Pericardium

14

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Abbreviations

2D	Two-dimensional
CP	Constrictive pericarditis
RA	Right atrium
RAP	Right atrial pressure
RCM	Restrictive cardiomyopathy
TEE	Transesophageal echocardiography
TTE	Transthoracic echocardiography

Introduction

Basic perioperative transesophageal echocardiography (TEE) guidelines suggest that knowledge of the echocardiographic manifestations of the pericardium and its associated pathologies are essential to the basic perioperative echocardiographer [1]. This chapter will review the anatomy and physiology of the pericardium, essential transesophageal and transthoracic echocardiographic (TTE) views for evaluation, and major pathologies of the pericardium, including pericardial effusion, pericardial hematoma, cardiac tamponade, and constrictive pericarditis.

Anatomy and Physiology of the Pericardium

Anatomy

The pericardium is a fibrous sac surrounding the heart and proximal great vessels with an inner serous component and an outer fibrous component (Fig. 14.1a, b). The serous component is further divided into visceral and parietal layers. Each of these serosal layers is composed of a single layer of mesothelial cells and a surrounding layer of loose connective tissue. The visceral layer is an inner, thin, translucent layer, which is adherent to the epicardium. The parietal layer is an outer, thicker layer, which is adherent to the fibrous component. This outer fibrosa attaches to the sternum anteriorly and diaphragm inferiorly.

The visceral and parietal layers exist in continuity and join together at reflections points, called sinuses. The transverse sinus is created by

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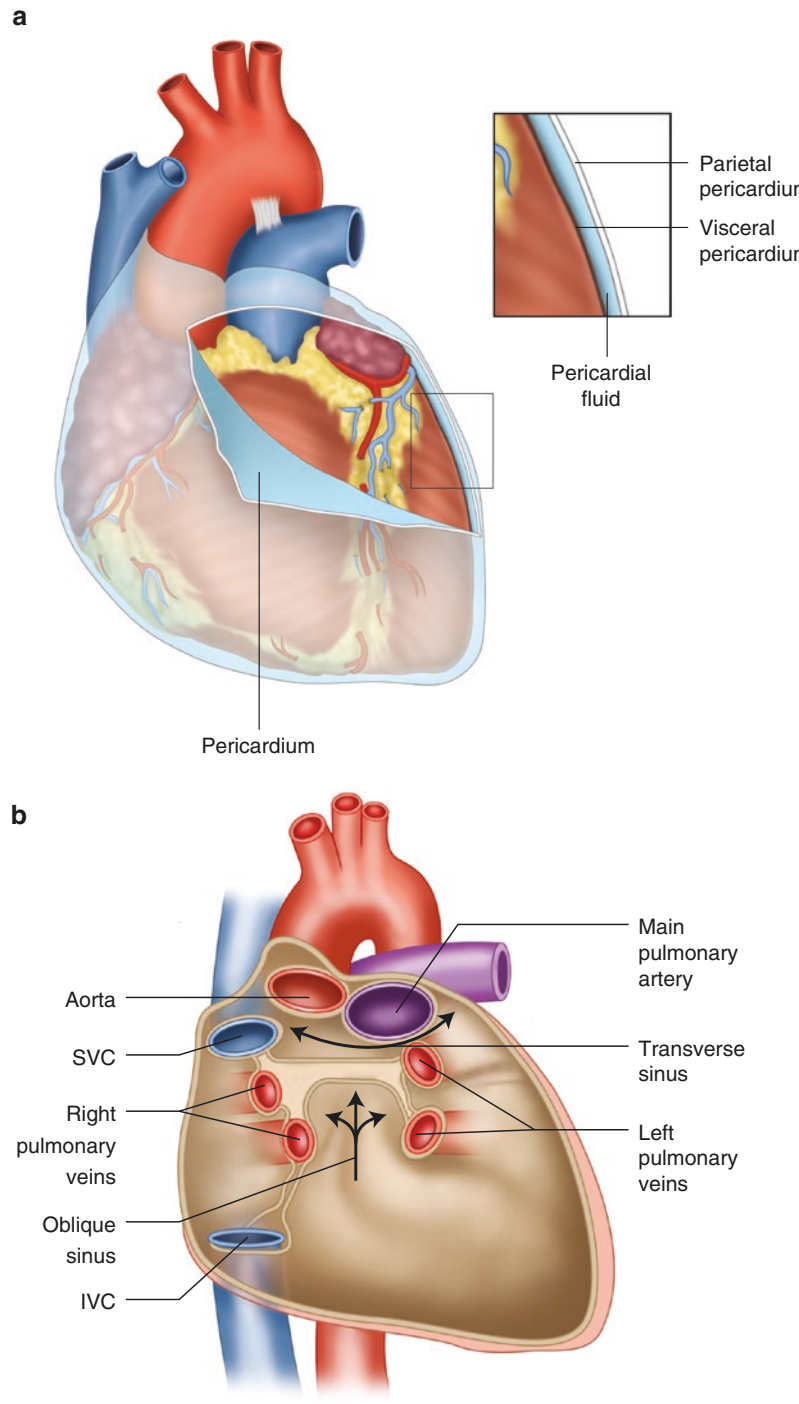

Fig. 14.1 (**a**) Anatomical drawing of the pericardium surrounding the cardiac structures. The zoomed-in portion demonstrates the parietal and visceral components of the pericardium separated by pericardial fluid. (**b**) Anatomical drawing of the pericardial space with the heart removed, demonstrating the relationship of the pericardium to the major vessels of the heart and the sinuses created at its reflection points

reflections at the base of the heart anterior to the superior vena cava and posterior to the great vessels. The oblique sinus is created by reflections posterior to the left atrium between the inferior vena cava and pulmonary veins (Fig. 14.1). The potential space created between the visceral and parietal layers is referred to as the pericardial cavity. Under normal physiologic conditions, this cavity contains between 5 and 50 mL of serous pericardial fluid.

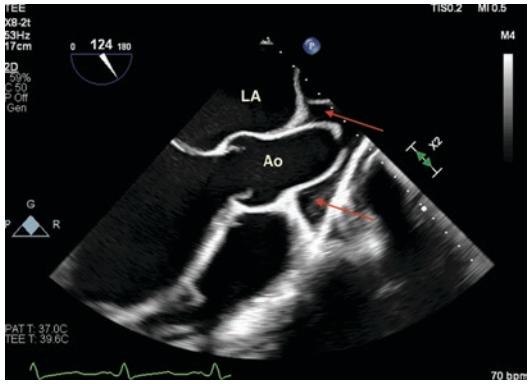

Fig. 14.2 Midesophageal long-axis view. The *red arrows* point to the transverse sinus. *LA* left atrium, *Ao* aorta

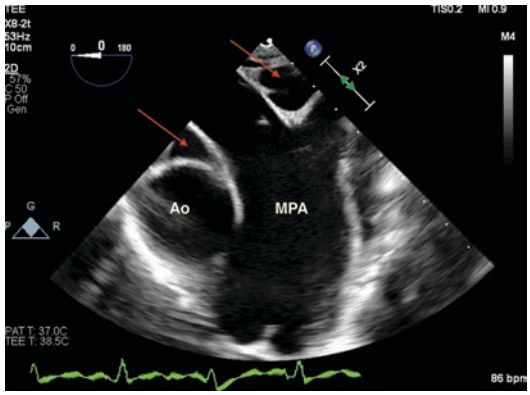

Fig. 14.3 Midesophageal ascending aorta short-axis view. The *red arrows* point to the transverse sinus. *Ao* aorta, *MPA* main pulmonary artery

Transverse Sinus

The transverse sinus is the potential space at the base of the heart anterior to the superior vena cava, superior to the atria, and posterior to the great vessels. While routinely visible when a pericardial effusion is present, the transverse sinus may be visible even when a physiologic amount of fluid is within the pericardial cavity. Fluid within the sinus can be readily appreciated as an echolucent space located between the left atrium, ascending aorta, and pulmonary artery. In the midesophageal two-chamber view, an echolucent space may be visible surrounding the left atrial appendage. In the midesophageal right ventricular inflow-outflow view, an echolucent space may be visible between the aortic

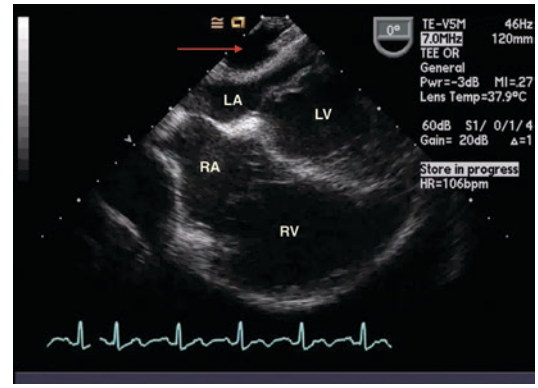

Fig. 14.4 Midesophageal four-chamber view. The *red arrow* points to the oblique sinus. *RV* right ventricle, *LV* left ventricle, *RA* right atrium, *LA* left atrium

valve and main pulmonary artery. In the midesophageal long-axis (Fig. 14.2; Video 14.1) and ascending aorta short-axis views (Fig. 14.3; Video 14.2), an echolucent space may be seen between the ascending aorta and right pulmonary artery [2]. When evaluating the sinus on echocardiography, it is important to note that hypoechoic epicardial fat may be confused with fluid accumulation or an abscess in this space.

Oblique Sinus

The oblique sinus is a thin pericardial space located posterior to the left atrium between the inferior vena cava and pulmonary veins. While the transverse sinus may be visible even with a physiologic amount of fluid, the oblique sinus is more commonly visualized when there is an abnormal fluid collection within this space. With TEE, the oblique sinus can be visualized at the level of the mid-esophagus with a collection of fluid noted posterior to the left atrium, between the left atrium and the apex of the echocardiographic window (Fig. 14.4; Video 14.3). Due to the proximity of the left atrium to the probe and the ultrasound sector width unable to visualize the entire posterior wall of the chamber, the full extent of fluid accumulation in the oblique sinus may not be appreciated with TEE [2]. Notably, post-cardiac surgery, this dependent space may accumulate blood or form a hematoma from surgical cannulation sites.

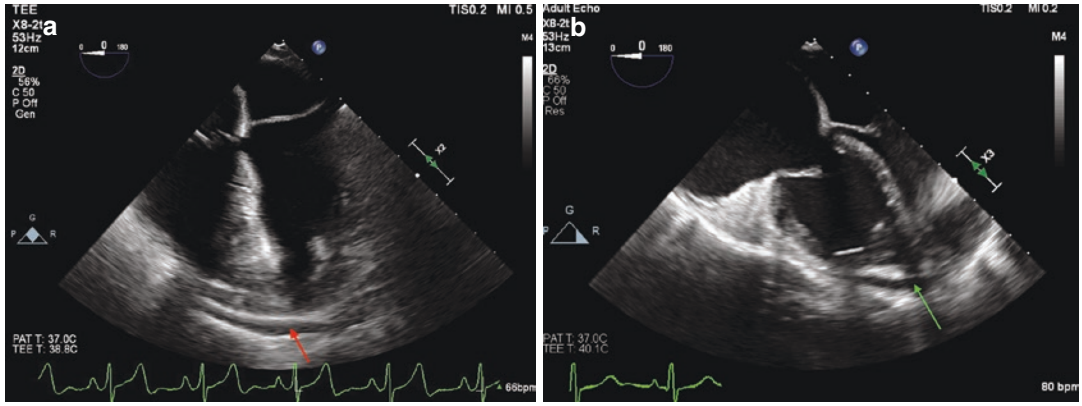

Fig. 14.5 (**a**) Midesophageal four-chamber view. The *red arrow* points to an epicardial fat pad. (**b**) Midesophageal four-chamber view with a *green arrow* pointing to a small pericardial effusion for comparison

Epicardial Fat Pad

In patients with increased visceral deposition of fat, an epicardial fat pad may be apparent in the pericardial space. Typically, this is visualized along the atrioventricular groove, the interventricular groove, or the right ventricular free wall. Epicardial fat contains coronary arteries, lymphatics, and nerve tissue. In comparison to the underlying myocardium, epicardial fat is less echogenic. Epicardial fat often has an echogenicity closer to blood, and therefore it may be difficult to distinguish from a small pericardial effusion (Fig. 14.5a, b). However, in contrast to an effusion, which is echolucent and motionless, epicardial fat is echogenic and moves in concert with the myocardium [3]. Additionally, epicardial fat has a subtle speckled appearance often lacking in effusions and is never associated with cardiac chamber collapse. In contrast to epicardial fat, which is located between the myocardium and visceral pericardium, pericardial fat may also be seen, which is adherent and external to the parietal pericardium (Fig. 14.6; Video 14.4) [4].

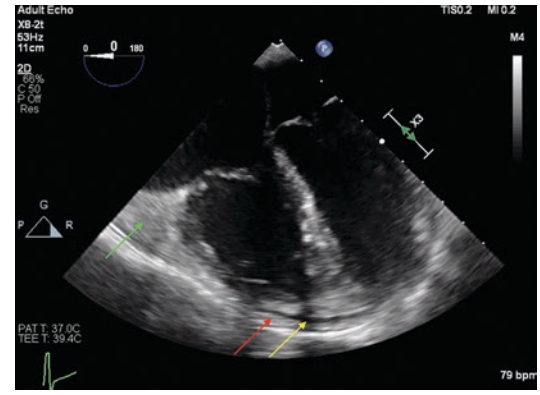

Fig. 14.6 Midesophageal four-chamber view. The *green arrow* points to epicardial fat located within the atrioventricular groove, internal to the visceral pericardium, while the *red arrow* points to pericardial fat located external to the parietal pericardium. A trace amount of pericardial fluid highlights the separation of the visceral and parietal layers (*yellow arrow*)

Function

There are several functions of the pericardium. The mesothelial cells secrete a plasma ultrafiltrate, to lubricate the heart and allow normal rotation and translation. They also hypertrophy in response to chronic fluid accumulation to allow expansion of the pericardial cavity. The thick and dense fibrosa anchors the heart within the chest and protects it from surrounding structures and infection. By encasing the heart, the pericardium serves to constrain filling of cardiac chambers, preventing overdistension and atrioventricular valvular incompetence [5].

Respirophasic Variation

Direct transmission of pressures from the intrathoracic cavity to the pericardial space alters car-

diac filling during both spontaneous negative pressure breathing and mechanical positive pressure ventilation. This is termed respirophasic variation. In a patient with normal pericardium, there is no increase in transmural pressure; therefore intrathoracic and pericardial pressures are approximately equal. The true filling pressure of a cardiac chamber is determined by the transmural pressure which is the intracardiac pressure minus the pericardial pressure:

$$\text{Effective RA filling pressure} = \text{RAP} - \text{Pericardial Pressure}$$

RA right atrium, *RAP* right atrial pressure

As pericardial pressure is nearly equal to intrathoracic pressure, true RA filling pressure can be determined by:

$$\text{True RA filling pressure} = \text{RAP} - \text{Intrathoracic Pressure}$$

Therefore, negative intrathoracic pressure (spontaneous inspiration) will augment filling by increasing the true RA filling pressure, and positive intrathoracic pressure (mechanical inspiration) will reduce filling by lowering the RA filling pressure.

During spontaneous inhalation, a more negative intrathoracic pressure increases venous return to the right heart and subsequently increases right ventricular stroke volume and output. Additionally, negative intrathoracic pressure increases pulmonary venous pooling and left ventricular afterload, and in turn, there is decreased left heart filling and output. During spontaneous exhalation, a less negative intrathoracic pressure causes the opposite effect, with reduced right heart filling and output and increased left heart filling and output. During positive pressure ventilation, the opposite filling pattern is true for both inhalation and exhalation, where inspiration leads to a more positive intrathoracic pressure with reduced right heart filling and a "squeeze" of pulmonary blood into the left heart with a subsequent increase in left heart filling. These changes are reflected in the spectral Doppler patterns of the transtricuspid and transmitral inflow velocities (Fig. 14.7). Techniques for obtaining and interpreting these patterns are found in subsequent sections of this chapter and in Chap. 20.

Echocardiographic Views of the Pericardium

When visualizing the pericardium with echocardiography, the layers appear as thin, echogenic lines that surround the myocardium. Echocardiography can be used to measure the thickness of these layers, with the normal thickness being less than 2–3 mm. As mentioned, the pericardial sac normally contains a small amount of fluid interposed between these layers, which may result in a very small physiologic separation. The separate layers will be best visualized during systole as the heart contacts, with the heart and visceral pericardium sliding within and independently from the parietal pericardium. Due to the motion of the heart throughout the cardiac cycle and limitations in image quality with echocardiography, it may be difficult to distinguish between these layers. In pathologic conditions, as the layers thicken, and as fluid accumulates within the pericardial space, these anatomic features become more visible.

TEE and TTE Views

In comparison to TTE, TEE is better suited for imaging of the pericardium, as it avoids the echogenic attenuation caused by epicardial fat. When compared to computed tomography, TEE was found to be more reliable than TTE for the measurement of pericardial thickness [3].

As the pericardium encases the heart and its proximal great vessels, there are multiple basic views on two-dimensional (2D) TEE that are useful for a global assessment of the pericardium. The midesophageal four-chamber, two-chamber, long-axis, right ventricular inflow-outflow, and bicaval views allow for assessment and diagnosis of pericardial disease location, pericardial thickness, fluid collection size, and collection characteristics. Together, these views allow the echocardiographer to determine disease extent and potential suitability for procedures such as pericardiocentesis.

The midesophageal four-chamber view demonstrates the pericardium surrounding the right atrium, left atrium, right ventricular free wall in long-axis,

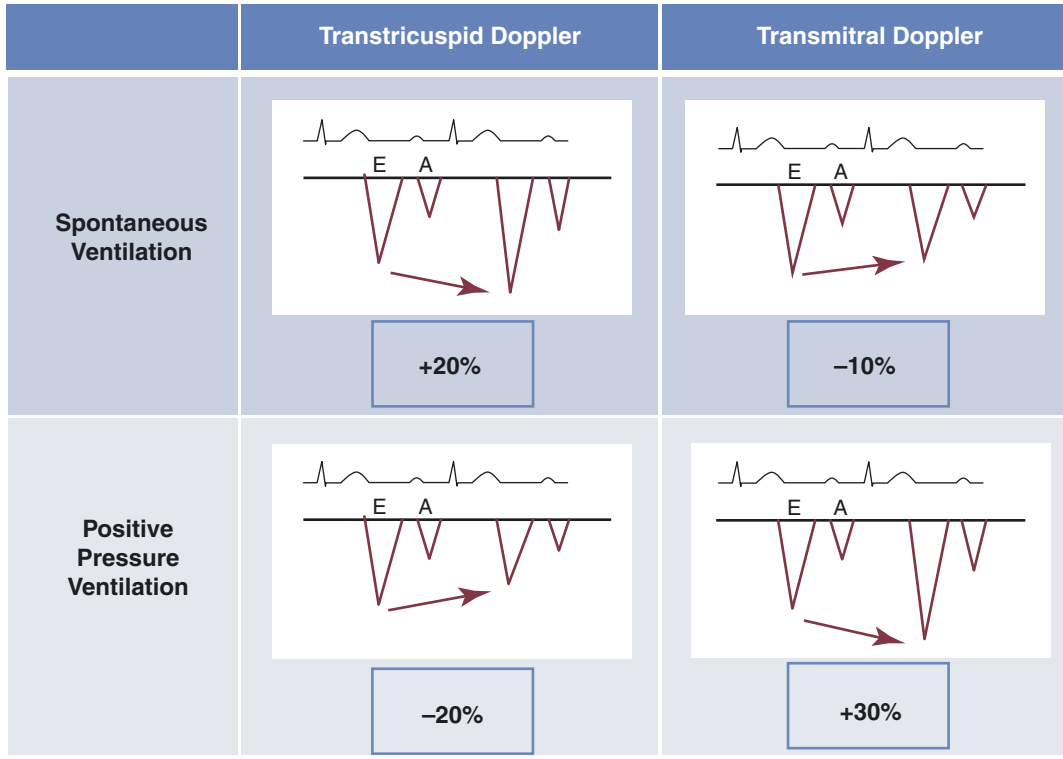

Fig. 14.7 Spectral Doppler patterns of the transtricuspid and transmitral inflow demonstrate normal respirophasic variation during inspiration for both spontaneous ventilation and positive pressure ventilation

and left ventricular anterolateral wall (see Chap. 2) and is helpful in identifying pericardial thickness, effusion, and chamber collapse in tamponade. The midesophageal two-chamber view demonstrates the pericardium surrounding the left atrium, left ventricular inferior wall, and left ventricular anterior wall. The midesophageal long-axis view demonstrates the pericardium surrounding the left atrium, ascending aorta, left ventricular inferolateral wall, and right ventricular free wall. The midesophageal right ventricular inflow-outflow view demonstrates the pericardium surrounding the right ventricular free wall in short-axis and is particularly helpful in identifying right ventricular diastolic collapse in tamponade. The midesophageal bicaval view demonstrates the pericardium surrounding the right atrium and is notably helpful in identifying right atrial collapse. At the transgastric level, the mid-papillary short-axis view also assists in evaluation of the pericardium when determining if pathology exists regionally or circumferentially [1].

Transthoracic echocardiography can provide similar information regarding the pericardium, often with very similar perspectives. When visualizing the structures identified and portions of the pericardium with TTE, the apical four-chamber view corresponds to the midesophageal four-chamber view, the parasternal long-axis view corresponds to the midesophageal long-axis view, and the parasternal short-axis view corresponds to the transgastric short-axis view (see Chap. 3).

For evaluation of inflow patterns by spectral Doppler, the chosen view will depend on optimal alignment of the Doppler cursor with the tricuspid and mitral inflows. Evaluation of tricuspid valve inflow typically occurs in a midesophageal four-chamber or modified bicaval tricuspid valve view with TEE, while an apical four-chamber view is often well-aligned for Doppler with TTE imaging. Evaluation of the mitral valve inflow, with placement of the pulsed-wave Doppler sample volume at the mitral valve leaflet tips, is in the

midesophageal four-chamber with TEE, while this usually occurs in an apical four-chamber view with TTE imaging.

Indications for Echocardiography

The application of echocardiography is useful to diagnose pericardial disease, inform clinical management, and guide the success of various cardiac procedures, such as creating a pericardial window, pericardiocentesis, and pericardiectomy. As such, consensus guidelines recommend the use of echocardiography for evaluation of patients with suspected pericardial disease, including effusion, tamponade, or constrictive pericarditis, evaluation of patients with suspected bleeding into the pericardial space, and follow-up evaluation for recurrence of a pathologic pericardial process [3, 6, 7].

Pericardial Effusions (Highlight Box 14.1)

Highlight Box 14.1

Pericardial effusions	
2D	• Echolucent space between myocardium and pericardium • Semiquantification of effusion size • Circumferential vs. loculated/regional • Presence of clot or hematoma • Systolic right atrial collapse • Diastolic right ventricular collapse
CFD	• Typically not utilized
Spectral	• Transtricuspid inflow velocities (PWD) • Transmitral inflow velocities (PWD) • Exaggeration of respirophasic variation during spontaneous ventilation

PWD pulsed-wave Doppler

Pericardial effusions occur when the amount of fluid within the pericardial space exceeds the expected physiologic amount. Echocardiography is the initial imaging modality recommended for evaluation of a suspected effusion due to its accessibility, accuracy in diagnosis, and ability to describe its physiologic effects [3]. The most important elements of the echocardiographic exam with regard to an effusion are determining the size, location, its circumferential or loculated nature, and its hemodynamic significance.

With echocardiography, pericardial effusions are often appreciated as an echolucent stripe between the visceral and parietal layers of the pericardium. A linear measurement of this stripe perpendicular to the myocardial wall at end-diastole provides a semi-quantitative estimate of the effusion volume. Effusions measuring less than 10 mm are considered small (some sources consider < 5 mm "minimal" and 5–10 mm "small"); 10–20 mm are considered moderate; and > 20 mm are considered large (Table 14.1) [8] .

Optimal imaging planes depend on the location of the effusion and whether it is circumferential (Fig. 14.8a, b) (surrounding the entire heart) or loculated (Fig. 14.9) (adjacent to a specific cardiac chamber[s]). When located anteriorly, effusions can be better appreciated in the midesophageal or parasternal long-axis views. When located posteriorly or circumferential, effusions can be better appreciated in the transgastric short-axis or parasternal short-axis views. To optimize the imaging sector, decreasing the gain setting will allow the echocardiographer to better visualize the pericardial interface, which is brightly reflective.

Pericardial Hematoma

Depending on the etiology, an effusion may be composed of differing fluid types, including transudate (serous), exudate (cells), hemopericardium (blood), or pyopericardium (pus) [3]. Distinguishing between different types of collections within the pericardial space requires experience; however, certain characteristics may aid in diagnosis. While purely transudative or exudative fluids are often visualized as an anechoic space, fibrin stranding may be evident in long-standing effusions or early clot formation, and slow swirling of spontaneous echo contrast may be evident

Table 14.1 Pericardial effusion severity and semi-quantification of volume by transverse measurement on echocardiography

	Pericardial Effusion Severity			
	Minimal	**Small**	**Moderate**	**Large**
Transverse Measurement (mm)	< 5	5 – 10	10 – 20	> 20
Approximate Volume (mL)	50 – 100	100 – 250	250 – 500	> 500

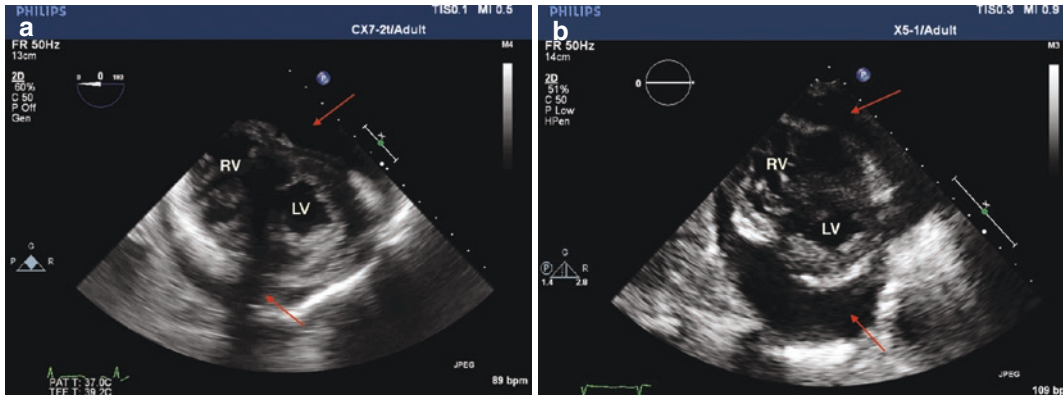

Fig. 14.8 The *red arrows* point to a circumferential pericardial effusion seen in transgastric short-axis (**a**) and parasternal short-axis views (**b**). *RV* right ventricle, *LV* left ventricle

with clumping of red blood cells (Fig. 14.10). A hemopericardium may evolve to form a thrombus, in which the collection has an echodensity approaching that of the myocardium (Fig. 14.11).

Cardiac Tamponade

The physiologic effects of a pericardial effusion depend on the size of the effusion and rate of fluid accumulation. Mesothelial cells of the serous pericardium hypertrophy to accommodate chronically expanding effusions, such that even large effusions may cause very little increase in pericardial pressure. Conversely, minimal to no hypertrophy occurs in rapidly expanding effusions (e.g., hemorrhagic), and even small effusions may cause markedly increased pericardial pressures, leading to tamponade. Accordingly, whether an effusion is large or small, once a threshold point is reached, small changes in volume will lead to large variations in pericardial pressure (Fig. 14.12) [9].

Cardiac tamponade is a clinical diagnosis and occurs when pericardial fluid accumulation impedes normal cardiac filling, which results in impaired hemodynamics. Increases in pericardial pressure are transmitted across the transmural space, and when they exceed the intracavitary pressure, collapse of the cardiac chamber occurs. The sequence of chamber collapse corresponds with the lowest intracavitary pressures (right atrium, then right ventricle) until there is equalization of the mean diastolic pressures across all chambers (equal to the pericardial pressure) and diminished cardiac output ensues. In the absence of tamponade, brief collapse

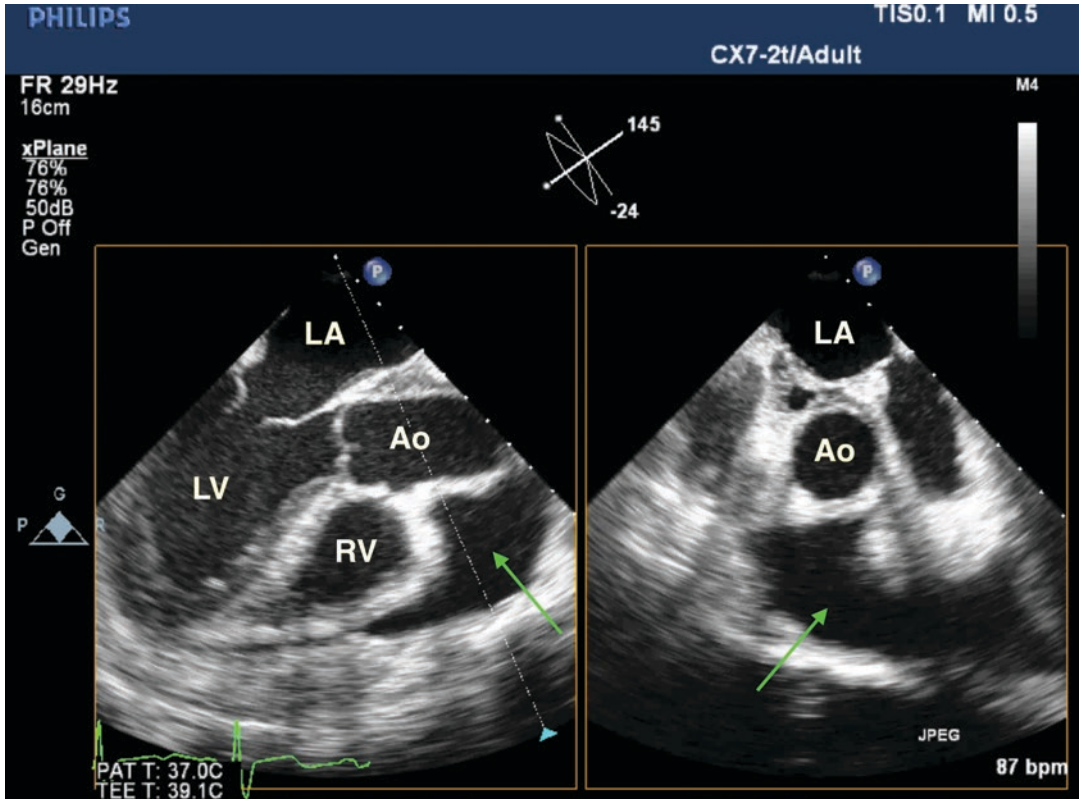

Fig. 14.9 Midesophageal long-axis view (left) with a 90-degree cross-plane image (right) at the level of the ascending aorta as indicated by the cursor. The *green arrow* points to a loculated pericardial effusion located anterior to the heart. *LA* left atrium, *Ao* aorta, *LV* left ventricle, *RV* right ventricle

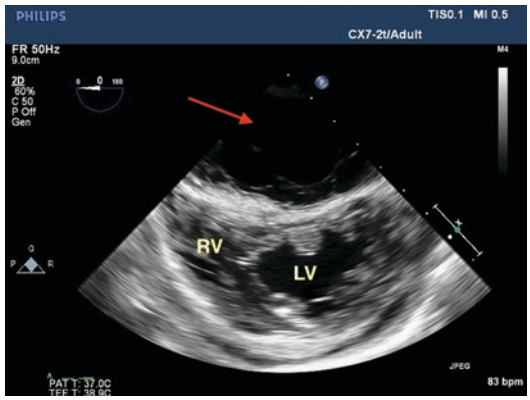

Fig. 14.10 Transgastric short-axis view. The *red arrow* points to a pericardial effusion located posterior to the left ventricle in which fibrin stranding can be seen, suggestive of early clot formation. *RV* right ventricle, *LV* left ventricle

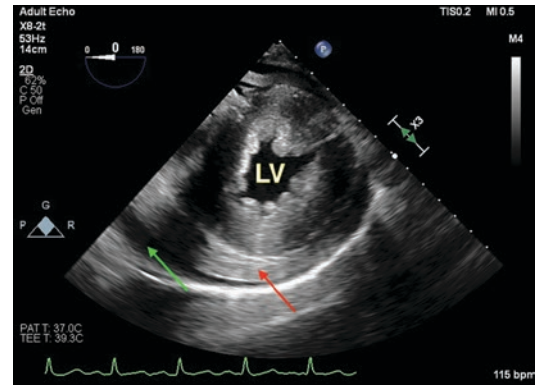

Fig. 14.11 Transgastric short-axis view. The *red arrow* points to a pericardial hematoma, while the *green arrow* points to pericardial effusion. *LV* left ventricle

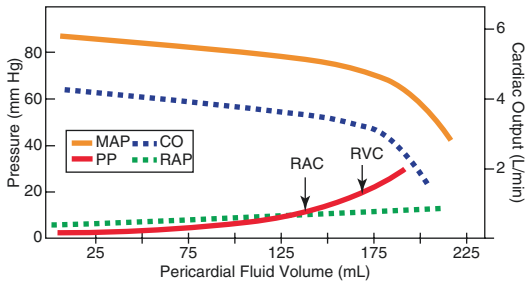

Fig. 14.12 Graphic relationship of increasing pericardial fluid volume to cardiac hemodynamics. *MAP* mean arterial pressure, *PP* pericardial pressure, *CO* cardiac output, *RAP* right atrial pressure, *RAC* right atrial collapse, *RVC* right ventricular collapse. (Reproduced with permission from Savage et al. [16])

of the right atrial free wall during diastole may occur in patients with low right atrial pressures. However, when the duration of right atrial collapse exceeds one-third of the cardiac cycle, it is 94% sensitive and 100% specific for tamponade [10]. The presence of collapse of the right ventricular free wall during diastole also suggests tamponade with the sensitivity and specificity improving as the duration of diastolic collapse increases [3]. Visualization of right atrial and right ventricular collapse is best observed from the midesophageal four-chamber and right ventricular inflow-outflow views (TEE) or the apical four-chamber view (TTE). Left atrial collapse (although rare), when present, is often visualized in conjunction with right atrial collapse. The thicker and higher-pressured left-sided structures are less likely to collapse, and when collapse is seen, it portends a bad outcome. The absence of collapse of any cardiac chamber has a negative predictive value of > 90% for tamponade [11].

During cardiac tamponade, the filling of the ventricles becomes dependent on each other, termed ventricular interdependence. As the patient spontaneously inspires, right ventricular filling is augmented. Due to higher pericardial pressure, the right ventricular filling deviates the septum toward the left, thereby impeding left ventricular filling. During expiration, the opposite occurs. Left ventricular filling increases deviating the septum toward the right and thereby impeding right ventricular filling. This phenomenon allows the spectral Doppler evaluation in spontaneous ventilating patients to help confirm the diagnosis of tamponade. As mentioned above, the variation of transtricuspid or transmitral inflow velocities is exaggerated in spontaneous negative pressure ventilation, and velocities are overall reduced in mechanical positive pressure ventilation.

The midesophageal four-chamber or apical four-chamber views allow for optimal alignment of the Doppler sample volume with the atrioventricular valves. To calculate the variation, place the pulsed-wave Doppler sample volume at the level of the atrioventricular valve leaflets, and set a sweep speed of 25–50 mm/sec to allow more beats per screen to be visualized. Using the peak E-wave velocities at expiration and inspiration, the percentage of respirophasic variation is calculated:

$$[\text{Percentage of variation} = 100\% \times (\text{expiration velocity} - \text{inspiration velocity})/\text{expiration velocity}]$$

While patients with normal physiology demonstrate a reduction in transmitral velocities of approximately 10% during spontaneous inspiration, an exaggerated reduction of > 30% of the peak mitral E-wave velocity is suggestive of tamponade (Figs. 14.13 and 14.14; Video 14.5) [3].

Regional cardiac tamponade may also be seen, in which a loculated effusion or localized hematoma causes collapse of adjacent cardiac chambers. This is often seen in the post-cardiac surgery patient where localized bleeding collects and can compress individual cardiac chambers. This may occur near cannulation sites, such as the right atrium from venous cannulation or the left atrium from vent placement or aortic cannulation. Lastly, isolated-left heart tamponade may also occur in patients with severely elevated right-sided pressures, such as in severe pulmonary hypertension, in which an elevated pericardial pressure leads to collapse of the left atrium

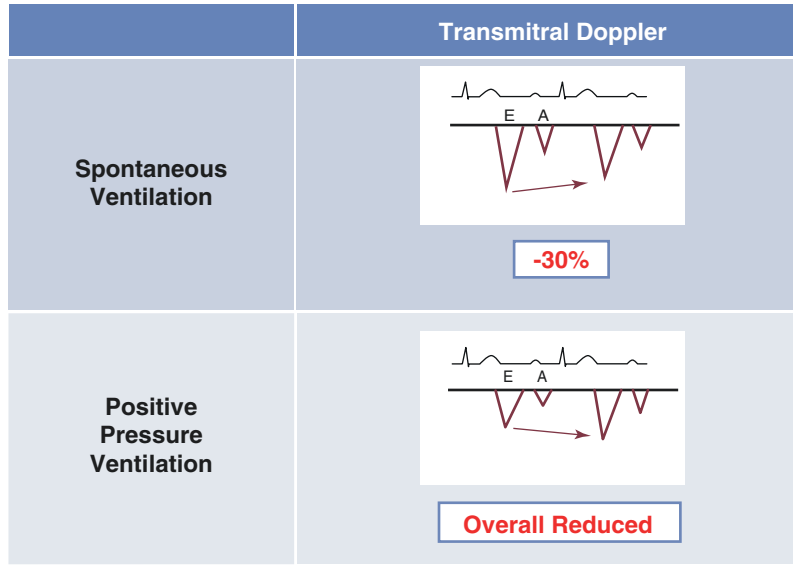

Fig. 14.13 In cardiac tamponade, spectral Doppler patterns of the transmitral inflow demonstrate exaggerated respirophasic variation during spontaneous inspiration and overall reduced inflow during positive pressure ventilation

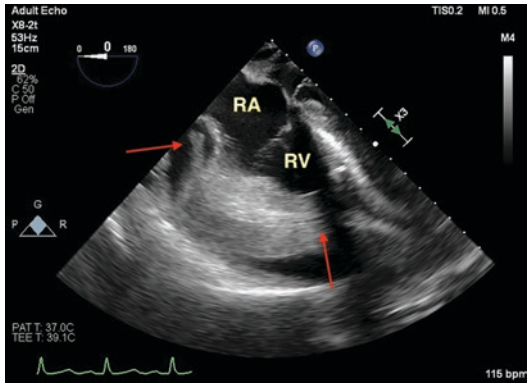

Fig. 14.14 Midesophageal four-chamber view with a right ventricular focus. The *red arrows* point to a complex pericardial effusion, with collapse of the free walls of the right atrium and right ventricle visualized. *RA* right atrium, *RV* right ventricle

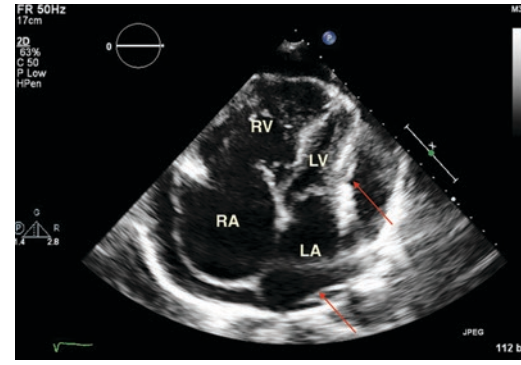

Fig. 14.15 Apical four-chamber view. A circumferential pericardial effusion can be seen with isolated collapse of the left-sided chambers (*red arrows*) in a patient with severe pulmonary hypertension. *RV* right ventricle, *LV* left ventricle, *RA* right atrium, *LA* left atrium

and/or left ventricle without collapse of the right-sided chambers (Fig. 14.15). These clinical settings require a high degree of suspicion by the echocardiographer.

Constrictive Pericarditis (Highlight Box 14.2)

Pericarditis is an inflammation of the pericardium that can be attributed to several etiologies, including immune-mediated, infectious, malignant, traumatic, uremic, and post-surgical. A normal pericardial thickness is 2–3 mm, and a thickened

Highlight Box 14.2

Constrictive pericarditis	
2D	• Thickened, highly reflective pericardium • Biatrial enlargement • Septal "bounce"
CFD	• Typically not utilized
Spectral	• Transtricuspid inflow velocities (PWD) • Transmitral inflow velocities (PWD) • Exaggeration of respirophasic variation • Restrictive diastolic profile (E much greater than A) • Normal tissue Doppler velocities

PWD pulsed-wave Doppler, *E* early diastolic mitral valve inflow velocity, *A* atrial contraction mitral valve inflow velocity

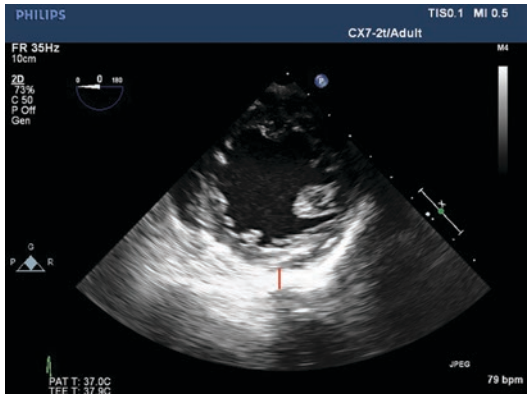

Fig. 14.16 Transgastric short-axis view. The *red line* indicates marked thickening of the pericardium

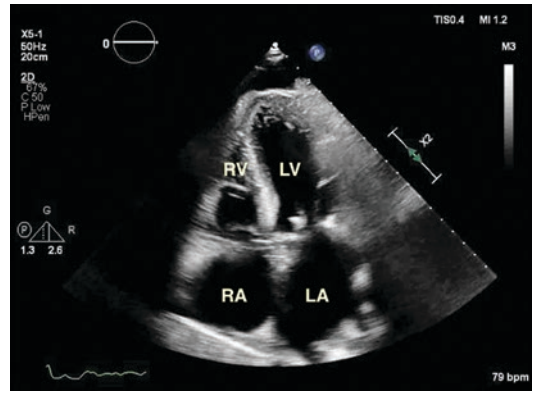

Fig. 14.17 Midesophageal four-chamber view. Biatrial enlargement in the absence of left or right ventricular enlargement can be seen in a patient with constrictive pericarditis. *RV* right ventricle, *LV* left ventricle, *RA* right atrium, *LA* left atrium

pericardium is defined as a pericardial thickness > 4 mm [3]. With echocardiography, a thickened pericardium becomes more echogenic, appearing brighter. Similar to the evaluation of pericardial effusion, multiple imaging windows should be utilized to investigate whether the thickening is localized or diffused (Fig. 14.16). Precise measurements of thickness by echocardiography are difficult, though measurements by TEE are superior to TTE [12] and comparable to those obtained by high-fidelity cardiac imaging [13].

Constrictive pericarditis (CP) is a clinical diagnosis that occurs when noncompliant pericardium leads to elevated and equalized cardiac pressures, similar to tamponade physiology. Although thickening of the pericardium may be frequently appreciated on echocardiography, it only progresses to hemodynamic compromise in 0.2–0.3% of cases [12]. As with the relationship between pericardial effusions and cardiac tamponade, a thickened pericardium may not always cause constrictive physiology, and CP may result from a pericardium with a normal thickness [14].

Several echocardiographic findings may be seen in patients with CP. As mentioned, the pericardium is often severely thickened, fibrotic, or calcified with or without pericardial adhesion (absence of detectable motion between the layers of the pericardium). Often the left and right atria are enlarged, with normal size and function of the left ventricle, in the absence of atrioventricular valvular disease (creating the "ice cream scoop on an ice cream cone" appearance) (Fig. 14.17).

Paradoxical septal motion is observed with CP, including a "septal bounce," in which early filling leads to an abrupt septal shift to the left during spontaneous inspiration followed by a second septal shift from filling by the atrial contraction. This gives the appearance of the ventricular septum having two "bouncing" movements to the left during a single cardiac cycle (Video 14.6) [15] . This septal motion is related to an exaggerated interventricular dependence, in combination with rapid early diastolic filling. Septal motion is best interrogated by TEE in the midesophageal four-chamber or transgastric mid-papillary short-axis views or by TTE with the corresponding views of apical four-chamber or parasternal short-axis views.

In addition to 2D echocardiography, spectral Doppler evaluation is useful to confirm the diagnosis of constrictive pericarditis. A restrictive filling pattern of the mitral inflow is confirmed by spectral Doppler analysis in which early diastolic filling (E-wave) is rapid and abruptly terminates as the diastolic pressure rises rapidly (see Chap. 12). The subsequent contribution from atrial contraction (A-wave) is minimal, such that the E-wave velocity exceeds the A-wave velocity by a ratio of 2:1 or greater [11] . In contrast to cardiac tamponade, in which there is primarily an

exaggeration of respirophasic variation during spontaneous ventilation, with CP, there is an exaggeration of respirophasic variation during both spontaneous ventilation and positive pressure ventilation (Figs. 14.18 and 14.19).

Differentiating Constrictive Pericarditis and Restrictive Cardiomyopathy

Restrictive cardiomyopathy (RCM) is often confused with CP, since both are characterized by hemodynamic compromise related to restrictive filling patterns. However, reduced compliance in CP is due to external constraint from the abnormal pericardium, while reduced compliance in RCM is due to abnormal elastic properties intrinsic to the myocardium or intercellular matrix. On echocardiography, both pathologies result in biatrial enlargement and severe diastolic dysfunction. Findings on echocardiography that favor CP include pericardial thickening, "septal bounce," and exaggeration of respirophasic variation. The two pathologies can be differentiated by tissue Doppler imaging, as CP possesses normal tissue velocities (particularly near the septum), while RCM with its abnormal myocardium possesses reduced tissue velocities throughout the heart.

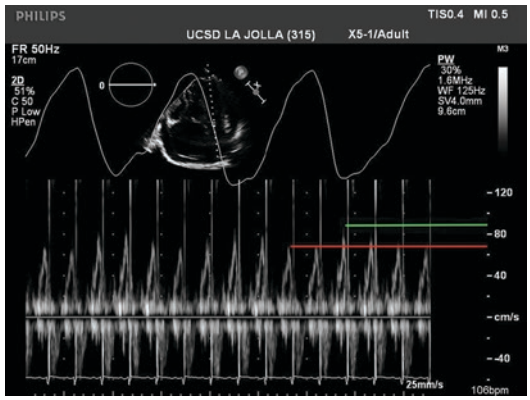

Fig. 14.18 Apical four-chamber view. Pulsed-wave Doppler of the transmitral inflow is performed during spontaneous ventilation, demonstrating the E-wave velocity decreasing by more than 25% from expiration (*green line*) to inspiration (*red line*)

Conclusion

Echocardiography is the preferred method for evaluation of the pericardium, owing to its test characteristics and ability to provide relevant physiologic data. Both transesophageal and transthoracic echocardiography can provide rapid, valuable information about the severity and sometimes the provenance, of pericardial disease, including pericardial effusion, cardiac tamponade, and constrictive pericarditis.

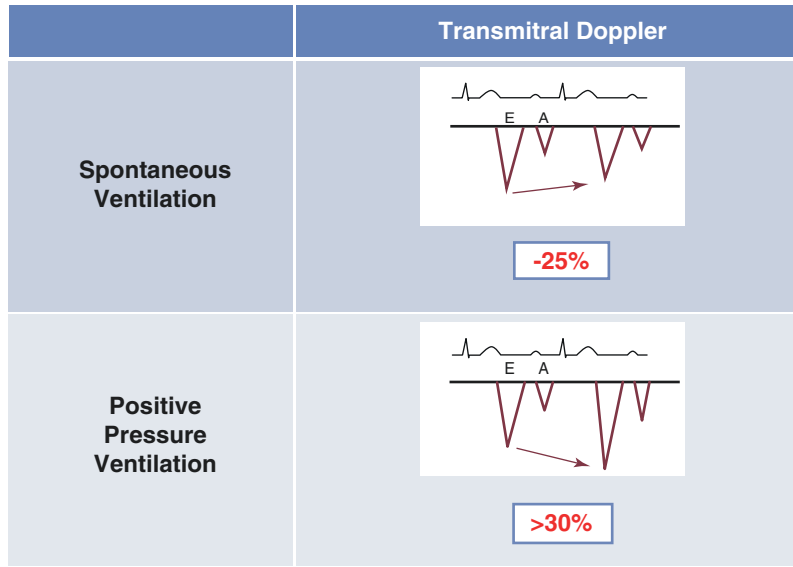

Fig. 14.19 In constrictive pericarditis, spectral Doppler patterns of the transmitral inflow demonstrate exaggerated respirophasic variation during inspiration for both spontaneous ventilation and positive pressure ventilation

Questions

1. Which of the following views does not demonstrate the transverse sinus?

   (a) Midesophageal two-chamber view
   (b) Midesophageal long-axis view
   (c) Midesophageal bicaval view
   (d) Midesophageal ascending aorta short-axis view

2. Which of the following is most true regarding differentiating epicardial fat from a pericardial effusion?

   (a) Epicardial fat is not associated with cardiac chamber collapse.
   (b) Pericardial fluid moves in concert with the myocardium.
   (c) Epicardial fat is more echogenic than myocardium.
   (d) Epicardial fat is adherent to the parietal pericardium.

3. In normal conditions, which of the following is most true?

   (a) Spontaneous inspiration results in increased filling of the left heart.
   (b) Positive pressure inspiration results in increased left ventricular stroke volume.
   (c) Spontaneous expiration results in increased pulmonary venous pooling.
   (d) Positive pressure expiration results in increased left ventricular afterload.

4. In normal conditions, which of the following is most true?

   (a) Spontaneous expiration results in decreased left ventricular afterload.
   (b) Positive pressure expiration results in increased right ventricular afterload.
   (c) Spontaneous inspiration results in increased left atrial filling.
   (d) Positive pressure inspiration results in increased right ventricular stroke volume.

5. Compared to normal respirophasic variation, what changes in atrioventricular inflow patterns do you see in constrictive pericarditis?

   (a) Exaggeration with spontaneous ventilation; exaggeration with positive pressure ventilation
   (b) Exaggeration with spontaneous ventilation; reduction with positive pressure ventilation
   (c) Reduction with spontaneous ventilation; exaggeration with positive pressure ventilation
   (d) Reduction with spontaneous ventilation; reduction with positive pressure ventilation

6. Which of the following is most true regarding cardiac tamponade?

   (a) The size of the effusion is proportional to the severity of hemodynamic effect.
   (b) Right atrial collapse indicates cardiac tamponade.
   (c) Left-sided chamber collapse is always preceded by right-sided chamber collapse.
   (d) The ventricles exhibit interdependence.

7. During cardiac tamponade, patients will exhibit which of these changes in peak E-wave velocity with spontaneous inspiration?

   (a) Increased transtricuspid; increased transmitral
   (b) Increased transtricuspid; decreased transmitral
   (c) Decreased transtricuspid; increased transmitral
   (d) Decreased transtricuspid; decreased transmitral

8. By semiquantitative assessment, a pericardial effusion with a transverse measurement of 15 mm is approximately how much volume?

   (a) < 100 mL
   (b) 100 to 250 mL

(c) 250 to 500 mL
(d) > 500 mL

9. Which of the following is not associated with constrictive pericarditis?

(a) Restrictive filling pattern
(b) Septal "bounce"
(c) Biatrial enlargement
(d) Decreased septal tissue velocity

10. Which of the following is more likely in the setting of constrictive pericarditis than tamponade?

(a) Septal "bounce"
(b) Exaggerated respirophasic variation during positive pressure ventilation
(c) Pulsus paradoxus
(d) All of the above are equally likely

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