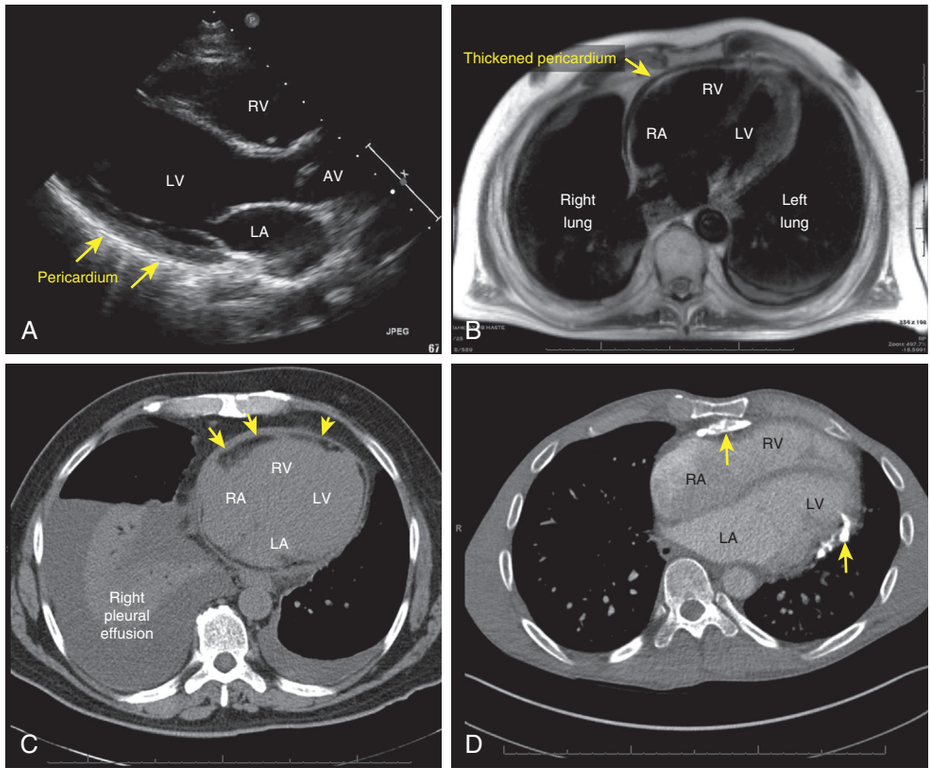

f0020 **Figure 140.2.** Imaging of pericardial thickness and calcifications. **A**, Transthoracic echocardiography (TTE): Although the pericardium can be visualized by TTE (*arrows*), the exact thickness of the pericardium cannot be accurately measured by this means. **B**, Cardiac magnetic resonance demonstrates thickened pericardium (*arrow*) adjacent to the right heart on a T2-weighted spin echo axial image—this is the sequence that often shows the thickening the best. **C** and **D**, Computed tomography (CT). **C**, Chest CT without contrast enhancement. Axial slice demonstrates thickened pericardium, most prominent anteriorly (*arrows*). **D**, Intravenous contrast–enhanced CT of the chest. Axial image shows areas of focal thickening with calcification in the pericardium (*arrows*). *AV*, Aortic valve; *LA*, left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

either is imperceptible or is seen only during ventricular systole as a slitlike echolucent area between the two pericardial layers. This small amount of fluid has multiple physiologic roles: It diminishes friction between the two pericardial layers; by being an incompressible fluid, it protects the heart from minor injuries; and it provides a source of vasoactive substances that may regulate the function of the heart and the coronary arteries.[8]

s0030 INTRAPERICARDIAL PRESSURE

p0530 The intrapericardial pressure (P) is a product of the intrapericardial fluid volume (V) and the pericardial stiffness (ΔP/ΔV):

Au12

$$P = V * \frac{\Delta P}{\Delta V}$$

p0535 Pericardial stiffness, an inverse of pericardial compliance, is the slope of the intrapericardial pressure-volume curve. Because a normal pericardium is not an impediment for transmission of intrathoracic pressure changes into the pericardial space during physiologic respiration and because the physiologic amount of pericardial fluid is small, a normal intrapericardial pressure is close to 0 mm Hg or even negative (subatmospheric).

p0540 Under pathologic conditions, the intrapericardial pressure may rise either because of an increase in the amount of pericardial fluid (as with pericardial effusion) or because of pronounced pericardial stiffness (as in rapidly accumulating pericardial effusion or with effusive-constrictive pericarditis). The pericardial pressure-volume relationship is nonlinear; initially the slope is flat but subsequently becomes very steep. This nonlinear relationship explains why increases in the size of pericardial effusion initially may only modestly elevate intrapericardial pressures when the slope is flat. However, once the steep portion of the curve is reached, even a small additional increase in the size of pericardial effusion leads to marked increases in intrapericardial pressure (Fig. 140.3, *A*).

p0545 When the intrapericardial pressure exceeds the pressure in any of the cardiac chambers during at least part of the cardiac cycle, tamponade physiology develops. Conversely, even removal of a relatively small amount of pericardial effusion may rapidly relieve signs and symptoms of tamponade. With slowly accumulating pericardial effusions, pericardial stiffness gradually falls, and thus the intrapericardial pressure remains near normal for longer periods of time when compared with acute pericardial effusions. Mathematically, this corresponds to a shift of the pressure-volume relationship to the right (see Fig. 140.3, *B*).

INTRAPERICARDIAL VERSUS EXTRAPERICARDIAL HEART STRUCTURES s0035

p0550 To fully understand pericardial physiology and pathology, it is important to recognize which heart structures lie within and which lie outside the pericardial sac. As noted earlier, the proximal portions of the great arteries (the ascending aorta and the main pulmonary artery) are located within the pericardial sac, whereas the superior portion of the left atrium and the ostia of the pulmonary veins are outside of the pericardial sac. Therefore dissections or other injuries of proximal portions of the great arteries may result in the development of a pericardial effusion.

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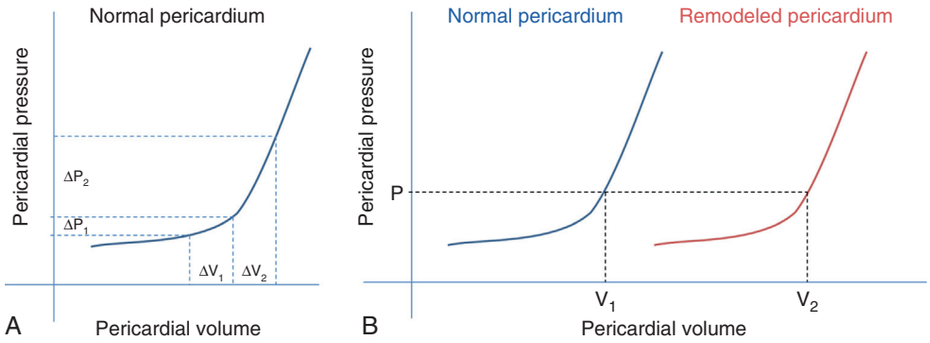


f0025 **Figure 140.3.** Pericardial pressure-volume relationship. **A**, Normal pericardium: pressure-volume relationship of a normal pericardium is nonlinear. Note that the same unit increase in the volume of pericardial effusion ($\Delta V_1 = \Delta V_2$) produces markedly different intrapericardial pressure changes ($\Delta P_2 \gg \Delta P_1$) depending on the slope of the pressure-volume relationship. The steeper the slope, the greater the increase in intrapericardial pressure relative to increases in intrapericardial volume. **B**, Normal versus remodeled pericardium: the curve on the left demonstrates pressure-volume relationship with acute pericardial effusion in the setting of normal pericardial stiffness. The curve on the right demonstrates pressure relationship with chronic pericardial effusion; the pericardium remodels to accommodate the slowly accumulating fluid. Note that much greater amounts of pericardial fluid (V_2 vs. V_1) are needed to produce the same pericardial pressure (P).

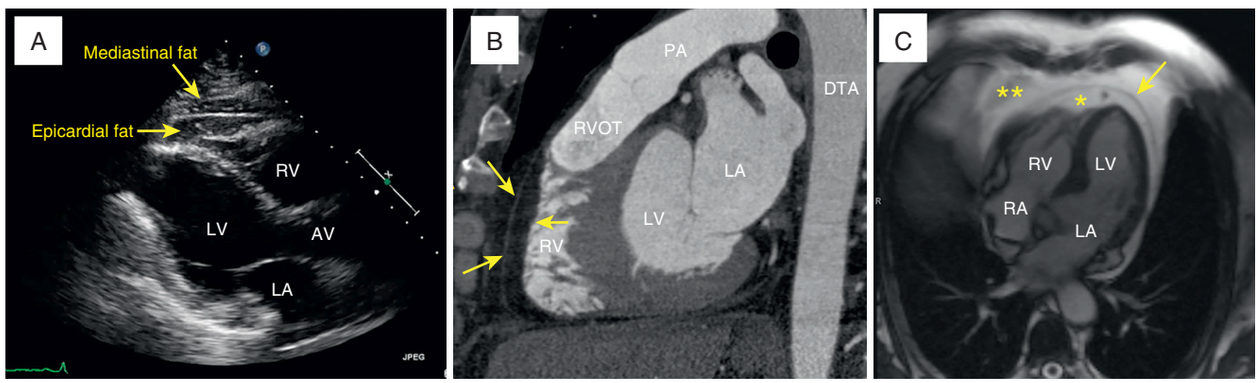
p0555 In contrast, the extrapericardial location of the pulmonary veins contributes to exaggerated respiratory variations in tamponade and constrictive pericarditis. Briefly, under physiologic conditions, during inspiration the intrathoracic pressure drops, which leads to a drop in both pulmonary vein and intracardiac pressures. The opposite occurs during expiration. Because intrathoracic pressures changes affect the pulmonary veins and the left heart almost equally, the pressure gradient between the pulmonary vein and the left atrium does not change substantially. Therefore, under physiologic conditions there is only a minor decrease of left heart filling during inspiration.

p0560 Significant pericardial effusions and constrictive pericarditis insulate intracardiac chambers from changes in intrathoracic pressures during respiration. Thus, in such pathologic conditions, the normal inspiratory drop in intrathoracic pressures will lead to a drop in the pulmonary vein pressure without a concomitant drop in the left atrial pressure. The resulting decrease in the pressure gradient between the pulmonary vein and the left atrium leads to a marked drop in the filling of the left heart during inspiration. The concept of these exaggerated respiratory variations in tamponade and constrictive pericarditis is further discussed in other chapters of this book.

PERICARDIAL FAT

s0040

p0565 A variable amount of fat may be present in and around the pericardial sac; collectively this adipose tissue is referred to as the pericardial fat pad. Intrapericardial fat accumulates preferentially along the coronary arteries and in the atrioventricular groove; this is referred to as epicardial fat. Additional fat tissue may also accumulate outside the pericardium in the nearby mediastinum, particularly anterior to the right heart; this is referred to as mediastinal fat. On imaging, the epicardial and mediastinal fat layers should not be mistaken for a loculated pericardial effusion. Echocardiographically, pericardial fat is a noncircumferential accumulation of ultrasonographically heterogeneous material that moves in concert with the heart. In contrast, pericardial effusions are typically stationary, echolucent, and circumferential rather than restricted to the region around the right heart. Pericardial fat can also be well visualized by cardiac CT or MRI (Fig. 140.4 and Video 140.4, *A*).



f0030 **Figure 140.4.** Imaging of pericardial fat. **A**, Transthoracic echocardiogram: pericardial fat pad consists of epicardial fat inside the pericardium and mediastinal fat just outside the pericardial sac. On TTE, pericardial fat (*arrows*) appears as noncircumferential accumulation of ultrasonographically heterogeneous material that moves in concert with the heart. In contrast, pericardial effusions are typically stationary, echolucent, and circumferential rather than restricted to the region around the right heart (see accompanying Video 140.4, *A*). **B**, Computed tomography (CT): intravenous contrast CT image in the sagittal projection demonstrates pericardial fat pad (*thick arrow*) area between the right ventricle (*RV*) and the fibrous pericardium (*thin* Au1 *arrow*). **C**, Cardiac magnetic resonance: SSFP (steady state free precession) image in axial projection demonstrates fat surrounding the pericardium (*arrow*). Epicardial fat (*asterisk*) is inside the pericardium, and mediastinal fat (*double asterisk*) is just outside the pericardium. AV, Aortic valve; DTA, descending thoracic aorta; *LA*, left atrium; *LV*, left ventricle; *PA*, pulmonary artery; *RA*, right atrium; *RV*, right ventricle; *RVOT*, right ventricular outflow tract.

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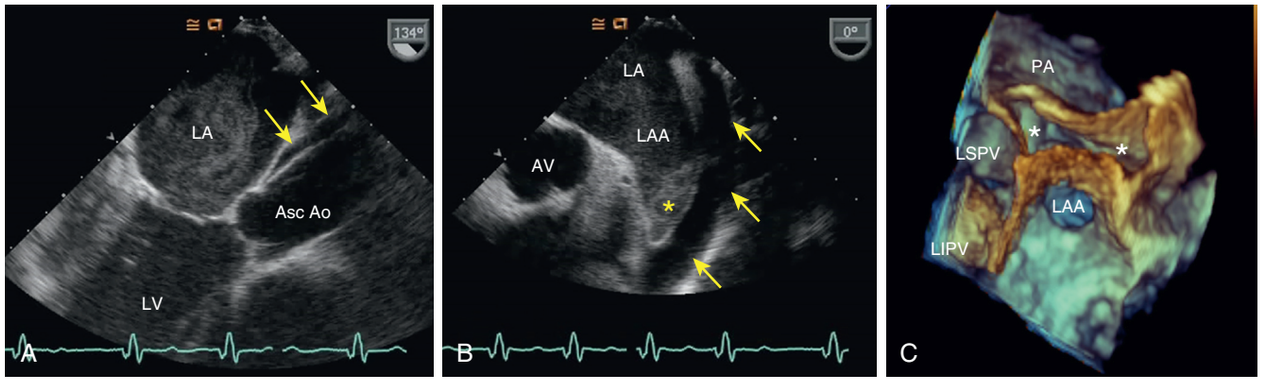

f0035 **Figure 140.5.** Pericardial extensions. **A**, Two-dimensional transesophageal echocardiography (2D TEE) demonstrates a small effusion in the aortic recess of the transverse sinus of the pericardium (*arrows*) (see accompanying Video 140.5, *A*). **B** and **C**, A small effusion in the pulmonic recess of the pericardium around the left atrial appendage (*LAA*) is seen on 2D TEE (*arrows* in **B**) and 3D TEE (*asterisks* in **C**) (see accompanying Videos 140.5, *B* and *C*). *Asc Ao*, Ascending aorta; *LA*, left atrium; *LIPV*, left inferior pulmonary vein; *LSPV*, left superior pulmonary vein; *LV*, left ventricle; *PA*, pulmonary artery.

s0045 ## PERICARDIAL EXTENSIONS

p0570 The main pericardial sac communicates with several extensions that are referred to as sinuses and recesses.[9] There are two sinuses (oblique sinus and transverse sinus) and multiple recesses. The two sinuses do not communicate directly. Occasionally, pericardial effusion may only be present in one or more of these sinuses and recesses and absent from the main pericardial cavity (Fig. 140.5 and Videos 140.5, *A-C*). The oblique sinus is a blind pouch or cul-de-sac that overlies the posterior aspect of left atrium, normally between all four pulmonary veins, as well as a portion of the right atrium. The transverse sinus is bounded anteriorly by the origins of the great arteries, inferiorly by the roof of the left atrium, and posteriorly by the superior vena cava, the atria, and the left atrial appendage.

p0575 Extensions of the transverse sinus include the superior aortic recess (between the ascending aorta and the superior vena cava), the inferior aortic recess (between the ascending aorta and the right atrium), and the right and left pulmonic recesses (around the right and left pulmonary arteries). Pericardial effusions localized in the transverse sinus and its recesses should not be mistaken for other pathologies such as type A aortic dissection. The postcaval recess is an extension of the main pericardial cavity; it lies posterior and to the right of the superior vena cava.[10,11]

p0580 Please access ExpertConsult to view the corresponding videos for this chapter.

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sc0020 # 141 Pericarditis

Au13 *Sonia Jain, MD, MBBS, Sunil Mankad, MD, FASE*

s0050 ## DEFINITION

p0585 Pericarditis refers to a symptomatic inflammation of the pericardium and can present as acute, chronic, or recurrent pericarditis. Myopericarditis implies associated inflammation, often with coinciding tissue necrosis of the myocardium.[1] Acute pericarditis (AP) is the most common manifestation of pericardial disease.

s0055 ## EPIDEMIOLOGY

p0590 The incidence and prevalence is difficult to determine because of the presence of subclinical disease, the variability of the clinical presentation, lack of uniform diagnostic criteria, and referral bias. The reported autopsy prevalence is 1.06% in one large series.[2]

s0060 ## ETIOLOGY

p0595 Acute, recurrent, or chronic pericarditis can be encountered in a myriad of clinical settings. An elegant yet simple etiologic classification has been described and includes (1) infectious, (2) autoimmune, (3) reactive, (4) metabolic, (5) traumatic, and (6) neoplastic.[3] The vast majority of cases in Western Europe and North America are of presumed viral etiology and commonly referred to as "idiopathic." There is a global geographical variation in infectious

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