Pericardial Diseases

20

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Abstract

The pericardium is a thin fibroelastic sac, comprised of both a serous visceral and fibrous parietal layer, which surrounds the heart and the great vessels. Echocardiography remains the initial imaging modality of choice and should be performed as a complete study or as a point of care when suspecting acute pericarditis. Presence of an abnormal septal motion from right to left in early diastole during inspiration, the presence of a plethoric IVC and dilated hepatic veins, and evidence of ventricular interdependence are the cornerstones of constrictive pericarditis physiology. Annulus paradoxus and annulus reversus are also strong indicators of constrictive pericarditis. The location, size, extent of the pericardial effusion and presence of loculations should be characterized using all four echocardiographic windows including PSLAX, PSAX, Apical, and the Subcostal views. Chamber collapse is an important indicator of the presence of tamponade physiology along with a septal bulge and peak mitral and tricuspid E-inflow velocity respiratory variation. Most cases of cardiac tamponade are treated with a pericardiocentesis.

Keywords

Acute pericarditis · Pericardial effusion · Effusive constrictive pericarditis · Constrictive pericarditis · Tamponade · Pericardiocentesis

Abbreviations

2D	Two dimensional
3D	Three dimensional
ASA	Aspirin
CMR	Cardiac magnetic resonance
CP	Constrictive pericarditis
CRP	C-reactive protein
EF	Ejection fraction
E	Peak E-wave velocity of the mitral valve (cm/sec)
e′	TDI of the mitral annulus
EKG	Electrocardiogram
IVC	Inferior vena cava
LA	Left atrium
LV	Left ventricular
LVEF	Left ventricular ejection fraction
NSAID	Non-steroidal anti-inflammatory drug
PSLAX	Parasternal long axis
PSAX	Parasternal short axis
RV	Right ventricle
STE	Speckle-tracking echocardiography
TDI	Tissue Doppler imaging
TEE	Transesophageal echocardiography
TTE	Transthoracic echocardiography

Introduction

- **The pericardium is a thin fibroelastic sac, comprised of a visceral and parietal layer, which surrounds the heart and the great vessels. The normal pericardial cavity contains less than 50 cc of serous fluid** [1–4]. Pericardial diseases are common in clinical practice and have been reported to have significant morbidity and mortality [5, 6]. Diseases such as acute pericarditis, pericardial tamponade and constrictive pericarditis can be isolated in nature or a manifestation of a systemic process [7–9]. Echocardiography allows for the evaluation of the pericardium by two-dimensional imaging and hemodynamics by doppler methods [7].
- **A healthy pericardium appears as a thin hyperechogenic structure with echocardiography. Its normal thickness is less than 3 mm** and only under pathological states will the visceral and parietal layers become apparent [10].

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Acute Pericarditis

- **Acute pericarditis is the most common form of pericardial disease worldwide and can be associated with significant morbidity** [11]. **In the western world, the most common form is idiopathic pericarditis**. In a small percentage of cases, specific etiologies can be identified such as infectious, autoimmune, neoplastic, and iatrogenic. (Table 20.1)
- **Patients present with pleuritic chest pain (85–90% of cases), a pericardial friction rub (less than 33%), typical changes on electrocardiogram (60% of cases) and a pericardial effusion (60%). The presence of two or more of these features allows for the diagnosis of acute pericarditis**.
- The presence of an elevated CRP can be useful for the diagnosis and follow-up of patients to document biochemical resolution. **In the setting of diagnostic uncertainty, delayed gadolinium enhancement of the pericardium on CMR has been shown to be of value** [8–15].

Table 20.1 Etiologies of pericarditis

Idiopathic	Most common cause
Infectious	Viral: Enteroviruses, Ebstein-Barr Virus, Coxsackieviruses, herpes viruses, adenoviruses
	Bacterial: *Mycobacterium Tuberculosis, Coxiella Burnetti, Borrelia Burgdoferi*
	Fungal: *Histoplasma* species, *Aspergillus, Blastomyces* and *Candida* species
	Parasitic: *Toxoplasma* species, *Echinococcus*
	Infective Endocarditis (extension)
Autoimmune and Auto-inflammatory	Connective Tissue Diseases: Systemic lupus erythematosus, Sjogrens syndrome, rheumatoid arthritis, scleroderma
	Vasculitis: eosinophilic granulomatosis with polyangiitis, Takayasu disease, Horton's disease, Behçet's syndrome
	Auto inflammatory diseases: Familial Mediterranean fever, tumor necrosis factor receptor-associated periodic syndrome
	Sarcoidosis
	Post myocardial infarction: Dressler's
Neoplastic	Primary cardiac tumors: pericardial mesothelioma
	Secondary Metastatic tumors: lung cancer, breast cancer and lymphoma
Metabolic	Uremia/Dialysis
	Myxedema/Hypo-hyperthyroidsim
	Anorexia nervosa
Traumatic	Direct or indirect thoracic injury
	Radiation injury
	Post myocardial injury
Iatrogenic	Post coronary percutaneous intervention
	Post pacemaker lead insertion
	Post radiofrequency ablation
	Post radiation
Drug related	Hypersensitivity pericarditis with eosinophilia
	Pericardial and myocardial injury secondary to antineoplastic drugs including doxorubicin, daunorubicin, fluorouracil, cyclophosphamide
	Drug induced lupus with pericarditis: procainamide, hydralazine, methyldopa, isoniazid, phenytoin

Echocardiography

- Echocardiography remains the initial imaging modality of choice and should be performed either as a complete study or as a point of care tool when suspecting acute pericarditis.
- Echocardiography can identify the presence, characterize and quantify a pericardial effusion as well as assess for any hemodynamic compromise [7, 10, 14].
- Increased pericardial thickening can sometimes be appreciated with or without an associated effusion [7].
- **Global left ventricular systolic function and regional wall motion abnormalities can be ascertained in the presence of concomitant myocarditis** [8, 14].

Treatment

- **High dose aspirin or non-steroidal anti-inflammatories are the mainstay of treatment for acute pericarditis** [8, 10, 16, 17].
- **Weight adjusted Colchicine is recommended to prevent recurrences** [18, 19].
- **Low dose corticosteroids (0.2–0.5 mg/kg/day) with a slow taper are indicated as a second line therapy when there are contraindications to or failure of NSAIDs/ASA.** They may be indicated as a first line therapy in cases of pregnancy related pericarditis or autoimmune etiologies [20].
- **Independent of the choice of therapy, initial dosing should be maintained until there is resolution of symptoms and normalization of the CRP.** Subsequently, the dose can be tapered according to the patients estimated recurrence risk.
- Patients should be counseled on restricting physical activity until there is clinical and biochemical resolution of the pericarditis as well as normalization of the EKG and echocardiogram [21, 22].

Prognosis

- Fever (≥38.0 °C), a subacute presentation, failure of initial therapy, a pericardial effusion of greater than 20 mm, or tamponade physiology are features of poor prognosis and their presence may warrant a hospitalization [23].

Expert opinion considers myocarditis, history of immunosuppression or trauma, and patients on anticoagulation to be also at risk and should be monitored closely. Otherwise, outpatient care with close follow-up is appropriate [9].

Constrictive Pericarditis

- Constrictive pericarditis is a condition caused by a thick non-compliant, fibrotic and/or calcified pericardium which limits the diastolic filling of the ventricles.
- While it can evolve from cases of idiopathic acute pericarditis, it has been shown to be associated with cardiac surgery, mediastinal radiation, connective tissues diseases and infections such as tuberculosis [7, 9, 10, 24, 25].

Pathophysiology

- The heart chambers become confined to a non-compliant space due to a thickened and fibrosed pericardium. The restrictive nature of the pericardium causes a rapid early diastolic filling followed by an abrupt stop. As this disease progresses, the systemic venous pressures continue to rise to systemic venous congestion [7, 9, 24].
- Moreover, the constricting pericardium insulates the heart from the respiratory intrathoracic pressure changes. Normally, with inspiration, the venae cava, the pulmonary veins as well as the cardiac chambers have a contiguous drop in pressure during inspiration. As such, the gradients are maintained and forward flow is only slightly affected. With constrictive physiology, the pulmonary veins have a drop in pressure while the left ventricle and left atrium remains the same. This results in a smaller pulmonary vein to left atrium gradient during inspiration resulting in a drop of left atrial filling and transmitral inflow [7, 24].
- Lastly, with a fixed combined volume of the right and left ventricles and reduced left sided filling during inspiration, the septum shifts towards the left ventricle. This phenomenon known as ventricular interdependence is a characteristic finding in constrictive pericarditis [8, 24, 26].

Echocardiography

- Echocardiography is recommended as the initial cardiac imaging modality for patients with suspected constrictive pericarditis. It allows the operator to identify both the structural and hemodynamic features of constrictive pericarditis [7, 9, 24].

2D Echocardiography Findings Include:

- **Presence of an abnormal septal motion from right to left in early diastole during inspiration signifying the presence of ventricular interdependence. (Sensitivity of 93%)** [7, 27] (Fig. 20.1).
- The presence of a plethoric IVC, defined as an IVC diameter greater than 2.1 cm and associated with less than 50% reduction in its diameter during inspiration along with the presence dilated hepatic veins signifying increased right atrial filling pressures [7] (Fig. 20.2).
- Normal systolic ventricular function is what is commonly seen however the presence of some wall motion abnormalities have been reported owing to pericardial adhesions and tethering of the wall rather than damaged cardiomyocytes [24, 28].
- The presence of a thickened pericardium is best appreciated on the PSLAX view at the posterior edge of the heart or in the PSAX view. However, other imaging modalities such as CT, are superior for the detection of pericardial thickening and calcification [7].

M-mode Echocardiography Findings Include:

- **The presence of a septal "shudder" or septal "bounce" as depicted by an abrupt posterior motion of the ventricular septum in early diastole with inspiration**. This phenomenon signifies an abrupt increase in early diastolic right ventricular pressure, which overtakes the left ventricular diastolic pressure during the cardiac cycle [9, 24].
- Occasionally, premature opening of the pulmonic valve can be observed if the pulmonary vascular resistance is normal [9].

Doppler Flow Velocity Findings Include:

- **Characteristic hepatic vein diastolic flow reversal during expiration. This is a specific echocardiographic finding associated with constrictive physiology** [7, 27] (Fig. 20.3).
- **A restrictive pattern of filling using pulse wave doppler of the mitral inflow. On inspiration, a high early E velocity with a short deceleration time and a reduced atrial wave is noted. This results in an E/A greater than 1 representing a rapid equilibration of the left atrial and ventricular pressures** [7, 27].
- **Due to the dissociated intrathoracic and intracardiac pressures, during the first beat of inspiration, the mitral inflow velocity usually falls by as much as 25–40% and the tricuspid inflow velocity increased by as much as 40–60%** [10, 24, 27, 29] (Fig. 20.4).

Tissue Doppler Findings Include:

- **In constrictive pericarditis, the early diastolic velocity of the medial mitral annulus e′ is prominent. As the degree of constriction advances, the e′ increases**

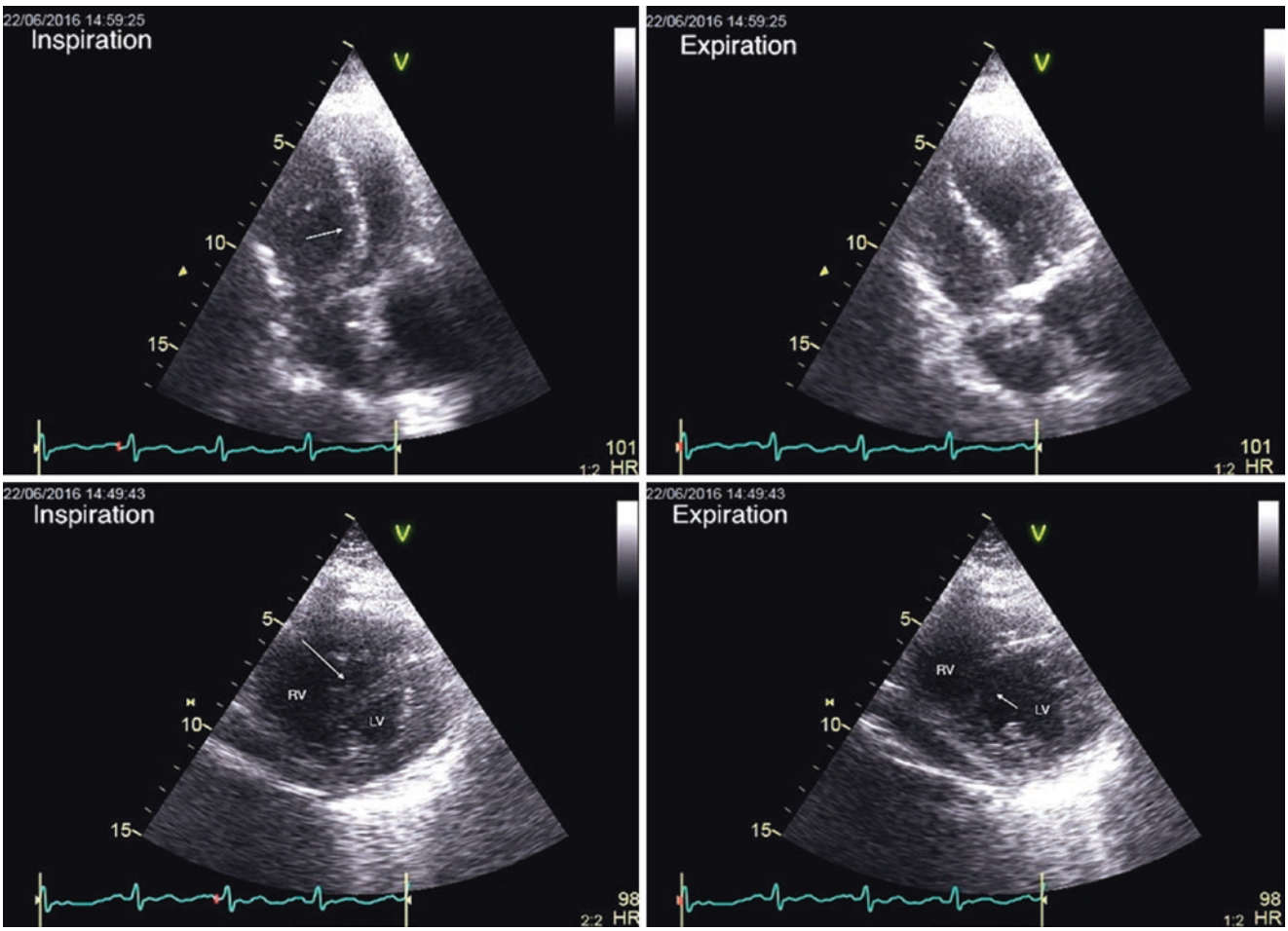


Fig. 20.1 Two-dimensional apical and parasternal short axis views of the heart of a patient with constrictive pericarditis. On inspiration (*left)*, the ventricular septum is seen moving into the left ventricular cavity (*white arrow*) and back towards the right ventricle on expiration (*right)*

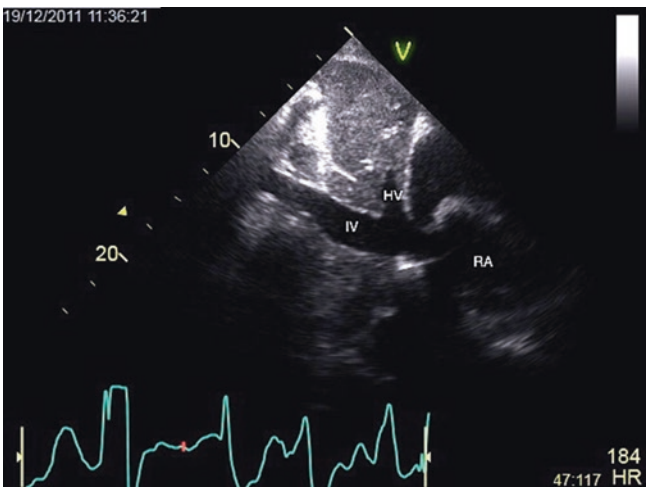


Fig. 20.2 Two-dimensional subcostal view of a patient in tamponade showing a plethoric inferior vena cava without respiratory collapse (*IV* Inferior vena cava, *HV* hepatic vein, *RA* right atrium)

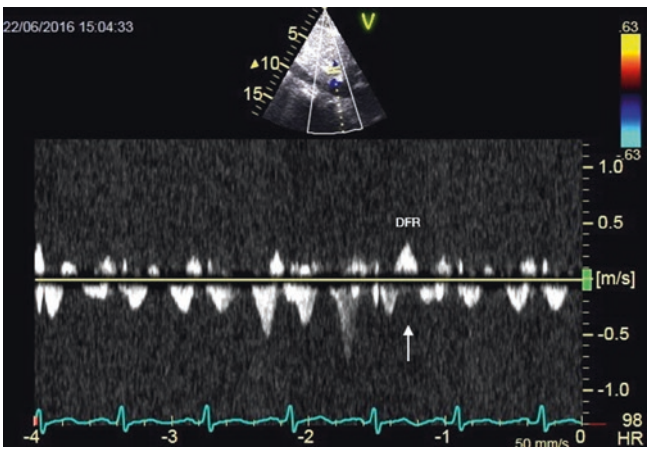


Fig. 20.3 PW Doppler echo of the hepatic vein demonstrating diastolic reversal velocity (*DFR)* during expiration

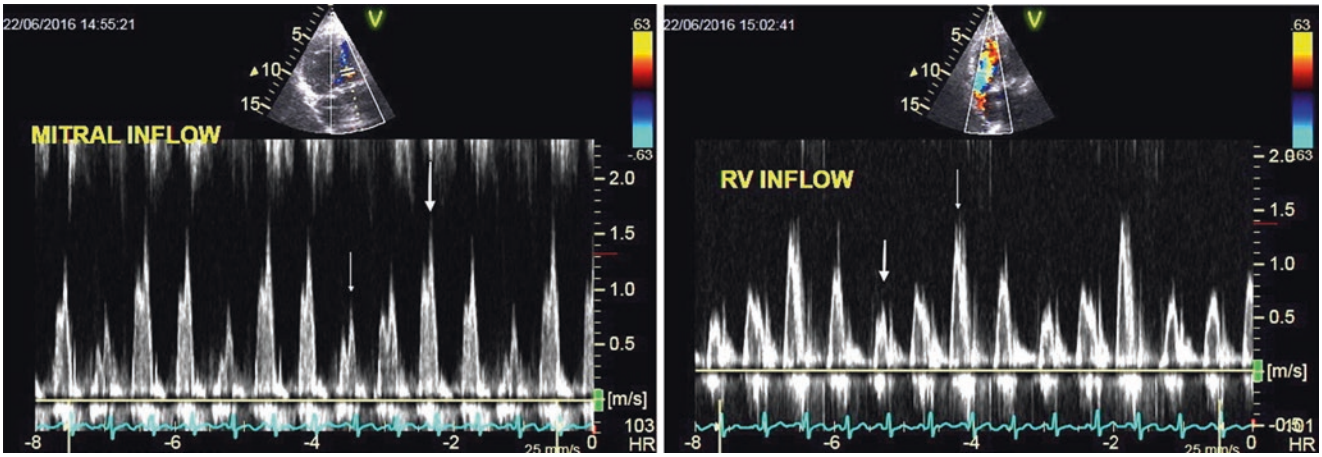


Fig. 20.4 Pulse wave Doppler echo of the mitral inflow velocity (*left)* and of tricuspid inflow (*right)* during normal respiration. On display is the characteristic respirophasic variation of the inflow velocities with inspiration (*thin arrow*) and expiration (*thick arrow*)

progressively rather than decreases, giving a lower than expected E/e′ ratio. This is in contrast to the expected decrease in e′ and E in individuals with worsening myocardial function and is therefore referred to as **annulus paradoxus** [27, 30–32].

- **In healthy individuals, the lateral e′ is generally 25–50% larger than the medial mitral annulus e′. In constrictive pericarditis, there is tethering of the lateral mitral annulus with an exaggerated compensatory medial longitudinal expansion and a resultant increase in the e′ of the mitral medial annulus; a phenomenon commonly referred to as annulus "reversus"** [27, 33–35].
- **Annulus paradoxus and annulus reversus can normalize after pericardiectomy** [28].

Strain Imaging

- **Global longitudinal strain is preserved in pure constriction.** Reduced global longitudinal strain is found if there is concomitant myocardial disease [24, 36, 37].

Transesophageal Echocardiography and 3D Echocardiography

- There is a limited role for transesophageal echocardiography as well as 3D echocardiography in the setting of constrictive pericarditis.
- One particular utility of TEE is the measurement and characterization of the pericardial thickness which has shown to have a good correlation with the computed tomography [38, 39].

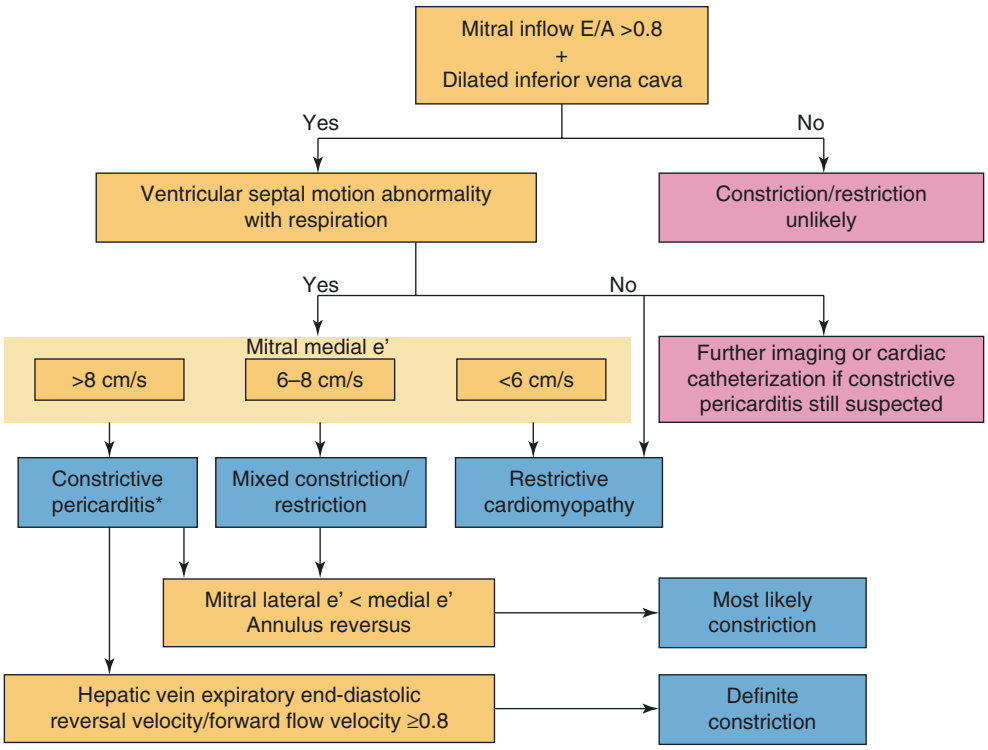
Practical Approach to Constriction [24, 27]: Table 20.2

- **The absence of a mitral inflow E/A ratio greater than 0.8 and dilated inferior vena cava renders the diagnosis of constrictive pericarditis unlikely**
- **If the former echo feature criteria are met, abnormal septal motion should be evaluated followed by the medial mitral annulus e′.**
- **Constrictive pericarditis is diagnosed with the presence of a respirophasic septal shift and a mitral annular e′ greater than 8 cm/s on the tissue Doppler.**
- **The diagnosis is then confirmed with the presence of hepatic vein expiratory end-diastolic reversal.**
- In the absence of an abnormal septal motion and a persistent suspicion of CP, further diagnostic modalities should be considered.

Treatment

- Approximately 10–20% of patients with constrictive physiology will respond to anti-inflammatories within a few months. This small subset of patients can be identified with elevated inflammatory markers like CRP and with other imaging modalities such as CT or MRI [13, 24, 40, 41].
- Pericardiectomy remains the treatment of choice for the majority of individuals with constrictive pericarditis including those who fail medical therapy [24].

Table 20.2 Reprinted by permission from Macmillan Publishers Ltd.: Nature Review: *Welch TD. Echocardiographic diagnosis of CP: Mayo clinic criteria Circ Cardiovasc Imaging* copyright (2014) Reprinted by permission from Dr. J Oh, corresponding author



Echocardiography diagnostic criteria algorithm for constrictive pericarditis. *In patients with obstructive airways disease or increased respiratory effort, ventricular interaction occurs and e' can be high especially in a young patient. In that situation, increased flow in the superior vena cava during inspiration is also seen and the hepatic vein also shows markedly increased flow with inspiration. Abbreviations: A, late mitral inflow velocity; E, early mitral inflow velocity; e', early mitral annulus diastolic velocity.

Other Considerations

Transient Constrictive Pericarditis

- Transient constrictive pericarditis is a form of constrictive pericarditis characterized by improvement or resolution of the clinical features of constriction either spontaneously or with anti-inflammatory medication.

Effusive-Constrictive

- Effusive-constrictive pericarditis is an uncommon form of pericardial disease and should be suspected in patients who present with pericardial tamponade and fail to improve after removal of the pericardial fluid. This disease occurs when there is pericardial fluid accumulation between a thickened, edematous or fibrotic parietal and visceral pericardium. After the pericardial fluid is removed, the cardiac chambers remain constricted within the thickened pericardium and demonstrate elevated RA, end diastolic RV and LV pressures as well as a dip and plateau ventricular waveform and respiratory interventricular interdependence. **Once again, this may respond to anti-inflammatory therapy if there is an elevated CRP or late gadolinium enhancement of the pericardium on MRI** [7, 42, 43].

Echocardiography

- An effusion containing intrapericardial echogenic material, some of which may be fibrous strands that traverse the pericardial cavity, sometimes accompanies effusive-constrictive pericarditis. Otherwise, findings ranging from a sizeable pericardial effusion to tamponade to constrictive pericarditis can be observed depending on the stage of the disease [7, 44].

Pericardial Effusion

- The pericardial sac contains between 10–50 cc of plasma ultra-filtrate that acts as a lubricant between the pericardial layers. Various conditions including acute pericarditis and hypothyroidism result in an accumulation of this

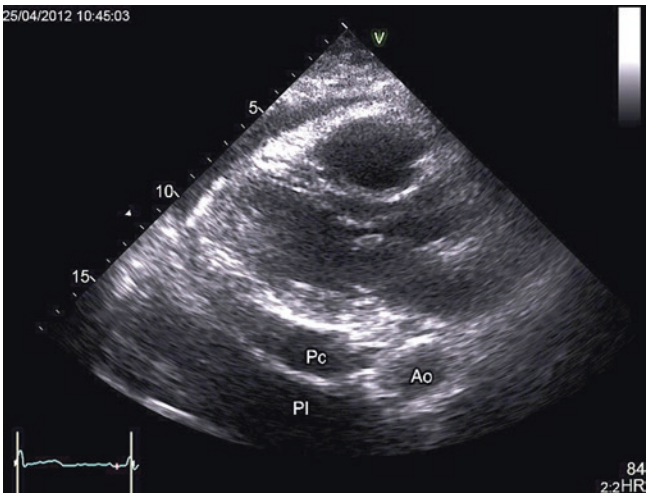


Fig. 20.5 Two-dimensional parasternal long axis view of the heart in a patient with a pleural and pericardial effusion. The echo-lucent fluid tracking the posterior wall of the myocardium remains anterior to the aorta (A) and is defined as the pericardial effusion (Pc). The echo-lucent fluid posterior to the aorta (A) is defined as the pleural effusion (Pl)

fluid. Moreover, depending on the underlying etiology, analysis of the pericardial fluid can reveal it to be a transudative effusion, an exudative effusion, hemopericardium, chylopericardium or pyopericardium [7, 9, 45].

- **Cardiac ultrasonography remains the primary imaging modality for the diagnosis of pericardial effusions.**

Echocardiography

2D Echocardiographic Findings Include:

- **A pericardial effusion can be differentiated by a pleural effusion in the PSLAX view with reference to the descending aorta. Fluid accumulation anterior to the descending aorta is pericardial while fluid accumulation posterior to the descending aorta is pleural in nature** (Fig. 20.5).
- **The location, size, extent of the pericardial effusion and presence of loculations should be characterized using all four echocardiographic windows including PSLAX, PSAX, Apical, and the Subcostal views** [7, 10]**.**
- **The standard measurement of the effusion is between the parietal and visceral pericardium at end-diastole** [7]**.**
- **Echocardiographic classification**
  - **Trivial effusion (seen only in systole)**
  - **Small effusion (less than 10 mm/300 mL)**
  - **Moderate effusion (10–20 mm/500 mL)**
  - **Large effusion (greater than 20 mm/more than 700 mL)**
  - **Very large effusion (greater than 25 mm)**
- All effusions should be evaluated for hemodynamic features of tamponade [7, 10, 46].
- **Epicardial fat, which is commonly mistaken for pericardial fluid, is brighter than myocardium under cardiac ultrasonography and also moves in concert with the heart in contrast to the echo-lucent and motionless pericardial fluid.**

3D Echocardiography

- The added role of 3D echocardiography after completion of a 2D echocardiographic exam in the assessment of pericardial effusion is limited and is therefore not recommended in routine assessment of effusions.
- The main limitation of 3D echocardiography is the full volume sector width which is not sufficiently large enough to capture the entire pericardial effusion and pericardium [47, 48].

Therapy

- **The initial management of a pericardial effusion remains the treatment of the underlying etiology. Objective evidence of pericardial inflammation should prompt a trial of NSAIDs with serial ultrasounds to follow the effusion.**
- **In the presence of a large effusion, pericardiocentesis may be considered to prevent eventual pericardial tamponade.**
- **A surgical pericardiotomy or a pericardial window should be considered when there is a history of recurrent large pericardial effusions.**
- **Certain causes of pericardial effusion should be treated as surgical emergencies such as aortic dissection or free wall rupture after a myocardial infarction. There is no role for pericardiocentesis in these circumstances** [9, 12, 49, 50]

Tamponade

- **Tamponade is a life threatening condition characterized by fluid accumulation in the pericardial sac which compresses the cardiac chambers and inhibits diastolic filling.**

Pathophysiology

- As the fluid in the pericardium collects and the pericardial reserve volume is surpassed, the pericardium begins to distend until it reaches the limits of the pericardial sac's

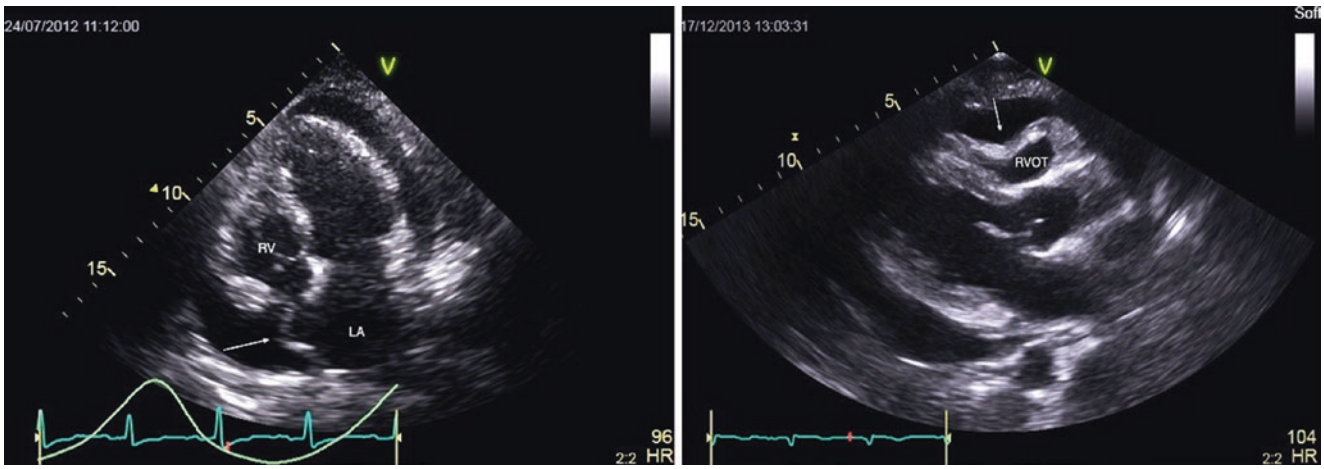


Fig. 20.6 *Left:* Two-dimensional apical view of patient in tamponade showing indentation of the right atrium in late diastole. *Right:* Two-dimensional parasternal long axis view of a patient in tamponade showing indentation of the right ventricular outflow tract in early diastole (*LA* left atrium, *RV* right ventricle, *RVOT* right ventricular outlflow tract)

ability to stretch. The limit of pericardial stretch is dependent on the amount of fluid and the rate of its accumulation. Any added volume thereafter exerts a pressure on the cardiac chambers. As tamponade continues to evolve, there is a rise in the intrapericardial pressures and eventual equalization with the diastolic pressures in the cardiac chambers. This results in reduced cardiac filling and reduced cardiac output.

- The accumulating pericardial fluid creates a fixed pericardial volume in which each side of the heart fills at the expense of the other side and an associated **exaggerated inspiratory decrease in systolic blood pressure known as pulsus paradoxus occurs** [51–53].

Echocardiography

- Individuals presenting with hypotension, tachycardia, a pulsus paradoxus greater than 10 mmHg along with evidence of increase central venous pressure should undergo cardiac echocardiography to determine if there is underlying tamponade physiology [9, 10, 54].
- **All views should be ascertained starting with the subxyphoid echocardiographic window to characterize the effusion as well as its hemodynamic effects**.

2D Echocardiography Findings Include:

- The presence of a pericardial effusion should be evaluated in its entirety to determine if it's circumferential or loculated.
- **Cavitary collapse is an important feature of tamponade physiology. It signifies that the intrapericardial pressures surpass the intracavitary pressures and filling of the chambers is impaired. Absence of any cavitary collapse has a greater than 90% negative predictive value for clinical cardiac tamponade**. M-mode echocardiography can often be utilized to determine the timing and duration of the collapsing chambers [9, 52].
- **Collapse of the right atrium is detected in late diastole (after atrial systole and near the peak of the R wave) and has a high sensitivity for tamponade** [55] (Fig. 20.6).
- **Diastolic compression of the RV free wall, observed in early diastole, is a more specific indicator**. Initially, it is only observed on inspiration, but as tamponade progresses, diastolic compression extends throughout the whole respiratory cycle. Moreover, the duration of indentation of the free wall which starts in early diastole correlates with the severity of tamponade (Fig. 20.6).
- In certain pre-existing conditions such as pulmonary hypertension, the high right ventricular pressures resist intracavitary collapse. In these particular cases, tamponade needs to be in its advanced stages before the intrapericaridal pressures are able to overcome the already elevated intracavitary pressures [52, 56, 57].
- **Left atrial collapse is a much rarer occurrence (25% of cases) but has a higher specificity for clinical cardiac tamponade.**

- **Ventricular interdependence is an important aspect of tamponade physiology and is demonstrable with a cardiac echocardiogram**.
- In healthy individuals, a compliant pericardial sac with minimal fluid, allows for normal left and right ventricular fillings without any restriction. In tamponade, the right ventricle can no longer expand outwards to fill in diastole, forcing the septal wall to shift into the left ventricular cavity leading to impaired filling of the left ventricle. This ventricular interaction is observed on imaging as an inspiratory bulge of the interventricular septum from right to left, and is best seen in the apical four chamber view, or the PSAX view.

- This however is not the case is certain pre-existing conditions with high left ventricular pressures such as left ventricular hypertrophy [58, 59].
- The presence of a plethoric IVC defined as an IVC diameter greater than 2.1 cm and associated with less than 50% reduction in its diameter during inspiration, as well as the presence dilated hepatic veins, signifies increased right atrial filling pressures [7, 60] (Fig. 20.2).

M-mode Echocardiography Findings Include:
- M-mode echocardiography can be utilized to determine the exact timing of the collapsing chambers
- Ventricular interdependence can also be demonstrated with M-mode echocardiography. When acquiring views in PSLAX, the RV diameter can be seen to increase with inspiration and decrease with expiration while the LV diameter can be seen to decrease in inspiration and increase with expiration.

Doppler Flow Velocities Findings Include:
- The inspiratory bulge during inspiration, signifying the presence of ventricular interdependence, causes a reduction in LV filling. Consequently, the transmitral doppler early diastolic E wave is reduced as is the LV outflow velocity. In the right heart, however, the opposite is observed. As the interventricular septum shifts into the left ventricle there is more right sided filling depicted by an increased tricuspid E wave and therefore an increased RV outflow velocity.
- Peak E wave mitral flow velocity, as measured by either Pulse or Continuous wave Doppler, is estimated to vary within 5% during the respiratory cycle of a healthy individual. **When tamponade physiology is present, the peak mitral E-inflow velocity has a maximal drop on the first beat of inspiration and exceeds 30% respiratory variation** [9, 61, 62].
- **The peak tricuspid E-inflow has a maximal drop on the first beat of expiration and exceeds 60% respiratory variation** [9, 61, 62].

- Normal hepatic flow is biphasic in nature with the systolic velocity greater than diastolic velocity; both of which are increased with inspiration. Moreover, it is normal to have reduced forward flow or even reversal of flow during the atrial contraction (atrial reversal) and/or during end-systole (venous reversal) [9].
- In the presence of tamponade physiology, hepatic forward flow velocities decrease (from 50 to 20–40 cm/s) and continues to do so with persistently rising pericardial pressures. **In the early stages of tamponade, systolic venous flow predominates, given that the heart is smallest in volume and the intrapericardial pressures are the lowest. In the presence of more advanced tamponade, diastolic flow becomes nearly absent but demonstrates some inspiratory variation. When the tamponade physiology has reached a severe state diastolic flow disappears completely.**
- **As the pericardial pressures continue rise, the systemic venous and intracardiac pressures equalize and only forward systolic flow is seen during inspiration from the hepatic veins to the RA. This usually indicates that cardiac arrest is imminent** [61, 63].
- Although the hepatic vein pulse wave doppler is a difficult variable to obtain, it has a high positive and negative predictive value (82% and 88% respectively) [61, 63].

Treatment
- The gold standard for the treatment of cardiac tamponade is pericardiocentesis [52].
- Medical therapy is of limited utility [64–66].
- Surgical intervention is usually required when there is failure of pericardiocentesis or in the setting of intrapericardial bleeding and clotted hemopericardium. A window, a connection between the pericardium and the absorbing surface of the pleura or peritoneum, may be necessary for malignant effusions that are recurrent [52, 67].

Pericardiocentesis

- Pericardiocentesis is often indicated in patients with tamponade and may be lifesaving if performed shortly after diagnosis.
- **Echo-guided pericardiocentesis is preferred over a blind subxiphoid approach and is associated with reduced complication rates.**
- **Initially, the echocardiogram should be utilized to determine the ideal entry point characterized by the most pericardial fluid in end-diastole with the least amount of interfering structures**.
- With or without direct ultrasound guidance, a standard 18 gauge thin walled needle or a smaller 21 G micropuncture needle is inserted into the pericardium and then exchanged with a 4F or 5F plastic catheter. **Positioning is then confirmed either with 6 mL of agitated saline or identification of the guidewire under ultrasound** (Fig. 20.7).
- Once the tip of the catheter is confirmed to be within the pericardial space, a 0.035″ guidewire followed by an 8.5F pigtail catheter is introduced using the Seldinger's technique. Progressive dilatation and/or a peel-away sheath may be necessary if the pigtail has a preformed curve.
- The pericardial fluid should be removed slowly and then left on negative suction. **The pigtail should remain in the pericardium until there is less than 30 mL/24 h for 24–48 h in order to decrease recurrence rates** [68, 69].

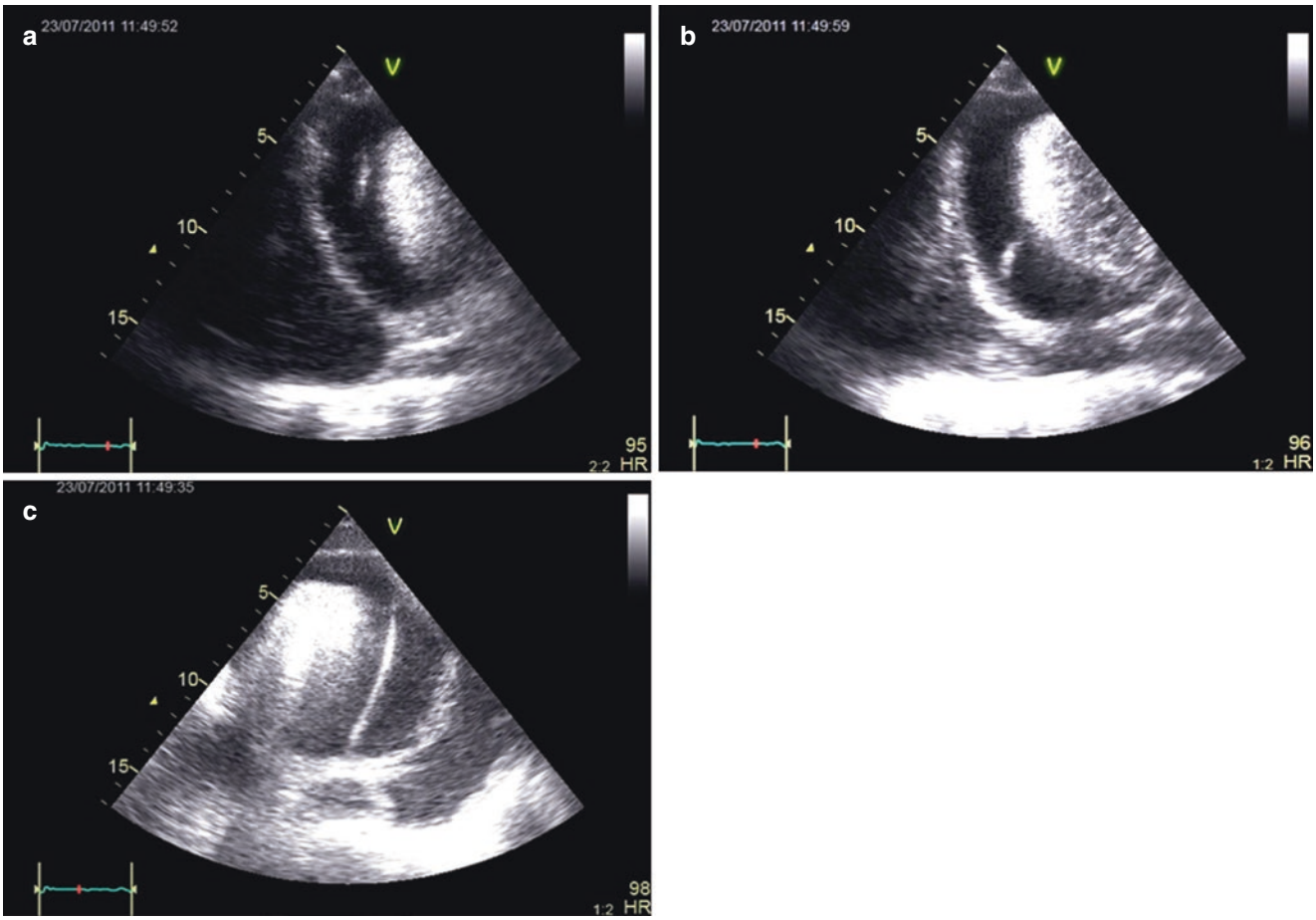


Fig. 20.7 Two-dimensional apical view of a patient in tamponade during a pericardiocentesis. The operator is confirming the presence of the guidewire in the pericardial space

Questions

1. The first line treatment of acute pericarditis includes:
   A. Non-steroidal anti-inflammatories and colchicine
   B. High-dose corticosteroids
   C. Low-dose corticosteroids
   D. Azathioprine

2. Ventricular interdependence is demonstrated echocardiographically by:
   A. Annulus paradoxus
   B. Annulus reversus
   C. A respirophasic ventricular septal shift in the apical four chamber window
   D. Abnormal mitral inlet inflow E/A ratio

3. After a pericardiocentesis, if the cardiac parameters reveal an elevated RA, elevated end diastolic RV and LV pressures as well as a dip and plateau ventricular waveform this is most likely
   A. Incomplete pericardiocentesis
   B. Effusive-constrictive pericarditis
   C. Transient effusive pericarditis
   D. The expected physiological response to pericardiocentesis.

4. Acute aortic dissection with a resultant pericardial effusion should prompt:
   A. High dose anti-inflammatories
   B. Urgent Pericardiocentesis
   C. Urgent cardiac surgery
   D. IV Tranaxemic acid

5. In Tamponade,
   A. Collapse of the right atrium in early diastole is highly sensitive
   B. Diastolic compression of the RV free wall is specific for tamponade physiology
   C. Left atrial collapse is the first sign of tamponade
   D. The definitive treatment of tamponade is fluid resuscitation.

Answers

1. A
2. C
3. B
4. C
5. B

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