Echocardiography is used extensively in the diagnosis and management of all forms of pericardial disease spanning from congenital anomalies of the pericardium to acute pericarditis, pericardial effusion, tamponade, constrictive pericarditis and pericardial tumors.
Echocardiography in Pericardial Disease

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For Physicians and Other Health Care Professionals

Introduction

Echocardiography is used extensively in the diagnosis and management of all forms of pericardial disease spanning from congenital anomalies of the pericardium to acute pericarditis, pericardial effusion, tamponade, constrictive pericarditis and pericardial tumors.

Due to its widespread availability, portability, safety, and ability to provide both anatomic and hemodynamic data, echocardiography is typically the initial imaging modality of choice for visualization of pericardial disorders as well as for guidance of pericardiocentesis. Additional imaging with computed tomography (CT) and cardiac magnetic resonance (CMR) may be necessary in selected cases, typically to overcome limitations of echocardiography as in patients with difficult echocardiographic windows, when there is a need for more precise measurement of pericardial thickness or when tissue characterization and/or relationship to structures surrounding the pericardium is required.

Dedicated guidelines of the European Society of Cardiology (ESC) [1] and the American Society of Echocardiography (ASE) [2] provide in-depth recommendations on proper use of echocardiography in pericardial disorders. Echocardiography is also an integral part of the CHASER pathway for the management of pericardial disease [3].

Normal Echocardiographic Appearance of the Pericardium

A brief overview of pericardial anatomy as it specifically relates to echocardiography is provided below.

Pericardial Thickness: Pericardium is a sac-like structure consisting of a parietal and a visceral (epicardial) layer. Normal pericardial wall thickness is approximately 1–2 mm. Unfortunately, transthoracic echocardiography (TTE) does not have sufficient image resolution and therefore is not recommended for measurements of pericardial thickness. In contrast, pericardial thickness can be measured by transesophageal echocardiography (TEE) [4] and such measurements approach the gold standard of CT and CMR.

Pericardial Fluid: Normally there is only a very small amount of physiologic pericardial fluid (<50 mL) and separation between parietal and visceral layers is either imperceptible on echocardiography or occurs only during ventricular systole, when a slit like echo lucent area between the two layers may be seen.

Pericardial Fat: A variable amount of fat may accumulate in and around the pericardial sac.
Intrapericardial (epicardial) fat tends to accumulate in the atrioventricular groove and along the coronary arteries. Additional fat tissue is seen extrapericardially in the surrounding mediastinum, especially anterior to the right heart. The epicardial and mediastinal fat layers should not be mistaken for a loculated pericardial effusion (Fig. 5.1, Panel a). Echocardiographically, pericardial fat is heterogeneous, non-circumferential and moves in concert with the heart. In contrast, pericardial effusion is typically echo lucent, stationary and circumferential rather than restricted to the region around the right heart.

**Intra vs Extrapericardial Structures:** For a full understanding of pericardial physiology and pathology, it is important to recognize which cardiac structures are within and which are outside the pericardial sac. The proximal portions of the great vessels (the ascending aorta and the main pulmonary artery) are within the pericardial sac. Thus injuries or dissections of proximal portions of these vessels may lead to pericardial effusion. In contrast, superior portion of the left atrium and the ostia of the pulmonary veins are not within the pericardial sac. This anatomic fact contributes to exaggerated respiratory variations in tamponade and constrictive pericarditis as further discussed in appropriate sections of this chapter.

**Pericardial Extensions:** The main pericardial space communicates with several extensions referred to as sinuses and recesses. Transverse sinus (Fig. 5.1, Panels b, c) is located around the ascending aorta. This finding should not be mistaken for a type A aortic dissection.
the origins of the great vessels and the left atrial appendage while the oblique sinus surrounds the ostia of the pulmonary veins. Pericardial effusion may occasionally be restricted to one or more of these sinuses and recesses. These localized pericardial effusions should not be mistaken for other pathologies such as the type A aortic dissection in the case of fluid accumulation in the transverse sinus.

**Congenital Absence of the Pericardium**

Partial or complete absence of the pericardium is a rare congenital disorder that could not reliably be diagnosed in vivo prior to advent of modern cardiac imaging. Congenital absence of the pericardium was first undoubtedly described in 1793 on autopsy by the Scottish physician and pathologist Matthew Baillie (1761–1823) [5] although an earlier and likely erroneous description might have been made by the Italian anatomist Realdo Colombo (1516–1559) [6]. The reported occurrence of congenital absence of the pericardium is 1 per 14,000 autopsies [7].

**Key Anatomic Features**

Since partial absence of the pericardium surrounding the left heart is the most common form, excessive displacement of the cardiac apex to the left (levoposition and levorotation of the heart) is the key anatomic feature.

**Echocardiography Indications**

Congenital absence of the pericardium may be an incidental finding or the patient may present with nonspecific symptoms such as chest pain, palpitations or shortness of breath.

**Echocardiography Findings**

Echocardiographically, congenital absence of the pericardium cannot be visualized per se but is rather deduced from indirect signs. Because the cardiac apex is displaced laterally in the partially absent left-sided pericardium, standard echocardiographic imaging windows provide unusual images of the heart. In the parasternal views, the right ventricle, although typically normal in size, appears enlarged. Additionally, there is increased cardiac motion due to absence of pericardial constraint and paradoxical interventricular septal motion (Fig. 5.2, Panel a). On the apical 4-chamber view the cardiac apex is displaced to the left and the heart has an unusual tear-drop shape.

Patients in whom the pericardium was surgically removed (Fig. 5.2, Panel b) may have echocardiographic findings similar to those in patients with congenitally absent pericardium (e.g. patients post pericardiectomy for constrictive pericarditis).

**Alternative Imaging**

Congenital absence of the pericardium may be suspected from an unusual shape to the cardiac silhouette on chest X ray (Fig. 5.2, Panel c). Definitive diagnosis of congenitally absent pericardium is usually established by CT or CRM.

**Pericardial Cysts and Diverticula**

Congenital pericardial cysts and diverticula may be considered as accessory pericardial spaces. Pericardial cysts are thin-walled unilocular structures filled with clear, watery fluid; they do not communicate with the pericardial sac. In contrast, pericardial diverticula are abnormal extensions that communicate with the main pericardial space.

In general, pericardial cysts are rare, typically benign and congenital in nature with a prevalence of 1 per 100,000 cases [8]. Congenital pericardial cysts arise from the primitive coelum, the progenitor of pericardial, pleural and peritoneal cavities. Their exact embryogenesis is uncertain. Acquired cysts, such as pericardial hydatid cysts, may be seen in parts of the world where parasitic infections with *Echinococcus* species are common. Like congenital cysts, hydatid cysts are filled with a watery fluid, thus the name (from Greek Υδατίς; stem Υδατίδ- meaning ‘a drop of water’).
Key Anatomic Features

Congenital pericardial cysts are typically located in one of the cardiophrenic angles, more often in the right than the left cardiophrenic angle. Their size varies from small fluid collection in the cardiophrenic angle to large masses filling the mediastinum.

Echocardiography Indications

Pericardial cysts and diverticula may be an incidental finding or the patient may present with nonspecific symptoms such as chest pain, palpitations or shortness of breath.

Echocardiography Findings

On ultrasound imaging, pericardial cysts were first characterized by M mode echocardiography in 1975 [9], and then by 2D echocardiography in 1983 [10]. The key echocardiographic findings of a pericardial cyst include an echo lucent, thin-walled structure located adjacent to the heart and above the diaphragm (most often in and around the right atrioventricular groove), filled with clear, stationary fluid and without obvious communications to any of the surrounding structures (Fig. 5.3, Panel a).

Fig. 5.2 Absence of the pericardium. Panel a: M mode recording shows paradoxical interventricular septal motion (arrow) in a patient with congenital absence of the pericardium. Panel b: Chest X ray demonstrates an unusual cardiac silhouette with lateral and cranial displacement of the cardiac apex (arrow) in a patient with congenital absence of the pericardium. Panel c: Transthoracic echocardiogram in the apical 4-chamber view demonstrates characteristic lateral displacement of the cardiac apex (arrow) in a patient with absent pericardium after surgical stripping (pericardiectomy). Abbreviations: LA left atrium, LV left ventricle, RA right atrium, RV right ventricle.
Differential Diagnosis

Other echo lucent structures adjacent to the heart such as pericardial and pleural effusion or ascites may be mistaken for a pericardial cyst. Pericardial effusions tend to be circumferential and often show signs of organization. Unlike pericardial cysts, left and right pleural effusions follow the anatomic boundaries of respective pleural spaces.

Vascular anomalies (especially in and around atrioventricular grooves) such as coronary artery aneurysms, coronary artery fistulas and enlarged coronary sinus may be differentiated from pericardial cysts with the intravenous administration of echocardiographic contrast agents. Unlike vascular anomalies, pericardial cysts do not communicate with the vascular pool and thus they do not opacify after echocardiographic contrast administration.

To differentiate pericardial cysts from coronary artery aneurysms and coronary artery fistulas, microbubble contrast agents (such as perflutrane) should be used while agitated saline can be used for the diagnosis of enlarged coronary sinus due to persistence of the left superior vena cava.

Alternative Imaging

The diagnosis of a pericardial cyst is often suspected on a chest X ray as a mass like density, typically located in the right cardiophrenic angle (Fig. 5.3, Panel b). The definitive diagnosis of pericardial cysts and diverticular is usually established by CT or CRM.

Pericardial Effusion

Pericardial effusion is an accumulation of fluid in the pericardial sac between the visceral and parietal layer of the pericardium. There are numerous causes of pericardial effusions including infectious, metabolic, rheumatologic, traumatic, neoplastic and idiopathic etiologies. In the developed countries, the predominant causes of pericardial effusion are idiopathic and traumatic (especially iatrogenic following surgical or percutaneous procedures).

The hemodynamic spectrum of a pericardial effusion spans from asymptomatic to tamponade, cardiovascular collapse and death.

Key Anatomic and Hemodynamic Features

The pericardial sac envelopes the entire heart except the cranial portion of the left atrium around the ostia of the pulmonary veins. The
proximal portions of the ascending aorta and the main pulmonary artery are also within the pericardial sac. In contrast, the descending thoracic aorta lies outside the pericardial sac; this anatomic feature helps differentiate a pericardial effusion from a left pleural effusion (see below).

The primary determinant of hemodynamic significance of a pericardial effusion is not the volume of intrapericardial fluid per se but rather the intrapericardial pressure exerted by that volume. This is further discussed in the Tamponade section below.

**Echocardiography Indications**

Pericardial effusion may be an incidental finding on an echocardiogram ordered for a different reason, or the patient may present with chest pain, shortness of breath or hypotension and shock.

**Echocardiography Findings**

Here the general echocardiographic features of pericardial effusions will be discussed (Fig. 5.4). Tamponade findings are described separately in a section below.

Pericardial effusions are typically circumferential (around the entire heart) but not necessarily...

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**Fig. 5.4** Pericardial effusion. Transthoracic echocardiogram demonstrates a large pericardial effusion (*asterisks*) in a 47-year-old woman with breast cancer. Panel **a**: Parasternal long axis view demonstrates typical interposition of the pericardial effusion (*asterisks*) between the heart and the descending thoracic aorta. This finding differentiates a pericardial effusion from a left pleural effusion in which there is no such interposition between the heart and the descending thoracic aorta. Panel **b**: Parasternal short axis at the level of the papillary muscles demonstrates that the pericardial effusion (*asterisks*) is larger posterior to the left ventricle that anterior to the right ventricle. This is due to gravity in this supine patient. Panel **c**: In the apical 4-chamber view, note that the pericardial effusion (*asterisks*) surrounds the cardiac apex. This feature helps distinguish a pericardial effusion from a pleural effusion. Panel **d**: Subcostal view demonstrates a large pericardial effusion (*asterisks*). Abbreviations: AV aortic valve, LA left atrium, LV left ventricle, RA right atrium, RV right ventricle.
symmetrical; a larger amount of fluid tends to occur in more dependent areas compared to less dependent ones. In a supine patient, circumferential pericardial effusions tend to be larger posterior to the left heart than anterior to the right heart. Loculated pericardial effusions may occur in any portion of the pericardial sac.

The consistency of pericardial fluid varies from clear, water like collections to partly organized (with strands spanning the two layers of the pericardium) to fully organized, tumor like densities (as in the case of hemorrhagic effusion).

On an echocardiography report, the size, location (circumferential vs. loculated) and fluid characteristics (clear vs. organized) should be described. The size of a circumferential pericardial effusion may be expressed and the end-diastolic effusion thickness plus anterior and posterior pericardial effusion thickness plus epicardial and parietal layers of the pericardium). Small pericardial effusions have end-diastolic thickness of <1 cm; moderate between 1 and 2 cm; and large >2 cm as described in the latest ASE guidelines [2].

The volume of pericardial fluid can roughly be estimated using the so-called cube rule which assumes that the heart is a prolate ellipsoid and that a volume of a cardiac chamber is the cube of its short-axis diameter [11]. In practical terms, one obtains 2D echocardiographic images of the heart in the parasternal long or short axis and then measures the end-diastolic thickness of the pericardial fluid (PF), end-diastolic diameters of the left (LV) and right ventricles (RV), and end-diastolic thickness of the right ventricular free wall (RVF), interventricular septum (IVS) and inferolateral wall (IL).

The end-diastolic diameter of the heart is RVF + RV + IVS + LV + IL; this diameter cubed gives the volume of the heart. The end-diastolic diameter of the pericardial sac is the sum of anterior and posterior pericardial effusion thickness plus the end-diastolic diameter of the heart; the cube of the end-diastolic pericardial sac diameter represents the volume of the pericardial sac. The volume of pericardial effusion is then the difference between the pericardial sac volume and the volume of the heart.

Example:
The patient has a pericardial effusion that measures 1.0 cm both anterior to the right heart and posterior to the left heart at end diastole; RVF=0.3 cm; RV = 1.7 cm; IVS = 07 cm; LV = 3.9 cm; IL = 0.6 cm.

Heart:
End – diastolic diameter of the heart = 0.3 + 1.7 + 0.7 + 3.9 + 0.6 = 7.2 cm
End – diastolic volume of the heart = (7.2)³ = 373 mL

Pericardial sac:
End – diastolic diameter of the pericardial sac = 1.0 + 1.0 + 7.2 = 9.2 cm
End – diastolic volume of the pericardial sac = (9.2)³ = 779 mL

Pericardial effusion:
Pericardial effusion = 779 – 373 = 405 mL.

It must be emphasized that this calculation method gives only a rough estimate of pericardial effusion volume and might overestimate effusion volume especially if cardiac diameters are large. When pericardial effusions are very large, one may observe a swinging motion of the heart with the cardiac apex moving toward and then away from the anterior chest wall. In such instances, one may notice the electrical alternans (a change in QRS voltage) on simultaneous EKG tracings which should accompany any echocardiographic recording. Additionally, overall EKG voltage may be diminished [12].

**Differential Diagnosis**

Typically, the differential diagnosis of pericardial effusion includes pericardial fat, pericardial cyst, pleural effusion and ascites.

Pericardial fat is typically non-circumferential and most prominent along the right heart border; it is heterogeneous in appearance, and moves in concert with the heart. In contrast, pericardial effusion is typically echo lucent, immobile and circumferential with the largest amount of fluid in dependent areas closest to the ground (Fig. 5.1, Panel a).
Pericardial cysts are thin-walled structures containing clear, stationary fluid; they tend to occur in the right atrioventricular groove and have no direct communication with any surrounding structure (Fig. 5.3, Panel a).

Left pleural effusion is located posterior to the left ventricle on parasternal views or lateral to the left ventricle on apical views (Fig. 5.5, Panel a). To differentiate a left pleural effusion from a pericardial effusion, one should pay a special attention to the relationship between the descending thoracic aorta and the left heart border on e.g., parasternal views. The larger the pericardial effusion, the more separation between the descending thoracic aorta and the left heart border there is. In contrast, left pleural effusion does not create an echo lucent area of separation between the descending thoracic aorta and the heart. Because there is no communication between the left and right pleural spaced at the cardiac apex, periapical fluid collection seen on apical views are more likely to be pericardial than pleural in origin.

Right pleural effusion follows the contours of the diaphragm which lies just cranial to the liver boundary. Panel c: Ascites – Transthoracic echocardiogram in the subcostal view demonstrates ascites (asterisk) between the liver and the heart. The presence of the falciform ligament (arrow) helps differentiate ascites from a pericardial effusion. Abbreviations: AV aortic valve, IVC inferior vena cava, LA left atrium, LV left ventricle, RA right atrium, RV right ventricle.

Fig. 5.5 Differential diagnosis of a pericardial effusion. Panel a: Left pleural effusion – Transthoracic echocardiogram in the parasternal long axis view demonstrates a left pleural effusion (asterisk) and a very small pericardial effusion (arrow). Note that unlike the pericardial effusion, the left pleural effusion does not penetrate the space between the heart and the descending thoracic aorta. Panel b: Right pleural effusion – Transthoracic echocardiogram in the subcostal view demonstrates a left pleural effusion (asterisk) and a pericardial effusion (arrow). Note that the right pleural effusion follows the contours of the diaphragm which lies just cranial to the liver boundary. Panel c: Ascites – Transthoracic echocardiogram in the subcostal view demonstrates ascites (asterisk) between the liver and the heart. The presence of the falciform ligament (arrow) helps differentiate ascites from a pericardial effusion. Abbreviations: AV aortic valve, IVC inferior vena cava, LA left atrium, LV left ventricle, RA right atrium, RV right ventricle.

Ascites is a subdiaphragmatic fluid collection containing the falciform (sickle-shaped) ligament.
which anchors the liver to the diaphragm (Fig. 5.5, Panel c).

Alternative Imaging
Echocardiography is typically sufficient to establish the diagnosis and hemodynamic significance of a pericardial effusion.

Imaging with CT and CMR may provide additional anatomic details; particularly related to extracardiac structures and their relationship to pericardial effusion (e.g. type A aortic dissection, mediastinal hematoma post cardiac surgery, thoracic tumor extension into the pericardial space etc.).

EKG has low sensitivity for detection of pericardial effusion; low voltage and electrical alternans are seen only infrequently [12].

Tamponade
Tamponade, originally a French word meaning 'plugging', refers to a clinical syndrome of impaired cardiac filling due to elevation of intrapericardial pressure in the setting of a pericardial effusion. As a clinical entity, cardiac tamponade resulting from a pericardial effusion was first described in the 1930s [13].

Cardiac tamponade is a form of diastolic heart failure. It is important to emphasize that tamponade is not a discrete point in time but rather a process of progressive impairment in cardiac filling extending from an asymptomatic phase to exercise intolerance to hypotension, shock and death. An increase in intracardiac pressures and tachycardia are compensatory mechanisms that delay the progression of tamponade physiology.

Key Anatomic and Hemodynamic Features
The primary determinant of hemodynamic significance of a pericardial effusion is not the size of a pericardial effusion but rather the magnitude of intrapericardial pressure. Cardinal features of tamponade are cardiac chamber collapse (due to extrinsic compression of cardiac walls by pericardial effusion), ventricular interdependence (manifested as exaggerated respiratory variations in cardiac filling), and frequently intracardiac pressure elevation including right atrial pressure elevation leading to a plethoric inferior vena cava.

A normal intrapericardial pressure is close to 0 mmHg or even negative (subatmospheric). In tamponade, intrapericardial pressure exceeds intracardiac pressures for at least part of the cardiac cycle.

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

\[ P = V \times \frac{\Delta P}{\Delta V} \]

Pericardial stiffness, an inverse of pericardial compliance, is the slope of the intrapericardial pressure-volume curve; it demonstrates a nonlinear relationship between the volume of pericardial effusion and the intrapericardial pressure.

At low effusion volumes, the slope is rather flat; initial increases in the size of pericardial effusion lead to only a modest rise in intrapericardial pressure. However, the slope becomes subsequently very steep; at this portion of the curve even a small increase in the size of pericardial effusion leads to marked increases in intrapericardial pressure which, in turn, may precipitate tamponade physiology. Conversely, in a patient with tamponade, even removal of a relatively small amount of pericardial effusion, may promptly relieve signs and symptoms of tamponade.

The location of this pressure-volume curve relative to the x axis (pericardial effusion volume) is dependent on the rate of pericardial fluid accumulation. With acute pericardial effusion (such as with a hemorrhagic effusion in a patient with type A aortic dissection) the curve and the inflection point occur at low volumes; in other words, a relatively small amount of acute pericardial effusion may lead to tamponade physiology. In contrast, with chronic effusion, pericardial stiffness is lower because pericardium has time to adapt to slowly increasing amounts of pericardial fluid and the pressure-volume curve is shifted to the right.

Once the intrapericardial pressure exceeds the intracardiac pressure, extrinsic compression...
by the pericardial fluid leads to invagination of a cardiac wall into its respective chamber (chamber collapse). Aside from chamber collapse, tamponade physiology is also characterized by exaggeration of normal respiratory variations in cardiac filling. Normally, left and right ventricle fill during diastole away from each other and not at the expense of each other; consequently, the interventricular septum stays in the middle during inspiration and expiration.

Pericardial fluid, like any other fluid, is uncompressible. Thus, pericardial effusion constrains ventricular filling and forces the two ventricles to fill at each other’s expense; the impact on each ventricle is dependent on the respiratory phase. During inspiration, the right ventricle fills at the expense of the left ventricle while during expiration the opposite is true. These respiratory variations in cardiac filling are the cardinal feature of tamponade physiology and together with signs of chamber collapse they provide the basis for echocardiographic diagnosis of tamponade.

**Echocardiography Indications**

Primary indications for echocardiography imaging in a patient with tamponade include clinical signs and symptoms of heart failure, hypotension and shock. Heart failure, when present, is typically diastolic with predominance of signs and symptoms of right heart failure (clear lungs, hepatomegaly, ascites, and lower extremity edema).

**Echocardiography Findings**

Echocardiographic diagnosis of tamponade is based on visualization of the three cardinal features of tamponade physiology in the setting of a pericardial effusion: chamber collapse, respiratory variations and elevation of right atrial pressure. Chamber collapse is relatively specific for tamponade physiology, while respiratory variations are also seen in constrictive pericarditis and several other conditions such as labored breathing, pulmonary embolism, obesity and chronic obstructive lung disease (COPD). Because an increase in intracardiac pressures is a compensatory mechanism in both tamponade and constriction, plethora of the inferior vena cava, a sign of right atrial pressure elevation, is frequently seen in tamponade and constriction.

Chamber collapse: Free walls of cardiac chambers invaginate into the chamber when intrapericardial pressure exceeds intracardiac pressure. Because intracardiac pressures are lower in diastole and since the right heart has thinner walls the left heart, it is the collapse of the right ventricular and right atrial free wall that is typically seen is tamponade. Because in cardiac timing ventricular events are used, the ventricular wall collapse is said to occur during ventricular diastole and the atrial wall collapse during ventricular systole [14].

Right atrial collapse: Right atrial collapse (Fig. 5.6, Panels a, b) is more sensitive but less specific than right ventricular collapse for the diagnosis of tamponade. When clinically significant, the right atrial collapse typically lasts at least 1/3 of ventricular systole; this finding was first described in 1983 on 2D transthoracic echocardiography [15].

Right ventricular collapse: Right ventricular collapse (Fig. 5.6, Panels c, d) is more specific but less sensitive than right atrial collapse for the diagnosis of pericardial effusion. Historically, this echocardiographic finding was described earlier than right atrial collapse. It was first reported in 1979 on M mode echocardiography [16] and then in 1982 on 2D transthoracic echocardiography [17].

Because right ventricular diastolic pressures are lowest at the onset of diastole, early diastolic collapse of the right ventricular free wall is an echocardiographic sign of tamponade physiology. The longer the duration of right ventricular collapse, the more pronounced tamponade physiology is.

Left heart collapse: Because the left ventricular wall is the thickest of all cardiac walls its invagination is not commonly seen in tamponade. Because only the distal portion of the left atrium is intrapericardial, it is only this portion that can be involved in tamponade-related collapse. When compression of the extrapericardial portions of the left atrium around the ostia of the pulmonary veins is seen, an alternative diagnosis should be considered.
Respiratory variations: Changes in filling patterns that are phasic with respiration can be observed either by both 2D and Doppler echocardiography. All modern ultrasound systems are capable of recording respiratory cycles (respirometry), typically by measuring chest impedance from existing EKG leads used during echocardiography. Alternatively, an add-on respirometer clipped to the nasal orifice and connected to the ultrasound system may be used. It is important to emphasize that respiratory variations described below refer to normal breathing (negative pressure ventilation) and cannot be applied when patients are intubated and mechanically (positive pressure ventilation).

On 2D, one observes the location of the interventricular relative to the respiratory cycle.

**Fig. 5.6** Tamponade: 2D echocardiographic findings. Transthoracic echocardiogram demonstrates tamponade physiology in a young woman with metastatic breast cancer and a large pericardial effusion (asterisks). Panels a, b: Apical 4-chamber views demonstrate right atrial physiology in tamponade. Characteristic bucking (collapse) of the right atrial free wall occurs during ventricular systole (Panel b) but not during ventricular diastole (Panel a). Note that the mitral and tricuspid valves are closed when right atrial buckling occurs. Panels c, d: Parasternal views demonstrate right ventricular physiology in tamponade. Characteristic buckling (-collapse) of the right ventricular wall occurs during ventricular diastole. Note that the mitral valve is open when right ventricular buckling occurs.

Abbreviations: LA left atrium, LV left ventricle, RA right atrium, RV right ventricle
In tamponade, there is a marked movement of the interventricular septum toward the left ventricle during inspiration. In contrast, the septum moves toward the right ventricle during expiration. Because respiratory rate is typically lower than the heart rate, this septal shift phasic with respiration does not occur with each cardiac beat.

Doppler recordings of mitral and tricuspid inflow, left ventricular outflow, as well as pulmonary and hepatic vein flow may be used to demonstrate respiratory variations in tamponade.

Mitral inflow: Flow velocity recording at the level of mitral valve may be obtained by either pulsed or continuous wave Doppler. Continuous wave Doppler is preferred when there is excessive translational movement of the heart in large pericardial effusions. On mitral inflow recordings one pays particular attention to the changes in the peak E wave velocity that are phasic with respiration (Fig. 5.7, Panel a) as well as to the overall mitral filling pattern of individual cardiac cycles. It tamponade, the peak E wave velocity is the lowest at the first inspiratory beat and the

Fig. 5.7 Tamponade: Doppler and M mode findings. Transthoracic echocardiogram demonstrates tamponade physiology in a young woman with metastatic breast cancer. Panel a: Mitral inflow – Spectral Doppler recordings at the level of mitral leaflet tips with simultaneous respirometry demonstrates marked respiratory variations in the peak velocity of the mitral E wave. Note that the E wave velocity is lower during inspiration (Ins) beat than during expiration (Exp). Panel b: Mitral inflow – Spectral Doppler tracings at faster sweep rate demonstrates morphology of individual mitral inflow flow velocity pattern. Note the abnormal relaxation filling pattern in this young woman; this is consistent with the diagnosis of tamponade as tamponade impedes early diastolic filling of the left ventricle. Panel c: Plethora of the inferior vena cava – M mode recording through the inferior vena cava (IVC) demonstrates an almost complete absence of respiratory variations in the IVC diameter; this is indicative of an elevated right atrial pressure in this patient with tamponade.
highest at the first expiratory beat. The percent respiratory variation in the peak mitral E wave in calculated as follows:

\[
Mitral Respiratory Variation = \frac{E_{\text{expiration}} - E_{\text{inspiration}}}{E_{\text{expiration}}} \times 100 \%
\]

Arbitrarily, a respiratory variation of >30% is considered significant and consistent with tamponade physiology. It is important to emphasize that these respiratory variations are indicative of ventricular interdependence; thus they may be seen in both tamponade and constrictive pericarditis.

One common explanation for these respiratory variations in mitral inflow is based on the anatomic fact that pulmonary veins are outside the pericardial sac. In normal individuals, inspiration is mediated by a drop in intrathoracic pressure. This drop in pressure affects equally the pulmonary veins and the left heart; thus there is no net change in the pulmonary vein to left heart pressure gradient, and no significant change in peak wave velocities.

In tamponade and constriction, there is still normal drop in pulmonary vein pressures during inspiration. However, there is no concomitant lowering of intracardiac pressures during inspiration because either pericardial fluid (in tamponade) or inelastic pericardium (in constriction) isolates the heart from the intrathoracic pressure changes. This results in a decreased pulmonary vein to left heart pressure gradient and lower E wave velocities during inspiration. Decreased filling of the left ventricle then facilitates the shift of the interventricular septum toward the left ventricle during inspiration.

In addition to respiratory variations in the peak velocity of the mitral E wave, one should also pay attention to the overall mitral filling pattern. In tamponade, the impediment to left ventricular filling occurs in early diastole. In constriction, on the other hand, the impediment is in late diastole. Consequently, tamponade (Fig. 5.7, Panel b) is characterized by an impaired relaxation filling pattern (prolonged isovolumic relaxation time, E/A <1 and prolonged deceleration time). In contrast, constriction is characterized by a restrictive filling pattern (short isovolumic relaxation time, E/A >2; short deceleration time, typically <150 ms).

Left ventricular outflow tract: Respiratory variations in peak LVOT velocities are also seen in conditions of ventricular interdependence including tamponade. Just as with mitral inflow recordings, the highest LVOT velocity is observed with the first expiratory beat and the lowest LVOT velocity with the first inspiratory beat. Although there are respiratory variations in LVOT velocities, no specific cutoff value is given in current guidelines.

Tricuspid Inflow: Respiratory variations in the tricuspid inflow are more pronounced and opposite of those in mitral inflow. The highest tricuspid E wave velocity typically occurs with the first inspiratory beat and the lowest with the first expiratory beat. The percent respiratory variation in the peak tricuspid E wave in calculated as follows:

\[
Tricuspid Respiratory Variation = \frac{E_{\text{expiration}} - E_{\text{inspiration}}}{E_{\text{expiration}}} \times 100 \%
\]

Arbitrarily, a respiratory variation with an absolute value of >60% is indicative of cardiac tamponade. Note that the above tricuspid respiratory variation formula results in negative numbers, while the mitral formula results in positive numbers.

Hepatic veins: A normal hepatic flow velocity pattern consists of two antegrade and two retrograde waves. The two antegrade waves are S (systolic) and D (diastolic) waves. Between S and D, there is a small ventricular retrograde (VR) wave. After the D wave and concomitant with the atrial contraction, there an atrial reversal wave (AR) wave. With inspiration, there is augmentation of both S and D waves.

In conditions of ventricular interdependence such as tamponade and constriction, there is a drop in systemic vein to right heart pressure which results in (1) diminished augmentation of antegrade waves during inspiration; D wave is especially affected and may disappear
completely in advanced tamponade; and (2) there is an enhancement of diastolic flow reversal that is most prominent on the first expiratory beat (expiratory flow reversal).

Right atrial pressure elevation: As cardiac compression by pericardial effusion progresses, there is a compensatory increase in all diastolic pressures in the heart (this is referred to as equalization of diastolic pressures on invasive intracardiac pressure recordings). Plethora of the inferior vena cava (IVC) is the primary echocardiographic manifestation of this phenomenon (Fig. 5.7, Panel c). In advanced tamponade, IVC is dilated (>2.1 cm) and collapses less than 50% with inspiration; this is indicative of elevated right atrial pressure (≥15 mmHg).

**Differential Diagnosis**

Differential diagnosis of tamponade includes other conditions with either chamber collapse or ventricular interdependence. Chamber collapse may be seen when there is extrinsic compression of a cardiac chamber by a surrounding structure (such as tumor or aortic aneurysm). Unlike tamponade, these extrinsic compressions occur throughout the cardiac cycles. Ventricular interdependence with respiratory variations is a feature of both tamponade and constriction. Pericardial effusion is present in tamponade but absent in pure constrictive pericarditis. Respiratory variations may also be seen in conditions of labored breathing such as pulmonary embolism, obesity and COPD. IVC plethora is a nonspecific finding and is observed in any condition that leads to right atrial pressure elevation (including right heart failure, constriction and significant tricuspid stenosis or regurgitation).

**Acute Pericarditis**

Acute pericarditis is an inflammatory disorder with numerous etiologies including infections (especially viral), connective tissue disorders and malignancies; it may be idiopathic or may occur after myocardial infarction, cardiac surgery and radiation therapy to the chest. Acute pericarditis is characterized by a combination of chest pain, abnormal auscultatory findings, EKG changes and pericardial effusion. Chest pain is positional and worsens with inspiration (‘pleuritic’ in nature). On auscultation, there is pericardial rub. EKG may demonstrate diffuse ST segment elevations and PR depressions (Fig. 5.8, Panel a).

**Echocardiography Indications**

Patients with acute pericarditis are referred for echocardiography primarily because of chest pain.

**Echocardiography Findings**

Primary echocardiographic finding of acute pericarditis is pericardial effusion which may vary from trace to large. There may be fibrin strands or other signs of organization in the pericardial space (Fig. 5.8, Panel b).

**Differential Diagnosis**

Differential diagnosis of acute pericarditis is essentially the differential diagnosis of pericardial effusion.

**Alternative Imaging**

CT and CMR may demonstrate not only the presence of pericardial effusion but they may also show active inflammation in thickened and non-calcified walls of the pericardium.

**Constrictive Pericarditis**

Constrictive pericarditis (often simply referred to as constriction) is a form of chronic pericarditis that leads to impaired cardiac filling due progressive thickening and calcification of the pericardial wall. Worldwide, tuberculosis is the leading

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782
cause; however, in the developed countries most cases are either idiopathic or related to prior cardiac surgery. Like tamponade, constrictive pericarditis presents clinically as diastolic heart failure with predominance of signs and symptoms of right heart failure (clear lungs, hepatomegaly, ascites, and lower extremity edema).

With respect to concomitant presence of pericardial effusion, constrictive pericarditis may be either noneffusive or effusive. Noneffusive constrictive pericarditis is much more common; thus the terms ‘constrictive pericarditis’ and ‘noneffusive constrictive pericarditis’ are often used interchangeably.

### Noneffusive Constrictive Pericarditis

#### Key Anatomic and Hemodynamic Features

Thickened and calcified pericardium with fusions between the visceral and parietal layers of the pericardium is the key anatomic features of constrictive pericarditis. Free walls of the ventricles which are adjacent to the abnormal pericardium are more constrained in their movement than the interventricular septum which anatomically is not covered by the pericardium.

Hemodynamically, in both tamponade and constrictive pericarditis the heart is constrained by the pericardium which then leads to ventricular interdependence and respiratory variations in cardiac flows. The major hemodynamic difference between tamponade and constrictive pericarditis is the mechanism and the timing of maximum pericardial constraint.

In tamponade the constraint is due to pericardial fluid, while in constrictive pericarditis it is due to inelastic pericardium. In tamponade, the constraint is most prominent during early diastole leading to an abnormal relaxation filling pattern. In constrictive pericarditis, the impediment occurs during late diastole resulting in the so-called restrictive filling pattern. Both tamponade and constrictive pericarditis are characterized by intracardiac pressure elevations.

Another difference between the two is the presence of absent of chamber collapse. In tamponade, the effusion leads to chamber collapse while in constrictive pericarditis the inelastic pericardium does not.

#### Echocardiography Indications

Patient with constrictive pericarditis are referred for echocardiography primarily because they presents with signs and symptoms of heart failure.

#### Echocardiography Findings

The role of echocardiography is to demonstrate the cardinal anatomic and hemodynamic features of constrictive pericarditis: a thickened and calcified pericardium, ventricular interdependence and respiratory variations in cardiac flows. The major hemodynamic difference between tamponade and constrictive pericarditis is the mechanism and the timing of maximum pericardial constraint.

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with respiratory variations in cardiac flows, the so-called restrictive mitral filling pattern, elevated right atrial pressure leading to inferior vena cava plethora, and regional differences in myocardial motion and deformation.

Pericardial thickness & calcifications: As previously noted, transthoracic echocardiography does not have sufficient image resolution to allow for reliable measurements of pericardial wall thickness. In contrast, transesophageal echocardiography may provide measurements of pericardial thickness that are comparable to reference techniques of CT and CMR.

Ventricular interdependence & Respiratory variations: Marked respiratory variations resulting from ventricular interdependence is a cardinal feature of constrictive pericarditis. As described above in the Tamponade section, ventricular interdependence and respiratory variations are not pathognomonic for constrictive pericarditis as they also occur in tamponade. Furthermore, respiratory variation may also occur in several conditions of labored breathing such as COPD, asthma, obesity and pulmonary embolism.

On M mode and 2D imaging, there is characteristic interventricular septal motion phasic with respiration (Fig. 5.9, Panels a, b). As described in details in the Tamponade section above, the interventricular septum moves

![Fig. 5.9](image)

Constrictive pericarditis: echocardiographic & CT findings. Panel a: M mode recording in a patient with constrictive pericarditis demonstrates characteristic respiratory variations in the right and left ventricular chamber sizes due to abnormal interventricular septal motion phasic with respiration. Note that interventricular septum bounces (arrow) toward the left ventricle (LV) during inspiration (Ins) and toward the right ventricle (RV) during expiration (Exp). Panel b: Transthoracic echocardiogram in the parasternal long-axis view demonstrates characteristic diastolic septal bounce of the interventricular septum (arrows) toward the left ventricle during inspiration. Panel c: M mode recordings through a dilated inferior vena cava (IVC) demonstrate complete absence of respiratory variations in the IVC diameter; this is indicative of an elevated right atrial pressure and consistent with the diagnosis of constriction. Panel d: 3D CT rendering of the heart demonstrates large area of pericardial calcifications (arrows). Abbreviations: AV aortic valve, Exp expiration, ILW inferolateral wall, Ins inspiration, LA left atrium, LV left ventricle, MV mitral valve, RV right ventricle.
toward the left ventricle during inspiration and toward the right ventricle during expiration. This then results in cyclical changes in the size of the two ventricles phasic with respiration (the right ventricle is largest and the left ventricle is the smallest at the first inspiratory beat; the opposite occurs in expiration).

On Doppler recordings, respiratory variations in the peak velocity of the E wave of at least 25% at the level of the mitral valve and at least 40% at the level of the tricuspid valve, and are consistent with the diagnosis of constrictive pericarditis (Fig. 5.10, Panel a).

Restrictive mitral filling pattern: In constrictive pericarditis, the overall mitral filling pattern is restrictive (short isovolumic relaxation time; E/A >2; short deceleration of the E wave [typically less than 150 ms]) as shown in Fig. 5.10, Panel b. The short deceleration time of the mitral E wave is the Doppler equivalent of the rapid y descent on central venous pressure recordings.

Inferior vena cava plethora: In both tamponade and constriction, there is a compensatory increase in all diastolic pressures in the heart (this is referred to as equalization of diastolic pressures on invasive intracardiac pressure recordings). Plethora of the inferior vena cava (IVC) is the primary echocardiographic manifestation of this phenomenon (Fig. 5.9, Panel c). In advanced constrictive pericarditis, IVC is dilated (>2.1 cm) and collapses less than 50% with inspiration; this is indicative of elevated right atrial pressure (≥15 mmHg). IVC plethora is consistent with but not pathognomonic for constrictive pericarditis as it may occur whenever the right atrial pressure is elevated (such as tamponade, significant tricuspid stenosis or regurgitation and right heart failure).

Hepatic vein flow velocities: In constrictive pericarditis, there is enhancement of expiratory flow reversal in spectral Doppler tracings of hepatic vein flows (Fig. 5.10, Panel c).

Abnormal mitral annular velocities: In constrictive pericarditis, one pays attention to the ratio between the lateral and medial peak e' velocities as well as to absolute mitral annular tissue e' velocities.

Tissue Doppler e' ratio (Annulus reversus): Normally, the peak velocity of the mitral annular tissue Doppler e' wave is higher at the lateral compared to the medial (septal) annulus. In other words:

Normal individual: Lateral e' > Medial e'

In constrictive pericarditis, pericardial adhesions to the underlying myocardium typically lead to decreased mobility of the lateral annulus; this then leads to lowering of lateral e' velocity below those of the medial e'. Since this is reversed from what is normally seen, the phenomenon is referred to as annulus reversus [19]:

Note the restrictive filling pattern (E/A >2; rapid E wave deceleration time (arrow)) as well as respiratory variations in the peak velocity of the mitral E wave (W wave velocity is higher during expiration (Exp) than during inspiration (Ins)). This is consistent with the diagnosis of tamponade as tamponade impedes preferentially the late diastolic filling of the left ventricle. Panel c: Hepatic vein spectral Doppler tracing demonstrates pronounced expiratory (Exp) flow reversal.
Constrictive pericarditis: Lateral $e'$ < Medial $e'$

[Annulus reversus]

Absolutes $e'$ velocities: It is important to note that despite lowering of lateral $e'$ velocities, both lateral and medical $e'$ velocity may still be within normal limits in constrictive pericarditis. This is in contrast to restrictive cardiomyopathy where mitral annular velocities are low.

Abnormal left ventricular strain: Normally, subendocardial fibers are primarily responsible for the left ventricular longitudinal strain, while subepicardial layers are primarily responsible for the circumferential strain. Given that pericardial adhesions in constrictive pericarditis lead to relative immobilization of epicardial layers of the left ventricle while leaving the subendocardial layers unaffected, the typical strain pattern of constrictive pericarditis consists of (1) diminished circumferential strain; and (2) preserved longitudinal strain. This is in contrast to restrictive cardiomyopathy where longitudinal strain is preferentially affected [20].

**Differential Diagnosis**

Differential diagnosis of constrictive pericarditis includes tamponade, restrictive cardiomyopathy, other forms of respiratory variations in cardiac flows, and other causes of heart failure. Constrictive pericarditis shares marked respiratory variations with tamponade, obesity, pulmonary embolism, COPD, and asthma. Lack of pericardial effusion differentiates non-effusive constrictive pericarditis from tamponade. Furthermore, constrictive pericarditis has a restrictive mitral filling pattern while tamponade has an abnormal relaxation pattern.

Restrictive cardiomyopathy typically does not demonstrate respiratory variations in mitral inflow. Furthermore, mitral annular tissue Doppler velocities are low in restrictive cardiomyopathy but normal in constrictive pericarditis. Unlike constrictive pericarditis, restrictive cardiomyopathy is not characterized by annulus reversus. The E/e' ratio is typically elevated in restrictive cardiomyopathy (indicative of elevated left atrial pressure) but frequently normal in constrictive pericarditis. With respect to left ventricular strain, restrictive cardiomyopathy typically shows diminished longitudinal strain while constrictive pericarditis typically demonstrates diminished circumferential strain.

**Alternative Imaging**

CT and CMR are the gold standard for measuring pericardial thickness. CT is the gold standard for detection of pericardial calcifications (Fig. 5.9, Panel d).

**Effusive Constrictive Pericarditis**

Effusive constrictive pericarditis is a rare form of constrictive pericarditis in which there is presence of both pericardial effusion and increased pericardial thickness. The patient presents initially with pericardial effusion. After removal of pericardial fluid via pericardiocentesis or surgical drainage, the patient remains symptomatic and echocardiography demonstrates findings typical of constrictive physiology described above.

**Pericardial Tumors**

Primary pericardial tumors are rare and typically benign. Most common benign tumors of the pericardium are lipomas, fibromas, hemangiomas, lymphangiomas and teratomas. Primary malignancies of the pericardium include mesothelioma and various forms of sarcoma.

Secondary tumors of the pericardium are much more common than the benign tumors; metastatic seeding of the pericardium may be seen with lymphomas and melanomas as well as with malignancies of the breast, lung, stomach or colon.

**Echocardiography Indications**

Pericardial tumors may be an incidental finding or the patient may present with nonspecific symptoms such as chest pain, palpitations or shortness of breath.
Echocardiography Findings

Benign pericardial tumors typically present as circumscribed masses adherent to the pericardium. Lipomas and fibromas are typically solid in appearance while teratomas, hemangiomas and lymphangiomas may demonstrate cysts and septations (Fig. 5.11, Panel a).

Malignant pericardial tumors are often diffuse and typically present with pericardial effusion that demonstrates signs of organization. These tumors typically are confined to the pericardium and do not infiltrate the myocardium. Melanomas are a notable exception as they demonstrate widespread myocardial involvement.

Circumscribed pericardial tumors may cause extrinsic compression of surrounding cardiac chambers while diffuse tumors may lead to constrictive physiology (Fig. 5.11, Panel b) [21].

Differential Diagnosis

Pericardial tumors need to be differentiated from other mediastinal masses; identification of points of mass attachment to the pericardium is an important clue that may differentiate pericardial from mediastinal tumors. Differential diagnosis of pericardial malignancies often includes pericardial effusion, tamponade and constrictive pericarditis.

Alternative Imaging

CT and CMR provide incremental value in localization and tissue characterization of pericardial tumors.

For Patients and Their Family

Information regarding the echocardiographic exam in general and findings specific to individual disorders is provided below.

Echocardiogram: You doctor may order an echocardiogram to examine the heart and the envelope that surrounds the heart called the pericardium. Echocardiogram used ultrasound wave to create an image of the heart. Ultrasound wave are similar to regular sound waves except that human ears cannot hear them. Ultrasound imaging has been performed for more than 50 years and diagnostic ultrasound wave have not been shown to cause any harm to humans.

Typical echocardiogram is called transthoracic echocardiogram, often abbreviated as TTE. During TTE you will be lying on a bed. A technologist or a physician will apply a small amount of nontoxic gel to your skin and then place a small probe to various points of your chest. The exam is not painful.
If TTE imaging is not sufficient, your doctor may order a different type of echocardiogram called transesophageal echocardiogram, often abbreviated as TEE. During a TEE, you will be lying on a bed. First you will be given medications through your veins; these medications will make you sleepy and will numb the pain. You will then be asked to swallow a small ultrasound camera mounted on a cable that is attached to the ultrasound machine. After swallowing, the camera will be placed into your food pipe (also called esophagus) and the stomach. Overall, TEE is considered a safe procedure and complications are unusual.

TTE and TEE may demonstrate a completely normal pericardium or may demonstrate one of the abnormal findings described above.

Congenital absence of the pericardium: Normally, the heart is surrounded by a protective envelope called pericardium. Very rarely, a person may be born without some or all parts of the pericardium. Doctors and other medical professionals call this condition 'congenital absence of the pericardium'. With this condition you may not feel anything unusual and the absent pericardium is often detected by chance, for instance on a pre-employment chest X-ray or on an echocardiogram ordered for a different reason (heart murmur, for instance). Based on echocardiogram and other forms of heart imaging, your doctor will be able to tell you if any treatment is necessary.

Pericardial cyst: Echocardiogram may show that you were born with a pouch filled with clear fluid called pericardial cyst. It is located near the heart. This is a rare condition and often causes no harm. Occasionally, people who have a pericardial cyst may notice shortness of breath, chest pain or abnormal heartbeat. If your discomfort is significant, your doctor may consider referring you to a surgeon who may need to perform a surgery to remove the cyst.

Pericardial effusion: Normally there is no significant amount of fluid inside the pericardial sac, the space between the heart and the envelope of the heart called pericardium. When fluid accumulates inside the pericardial sac, doctors and other medical professionals refer to that fluid as pericardial effusion. There are many causes of pericardial effusion; medical professionals taking care of you will tell you what the most likely cause of your pericardial effusion is. Sometimes, if you have a pericardial effusion you may feel nothing unusual (doctors would say that your pericardial effusion is asymptomatic). In other instances, pericardial effusion may lead to shortness of breath, chest pain, rapid heartbeat and low blood pressure.

It is important that you consult your doctor when you are diagnosed with a pericardial effusion; although you may originally feel nothing unusual, pericardial effusion is some instances may lead to a more serious condition called tamponade.

Tamponade: Tamponade is a very serious condition in which the fluid around the heart called pericardial effusion (see above) interferes significantly with your heart function. In tamponade, you may feel your heart racing or your blood pressure may be low and you may even pass out. If you are diagnosed with tamponade, please take that finding very seriously as you may die without prompt treatment. To treat tamponade, doctors may need to remove the pericardial fluid promptly from your chest. Fluid removal can be accomplished by placing a needle through the chest into the pericardial sac; this procedure is called pericardiocentesis. If pericardiocentesis is not successful, you may need to undergo chest surgery to create a hole in the pericardium; this is called a pericardial window procedure.

Acute pericarditis: After undergoing an echocardiogram you may be told that you might have pericarditis. This condition has many causes and in many instances it resolves with no lasting effects.

Constrictive pericarditis: Your echocardiogram may show that you have constrictive pericarditis. This condition develops over months or even years and leads to thickening and calcium deposits in the pericardium. You may have been referred for an echocardiogram because you experienced swelling of your legs, fatigue and shortness of breath. If your echocardiogram showed findings of constrictive pericarditis, you doctors may decide to wait and watch or they may refer you to surgeon who will consider surgical
removal of diseased pericardium during an open heart surgery.

Pericardial tumor: Your echocardiogram may demonstrate a mass inside the envelope of the heart called the pericardium. Such a mass could be a benign tumor or may represent a malignancy. Sometimes your echocardiogram will show abnormal fluid around the heart called pericardial effusion. In other instances, the echocardiogram may show thickening of the pericardium called constrictive pericarditis. Pericardial effusion and constrictive pericarditis are described in more detail above.

References


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