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*Review*

# Mastering Pericardial Pathology: Effusion or Tamponade?

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## *Abstract*

**Cardiac tamponade is characterized by increased pressure in the pericardial space and can significantly impair circulation. Differentiation between pericardial effusion and the critical condition of tamponade requires an understanding of the underlying physiology, as simple pericardial effusion—the accumulation of excess fluid—does not always lead to the development of tamponade. Hemodynamic and echocardiographic evaluations can guide clinical decision making to ensure proper management of these patients. With clear comprehension of these distinctions, clinicians can quickly identify and address cardiac tamponade, ensuring optimal patient outcomes.**

***Keywords:*** **Pericardial disease, echocardiography, pericardial effusion, pericardial tamponade, ventricular interaction.**

## Introduction

Cardiac tamponade is a medical emergency that can be effectively treated if diagnosed correctly. To clinically diagnose cardiac tamponade, it is essential to understand the underlying characteristics of both pericardial effusion and tamponade. While pericardial effusion is a key feature of cardiac tamponade, not every simple pericardial effusion results in tamponade. It's important to view pericardial effusion and tamponade as part of a continuum, rather than as separate conditions.

This manuscript provides a detailed review of the pathophysiology of pericardial effusion and cardiac tamponade. It includes comprehensive summaries of the clinical findings and echocardiographic features associated with both conditions, as well as management strategies.

## The pericardium

### *Anatomy and function of the pericardium*

The term "pericardium" is derived from the Greek word perikardion, which means "around the heart"[1]. The pericardium is a membranous sac which envelops almost the entire heart with the exception of the region of the left atrium around the pulmonary veins. This sac consists of an outer fibrous layer (denoted the fibrous pericardium) and an inner double-serous membrane layer which directly covers the heart (the serous pericardium, also called the epicardium). The serous pericardium is further divided into an outer and inner layer (parietal and visceral layers, respectively) separated by a space containing a small amount of clear serous pericardial fluid (Fig. 1). The parietal layer fuses with the fibrous pericardium to create an inseparable outer layer. Overall, the pericardial is thin, approximately 1–2 mm thick[2,3], and increased thickness and calcification are characteristic of constrictive pericarditis.

A normal pericardium does not impede the transmission of intrathoracic pressure (ITP) changes during physiologic respiration. Under normal conditions, the pericardial pressure is close to zero, oscillating between 5 and +5 mmHg depending on the change in ITP. Under pathological conditions, pericardial pressure may rise because of either increased volume of pericardial fluid or because of pronounced pericardial stiffness. The pericardial pressure-volume relationship is nonlinear; initially, the slope is flat, but subsequently becomes very steep[4]. This nonlinear relationship explains why increases in the size of pericardial effusion may

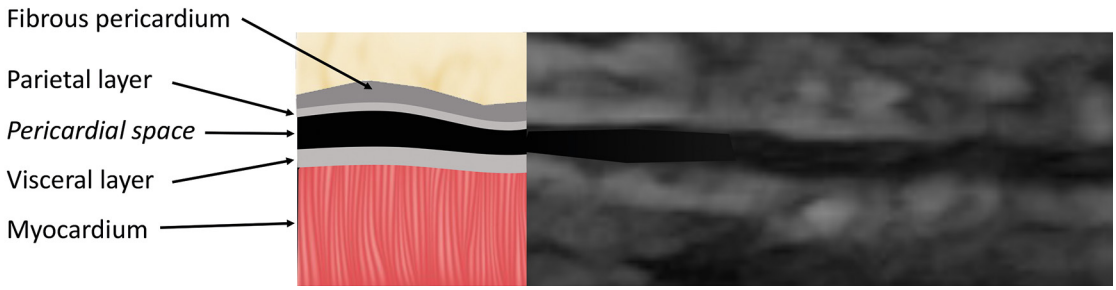


*Fig. 1* — The pericardium has an outer fibrous layer and an inner double-serous membrane, consisting of the parietal and visceral layers.

cause only modestly elevated pericardial pressures when the slope is flat. However, when the steep portion of the curve is reached, relating to the limit of the pericardial volume is reached, even small increases in pericardial fluid cause marked increases in PCP (Fig. 2).

The pericardium is not essential for life[3], and cases of congenital absence do occur rarely, with an incidence of about 1/10.000[5]. The majority of these cases are clinically silent and are, therefore, typically diagnosed incidently[5]. About one third of patients with congenital absence of the pericardium have comorbidities such as atrial septum defects, patent ductus arteriosus, bicuspid valve, or tetralogy of Fallot[6]. As the heart is "unrestrained," excessive motion of the heart may be observed in all views. Dilatation of the right ventricle (RV) can be present, as well as an elongated atria with widened ventricles (teardrop appearance), resulting in an abnormal atrial-ventricular angle, and tricuspid regurgitation due to annular dilatation[5,7,8] (Fig. 3).

The pericardium serves important mechanical functions (e.g., constraint of ventricular filling, ventricular interaction). The interaction of the pericardium becomes critical in disease states characterized by rapid increases in heart size, such as acute RV failure. Furthermore, the pericardium protects the heart, reduces friction, and releases vasoactive substances which regulate heart function and coronary arteries[3].

### *Normal physiological changes in hemodynamics during the respiratory cycle*

In order to understand the hemodynamic changes that occur when cardiac tamponade develops, it is important to recall the normal physiologic changes that occur during the respiratory cycle.

Venous return is driven primarily by low central venous pressure or right atrial pressure (RAP). Adequate RV function maintains a low RAP while providing adequate forward flow through the lungs toward the left heart[9,10]. However, changes in RAP due to changes in ITP influence venous return. During spontaneous inspiration, negative ITP and increased abdominal pressure owing to descent of the diaphragm enhance venous return[11]. While this increases RV volume, changes in LV volume are

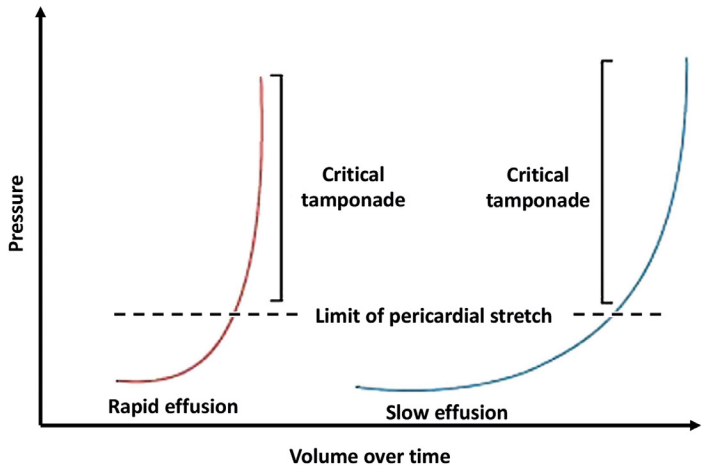


*Fig. 2* — Representative pericardial pressure-volume curves. The left-hand panel demonstrates an acute rapid increase in pericardial volume, which quickly reaches the limit of the pericardial reserve volume and exceeds the limit of pericardial stretch, causing a steep rise in pressure. The right-hand panel shows a slower—often chronic—rate of pericardial filling, which takes longer to reach the limit of pericardial stretch.
Figure used with permission[15].

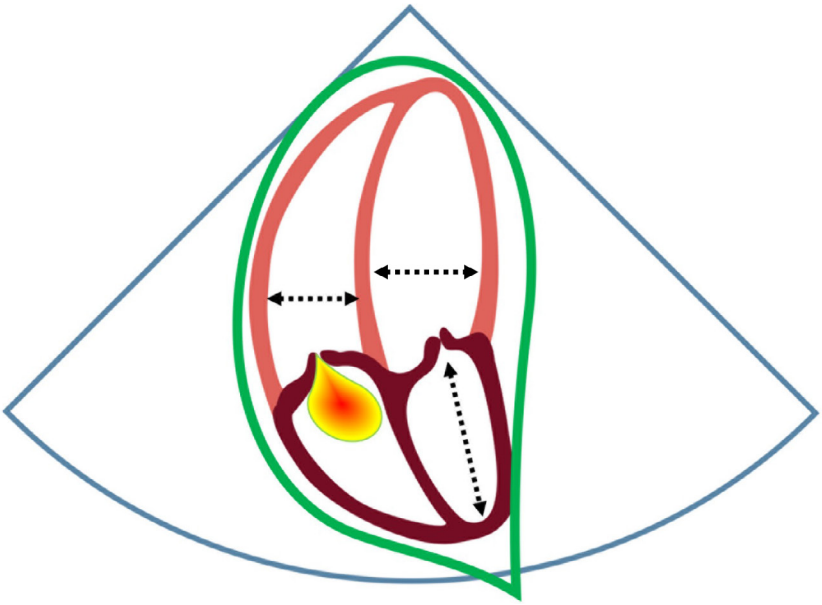

*Fig. 3* — Illustration of the echocardiographic appearance of congenital absence of the pericardium. Elongated atria with widened ventricles (teardrop appearance (teardrop-shape in green) can be seen, resulting in an abnormal atrial-ventricular angle and tricuspid regurgitation due to annular dilatation.

limited because the flow between the pulmonary veins and left atrium does not increase as both are subject to the same negative ITP. Along with these effects, lung expansion causes pulmonary blood volume and transit time to increase, and thus the increase in RV filling and output are transmitted to the left ventricle (LV) later in the respiratory cycle during expiration. Enlargement of the RV during inspiration expands the RV toward the septum and somewhat toward the pericardium (Fig. 4). Furthermore, negative ITP increases LV afterload by increasing transmural LV ejection

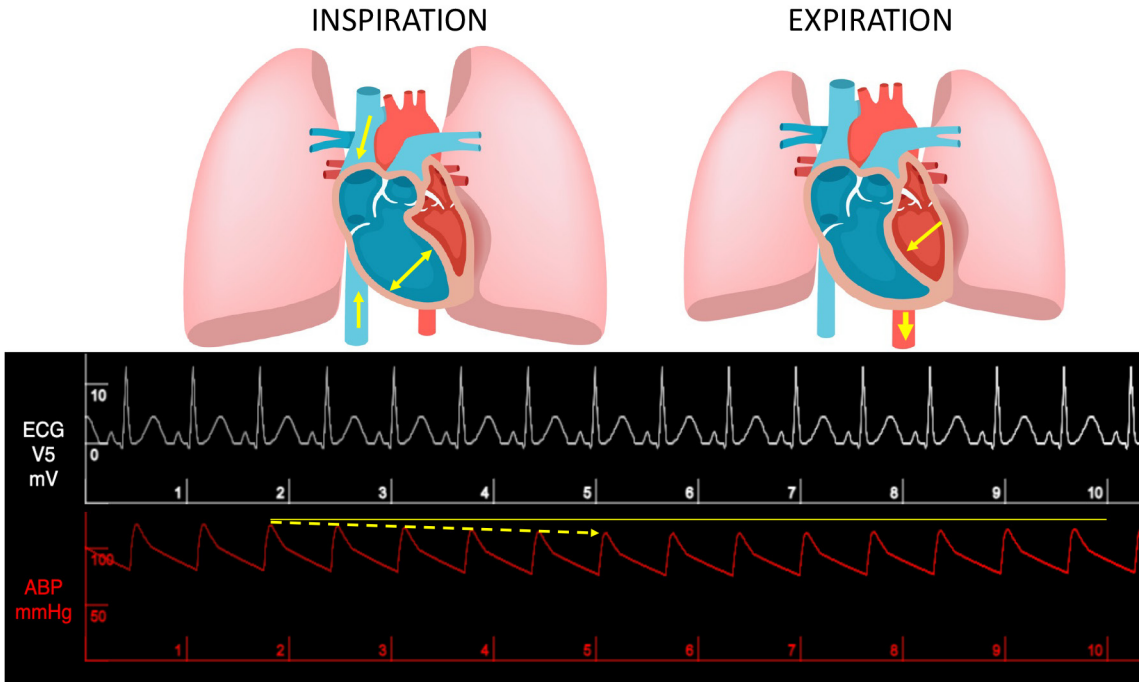


*Fig. 4* — Physiologic respiratory variation in cardiac volumes. Negative ITP is produced during inspiration, causing the systemic venous return to increase and, subsequently, increase RV filling. This pushes the septum toward the LV and the RV toward the pericardium (limited). The transit time of the RV stroke volume increases as the lungs expand during inspiration, leading to a reduction in the left-sided stroke volume and systolic blood pressure. Note that the dashed arrow shows this mild inspiratory decrease in arterial pressure that occurs during normal spontaneous breathing. Conversely, venous return decreases during expiration, leading to reduced RV filling and allowing the LV to fill more. This results in increased LV stroke volume and systolic blood pressure. Images were generated using Harvi cardiovascular simulation software[40].
Abbreviations: ABP Arterial Blood Pressure, ITP Intrathoracic pressure; LV Left Ventricle; RV Right Ventricle.

pressure[12]. During spontaneous breathing in healthy adults, these negative swings in ITP have a negligible effect on LV systolic performance because the normal LV can sustain ejection in the case of small increases in afterload[12]. As a result, small changes in LV stroke volume cause minor changes in systolic blood pressure (5-10 mmHg) during quiet spontaneous breathing[13,14].

## Pericardial effusion

Accumulation of fluid within the pericardial space can lead to increased PCP and concomitant clinical symptoms. Patients with pericardial effusion present with diverse clinical features depending on the volume and accumulation rate of effusion, the nature and cause of effusion, the thickness and compliance of the pericardium, and the presence of coexisting heart disease[15,16].

The causes of pericardial effusion are diverse, and understanding these various origins is essential for effective treatment (Table I).

Pericardial effusion is detected on echocardiographic imaging as an echolucent space between the heart and parietal pericardium. Fluid accumulation of >25 ml will result in an anechoic pericardial space that is readily detectable by echocardiography. An effusion is considered small when the separation between parietal and visceral pericardium measures <0.5 cm, moderate at 0.5–2 cm, and large when it is >2 cm[4].

Note that the echolucency of an effusion may be confused with pericardial fat, which includes both epicardial and pericardial adipose tissues. However, these are actually different fat deposits, and differentiation between fat and fluid can be challenging on echocardiography[17]. Fat is usually located more anteriorly than posteriorly and appears slightly more echogenic or granular compared with fluid. Furthermore, fat moves in concert with the heart and does not cause chamber collapse as occurs with cardiac tamponade (Fig. 5).

**Table I.** — Etiology of pericardial effusion.

| |
|---|
| **P**osttraumatic or postoperative after cardiac surgery |
| **E**causa ignota or idiopathic |
| **R**adiation therapy |
| **I**nfectious (viral, bacterial, fungal and parasitic) |
| **C**ancer (primary tumor e.g. rhadbdomyosarcoma; metastatic e.g. lung and breast cancer; paraneoplastic) |
| **A**cute myocardial infaction (early infarction pericarditis) |
| **R**ight heart failure |
| **D**issecting aortic aneurysm |
| **I**mmune mediated (systemic inflammatory disease e.g.lupus) |
| **U**remia (renal failure) |
| **M**edication (e.g. procainamide, dantrolene, penicillin, anticoagulants…) |

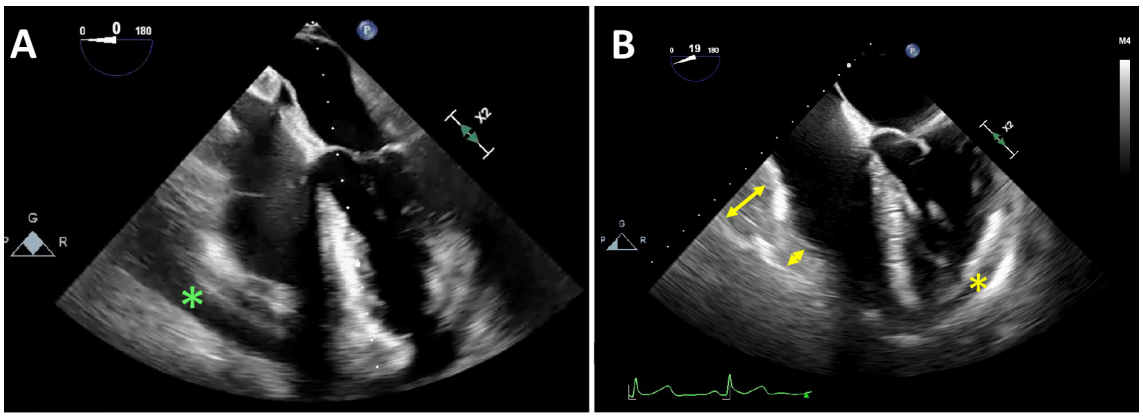


*Fig. 5* — Representative echocardiographic image (TEE ME Four Chamber view) of the presence of: (A) pericardial fluid (green asterisk) and (B) pericardial fat. Pericardial fat is more pronounced (yellow arrows) on the right side of the heart than on the left side (yellow asterisk).
Abbreviations: TEE Transesophageal Echocardiography; ME Midesophageal.

## Cardiac Tamponade

### *Pathophysiology and hemodynamic signs*

A pericardial effusion and tamponade should be understood as part of a continuum or progression of the same underlying condition, rather than as distinct pathologies. Cardiac tamponade is defined as a pericardial effusion that compresses one or more chambers, leading to hemodynamic compromise. In tamponade, the pericardial effusion distends the pericardium to the limits of its ability to stretch and results in a rapid increase in PCP (Fig. 2). In general, PCP exceeding 10-12 mmHg, is classified as moderate tamponade and causes compression of one or more cardiac chambers and impairs diastolic cardiac filling[18–21]. The atria, which are low-pressure chambers, are affected before the ventricles, which are higher-pressure chambers. Similarly, the right sided cardiac chambers will be impaired before the left sided chambers. Finally, the compressive effect of the pericardial fluid is most pronounced in the phase of the cardiac cycle when the pressure is lowest for each chamber: systole for the atria and diastole for the ventricles.

To best understand the hemodynamic changes that occur with cardiac tamponade, it is important to consider transmural pressure, the pressure of physiologic interest that represents the difference between cardiac chamber and PCP and determines ventricular preload or end-diastolic volume. Minor reductions in transmural pressure due to small increases in PCP may limit RV filling and reduce stroke volume. Further accumulation of pericardial fluid will increase PCP until the limits of "stretch" are reached (Fig. 2) and the steep portion of the pericardial pressure-volume relationship begins. At this point, any additional increase of pericardial fluid will cause a marked increase in PCP and lead to cardiac tamponade. Thus, vague symptoms like fatigue or dyspnea on exertion can indicate early hemodynamic compromise and herald early stages of tamponade[15,16].

In cardiac tamponade, spontaneous inspiration augments venous return to the right heart and causes the RV to preferentially expand toward the interventricular septum rather than the pericardium, which is limited by the increased PCP. Importantly, both parallel and series effects contribute to the inspiratory decrease in LV filling and stroke volume during inspiration. These pathophysiologic events explain the phenomenon termed "pulsus paradoxus," which is defined as a reduction in systolic pressure of >10 mmHg during spontaneous inspiration (Fig. 6). (Compare with the normal physiologic changes during the respiratory cycle, and Fig. 4). Pulsus paradoxus is one of the signature signs of cardiac tamponade, present in 98% patients[23]. While other conditions may cause pulsus paradoxus (massive acute pulmonary embolism, acute exacerbation of asthma, chronic obstructive pulmonary disease, tension pneumothorax, and large compressive pleural effusions)[23], these conditions are readily distinguished from tamponade by the presence of pericardial effusion in the latter.

Note that pulsus paradoxus is readily identified in a spontaneously breathing patient by observation of the arterial blood pressure waveform. In patients without invasive monitoring, a standard blood pressure cuff and stethoscope can be used. While slowly deflating the cuff and listening for Korotkoff sounds, note the systolic pressure at which the first sound is heard only during expiration and then note when sounds are heard throughout the respiratory cycle. The difference between these two systolic


*Fig. 6* — Simulation of pulsus paradoxus in a spontaneously breathing patient. Pulsus paradoxus is determined to be present when the decrease in systolic arterial pressure is >10 mmHg (yellow double arrow) during inspiration. Note: arterial pulse pressure also decreases during inspiration, which reflects the accompanying decrease in LV stroke volume (compare blue arrows). Images were generated using Harvi cardiovascular simulation software[40].
Abbreviation: LV Left Ventricle.

pressure values (just like examining the arterial pressure waveform) indicates pulsus paradoxus when there is a difference > 10 mmHg.

Typical fluid-based circumferential tamponade causes the progressive increase in PCP to increase filling pressures faster on the right than the left side. Accordingly, RAP will increase more quickly and earlier compared with the left-sided filling pressure. As the PCP increases further, it compresses all cardiac chambers during diastole until the pressures within the chambers equal the PCP[24]. Equalization of diastolic pressures across the cardiac chambers is another cardinal sign of cardiac tamponade and underscores the impairment of diastolic ventricular filling. Another notable hemodynamic feature of tamponade is the absence of a y-descent in the RAP waveform, since in this diastolic phase of the cardiac cycle, atrial volume is transferred to the ventricles leaving cumulative cardiac chamber volume relatively unchanged[24,25]. The RAP waveform maintains a steep systolic x-descent, however, since ventricular ejection reduces this "overall" volume and permits monophasic atrial emptying in the ventricles during systole. As a result, blood can only enter the atria when it simultaneously exits the ventricles during the x-descent (Fig. 7). Because early diastolic ventricular filling is so impaired in cardiac tamponade, loss of sinus rhythm and end-diastolic atrial contraction can further impair ventricular filling, cardiac output, and systemic blood pressure.

Sinus tachycardia is another hemodynamic sign of cardiac tamponade and results from physiological compensation for low stroke volume. In addition, dyspnea is common as a consequence of low cardiac output (ventilation-perfusion mismatch, stimulation by humoral factors such as acidosis and lactate, etc.).

In summary, the most common clinical signs of cardiac tamponade are an elevated RAP with a monophasic x-descent in the waveform, sinus tachycardia, dyspnea, hypotension, reduced pulse pressure and pulsus paradoxus (Fig. 8).

### *Atypical tamponade*

Loculated pericardial collections that cause "regional" tamponade are typically seen in the early postoperative stages following cardiac surgery and usually involve the collection of blood leading to compression of an individual chamber, most commonly the atria. Loculated effusions that compress the left-sided chambers in the absence of right-heart abnormalities are frequent and can lead to severe hemodynamic compromise[26], particularly in patients with a left ventricular assist device (LVAD) where typical clinical, hemodynamic, and echocardiographic signs of tamponade may not be present. Physiologically, placement of LVAD results in lower pressures on the left side of the heart, with resulting accumulation of pericardial fluid or blood in the left-sided chambers, while the right heart remains unaffected[27]. Tamponade should be suspected in patients with LVAD whenever pump flows are decreased and filling pressures are increased, especially if there is no response to a fluid challenge[28].

### *Diagnostic tests for cardiac tamponade*

#### *ECG*

- Sinus tachycardia, low QRS voltage and electrical alternans are typical ECG findings in tamponade. Sinus tachycardia is the most common finding and reflects a compensatory response to maintain cardiac output as stroke volume decreases.
- The amplitudes of the QRS complexes can be low as the fluid collection between the heart and the recording electrodes generate a damping effect. Low amplitude QRS voltage

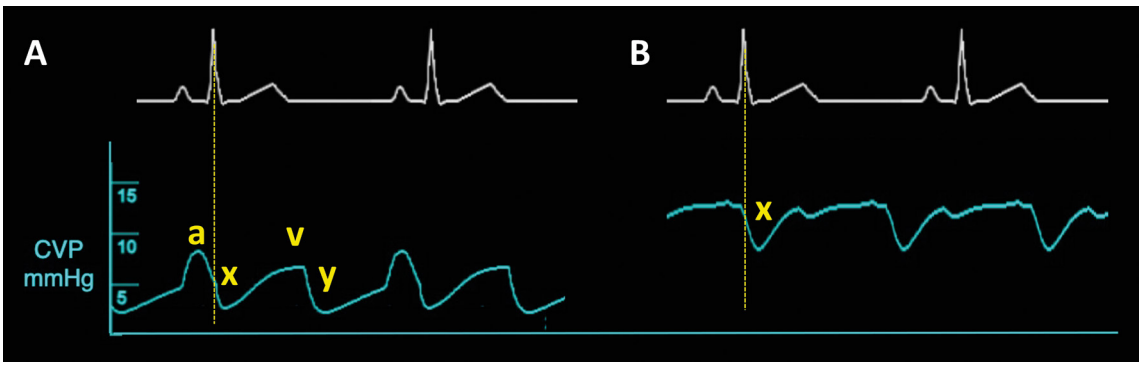


*Fig. 7* — Differences in RAP tracing between normal and tamponade hemodynamics.
(A) Normal RAP waveform showing low RAP, along with normal a and v waves and x- and y-descents. (B) RAP waveform in tamponade: RAP is increased and the y-descent is not present as diastolic flow to the RV is absent in tamponade. Images were generated using Harvi cardiovascular simulation software[40].
Abbreviations: RAP Right Atrial Pressure; RV Right Ventricle.

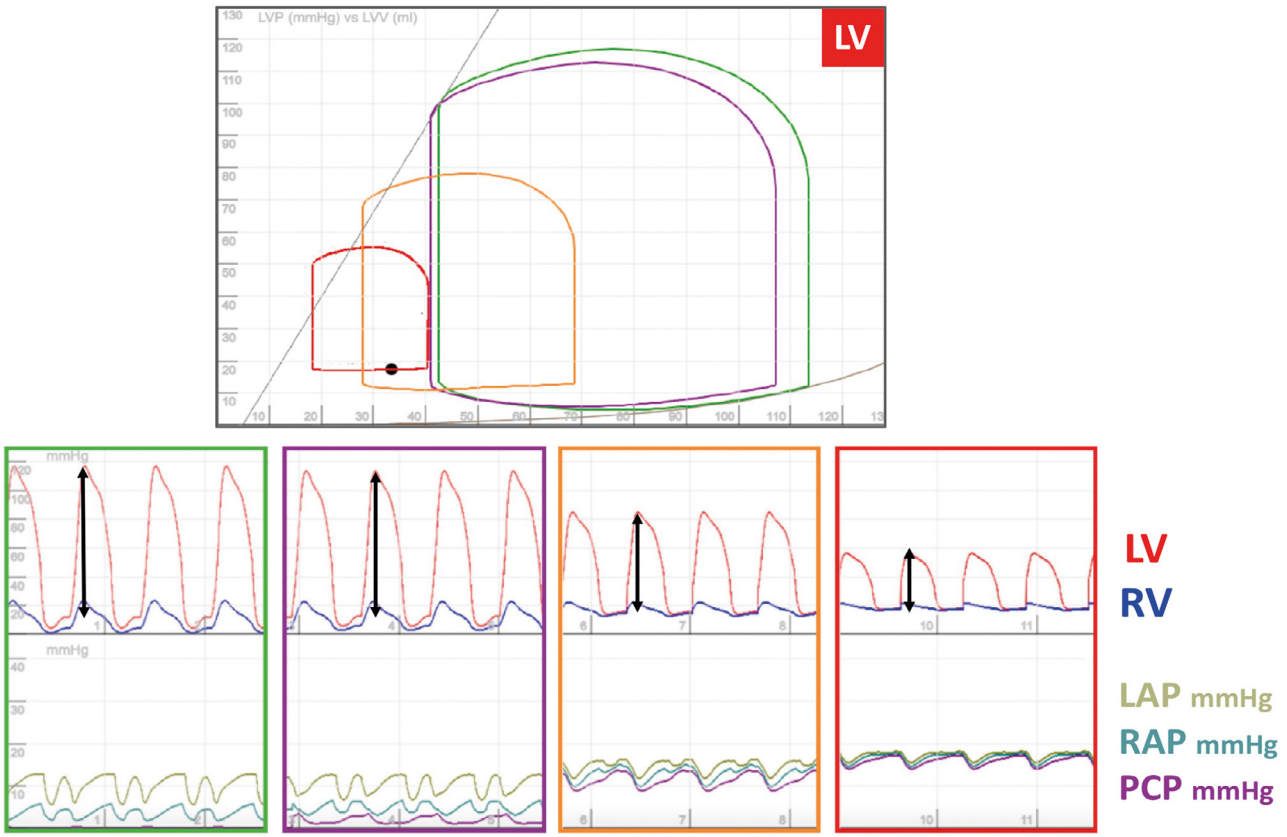


*Fig. 8* — Sequential hemodynamic changes during the development of cardiac tamponade. The top box shows the LV pressure-volume loop during the progression to cardiac tamponade. The four boxes below show these phases.
A: normal hemodynamics with normal heart rate, blood pressure and pulse pressure (double black arrow) and a low RAP.
B: a limited accumulation of pericardial fluid raises the PCP and slightly increases the RAP.
C: moderate tamponade. A significant rise in PCP leads to an increase in RAP (blue arrow), while LAP remains within normal limits. The RAP tracing begins to display a monophasic x-waveform. The pulse pressure is reduced (double black arrow).
D: Severe tamponade. PCP is high, causing an equalization of filling pressures. The RAP is high. The reduction in diastolic filling leads to a decreased CO, resulting in hypotension, a decreased pulse pressure (double black arrow) and the presence of pulsus paradoxus. A significant reduction in systolic blood pressure is highlighted by the red arrow. Images were generated using Harvi cardiovascular simulation software[40].
Abbreviations: RAP Right Atrial Pressure, LAP Left Atrial Pressure, PCP Pericardial Pressure, CO Cardiac output, RV Right Ventricle, LV Left Ventricle.

can also be present in hearts with considerable loss of viable myocardial tissue[15].

- Electrical alternans is a phenomenon characterized by the alternating height of QRS complexes. This occurs owing to the buildup of fluid in the pericardium that leads to a pendulous movement of the heart[15,29]. Although electrical alternans may be seen in other conditions, including arrhythmias such as atrioventricular nodal reentrant tachycardia[30], it is primarily seen in the presence of large pericardial effusions, with or without tamponade (Fig. 9).

*Chest X-ray*

The enlargement of the heart's silhouette on a posterior-anterior chest X-ray resembles the shape of a water bottle, referred to as the "water-bottle sign"[29]. This appearance typically occurs due to the stretching of the pericardium, which happens when a large volume of fluid accumulates in the pericardial space (Fig. 10).

*Echocardiography in cardiac tamponade*

Echocardiographic findings that support the hemodynamic signs of tamponade include the presence of a pericardial effusion together with the echocardiographic features discussed below.

- Right atrium collapse

As PCP rises, it will first exceed RAP in late diastole or early systole - when RAP is lowest - at the onset of atrial relaxation near the peak of the ECG R wave. RA collapse often occurs early in the course of tamponade physiology, commonly preceding clinical signs such as hypotension or pulsus paradoxus. Thus, RA collapse is sensitive, but not specific, sign of cardiac tamponade. The RA is a thin-walled structure, meaning that brief inversion of the RA wall may be observed

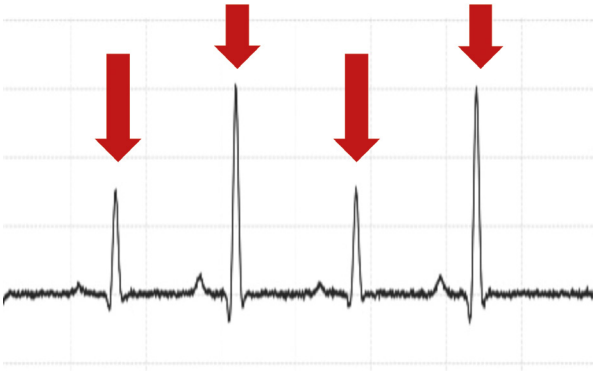

*Fig. 9* — The ECG shows alternating heights of QRS complexes (red arrows) caused by the heart's motion within a fluid-filled pericardium. This phenomenon is identified as electrical alternans.

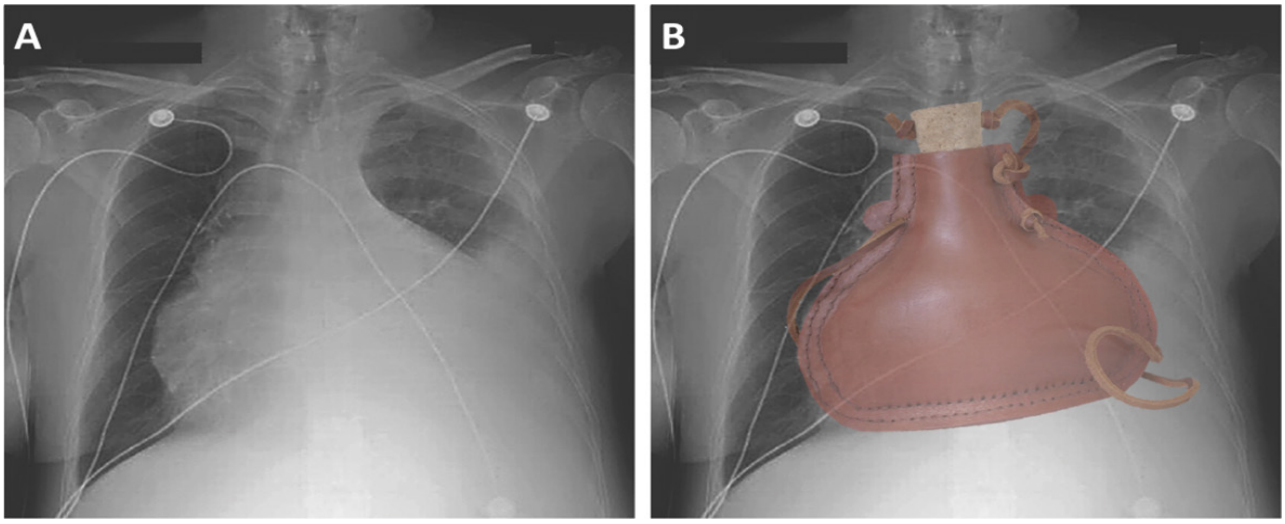


*Fig. 10* — A: Chest X-ray revealing the 'water-bottle' sign in which the enlarged cardiac silhouette in cardiac tamponade mimics the appearance of a water bottle. Figure used with permission29. B: The same image of A with a superimposed water bottle.

in the absence of cardiac tamponade. However, the sensitivity and specificity of this sign improve to 94% and 100%, respectively, if the duration of RA collapse exceeds 30% of the cardiac cycle[31]. In tamponade, prolonged RA collapse is typically observed during expiration and extends into mid or late RV ejection[32] (Fig. 11).

- Right ventricle diastolic collapse

RV diastolic collapse typically occurs in early diastole, when the intracavitary RV pressure and volume are at their lowest points[21]. As in RA collapse, inversion of the RV wall extends further into diastole (i.e., persists for a longer duration) as the severity of tamponade worsens. For the ventricles, collapse occurs in early diastole after the end of the T-wave[4,8]. Typically, RV diastolic collapse occurs with moderate-to-severe increases in PCP and is associated with significant decreased cardiac output (Fig. 11).

RA and RV collapse, as signs of compression, depend on PCP exceeding the intracardiac chamber pressure. Thus, preexisting conditions associated with elevated right heart pressure such as RV hypertrophy and significant pulmonary hypertension may mask these echocardiographic signs of tamponade.

- Inferior vena cava dilatation with reduced inspiratory collapse

A dilated IVC (≥2.1 cm) with associated minimal change in size during forced spontaneous inspiration (sniff) (<50%) is considered to be a sensitive sign of cardiac tamponade physiology, indicating the transmission of elevated PCP to the right heart chambers[4]. The sensitivity for detection of cardiac tamponade is reportedly 92% in patients with both pulsus paradoxus and dilated IVC[33]; however, since IVC plethora is characteristic of many other conditions, it is a nonspecific sign of cardiac tamponade.

- Echocardiographic Doppler indices

  - Transmitral and transtricuspid flow (Fig. 12 A, B): Doppler findings of inspiratory decrease of mitral E-flow of >25% or expiratory decrease

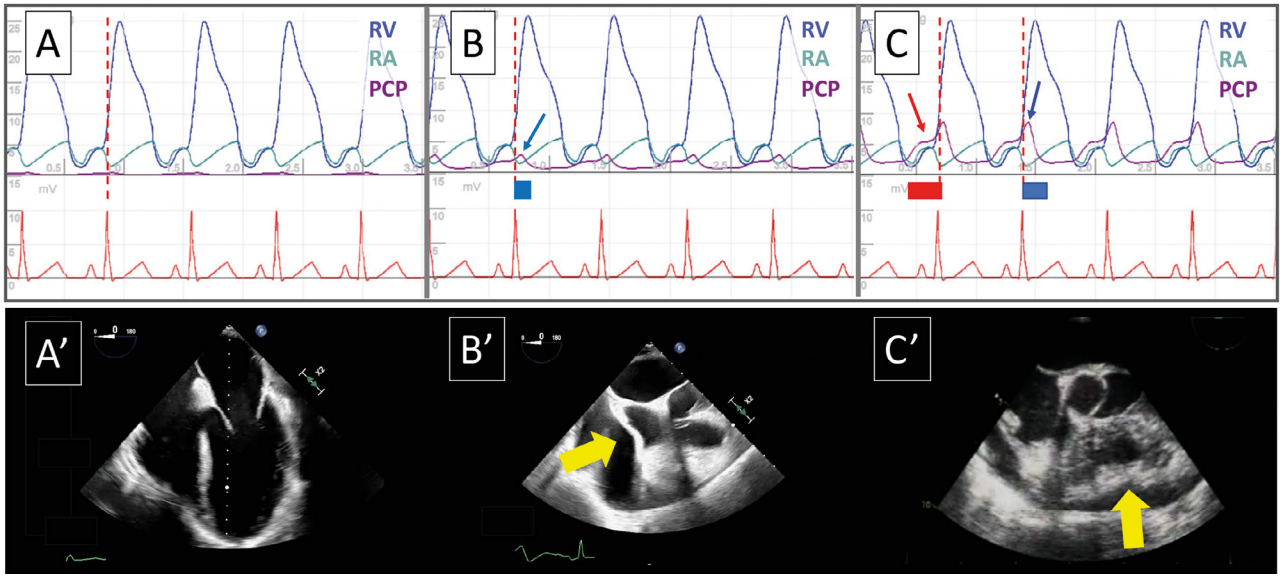


*Fig. 11* — Correlation between hemodynamic pressure and echocardiography. The hemodynamic traces at the top (A, B, C) show RV pressure (blue), RA pressure (green), and Pericardial pressure (purple). The transesophageal images at the bottom (A', B', C') correspond to the hemodynamic traces above.

(A) Normal hemodynamic tracing and echocardiographic appearance (A'). PCP is below the atrial pressure. (B) Pericardial fluid causing increased PCP. The increased PCP will exceed RAP in early systole (blue arrow). Duration of the RA free wall collapse is indicated by the blue box as identified echocardiographically (yellow arrow) during early systole (B'). (C) Further increase in PCP causes it to exceed the diastolic pressure of the RV (red arrow and red box). In addition, also during early systole the PCP exceeds RAP (blue arrow). The duration of atrial collapse (blue box) will be extended during systole. Echocardiography will reveal collapse of the RV (yellow arrow) (C'). Images were generated using Harvi cardiovascular simulation software[40].

Abbreviations: PCP Pericardial pressure; RA Right atrium; RAP Right Atrial Pressure; RV Right Ventricle.

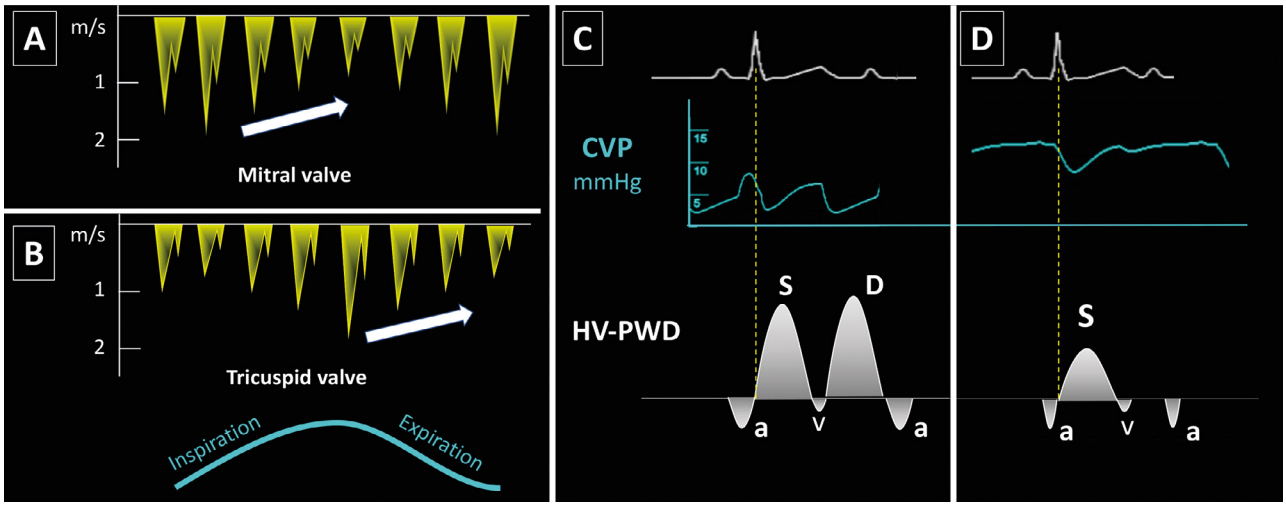


*Fig. 12* — Echocardiographic Doppler flow indices in tamponade.

(A) Decreased Doppler flow velocities are observed across the mitral valve during spontaneous inspiration in tamponade. A drop of >25% is considered consistent with significant tamponade physiology. (B) Tricuspid inflow E velocity decreases significantly with expiration (compared with inspiration). A drop of >40% is considered to be consistent with significant tamponade physiology. (C) Normal RAP tracing and hepatic vein pulsed wave Doppler flow. (D) RAP waveform in tamponade showing a monophasic x- descent (absent y-descent). Hepatic vein Doppler flow shows reduced systolic flow and absent diastolic flow. Images were generated using Harvi cardiovascular simulation software[40].

Abbreviation: RAP Right Atrial Pressure. HV PWD Hepatic vein pulsed wave Doppler.

of tricuspid flow of >40% during normal spontaneous breathing[4,8,34-36] correlate with a sudden leftward shift of the interventricular septum with inspiration on two-dimensional echocardiography. These are signs of increased ventricular interdependence, along with the clinical correlate of pulsus paradoxus. Transtricuspid and transmitral Doppler indices should be used as part of the diagnosis primarily in spontaneously breathing patients, rather than those under mechanical ventilation. Tamponade has been shown to blunt the variation in transmitral flow velocity during mechanical ventilation[34].

- Hepatic venous flow (Fig. 12 C, D). Normal hepatic venous flow is biphasic, with systolic velocity equaling or exceeding diastolic velocity. Peak systolic and diastolic flow velocities both increase during inspiration. Cardiac tamponade hinders ventricular filling, reducing the forward flow velocities and causing near absence of diastolic flow velocity with augmentation during inspiration[4,35-37]. Systolic forward flow is observed in marked tamponade, while diastolic forward flow is absent. The absent y-descent in the RAP tracing corresponds to the flat D-wave in hepatic vein flow (Fig. 12). When no hepatic forward flow is observed except during inspiration, systemic venous and intracardiac pressures are equalized, and cardiac arrest is imminent.

## Management of cardiac tamponade

Management of tamponade primarily aims to alleviate pressure on the heart from the pericardial fluid, lower chamber filling pressures, and restore forward blood flow. This is achieved by drainage, either through percutaneous or open surgical techniques. For the anesthesiologist, it is crucial to maintain the compensatory mechanisms (increased heart rate and contractility) in order to preserve the remaining cardiac function and prevent further reduction in preload. The hemodynamic goals in tamponade are therefore summarized as "fast, full, and squeezed tight"[38,39], meaning:

- Fast: Sinus tachycardia is an important compensation for the low stroke volume. Clinicians should ensure steady cardiac output, avoid bradycardia, and maintain a normal sinus rhythm.
- Full: The intravascular volume should be expanded to augment diastolic pressure and prevent chamber collapse (despite the high RAP observed in tamponade physiology, which relates to chamber compression and not increased transmural filling pressure). This is of particular importance where depletion of the circulating volume is also present.
- Tight: Maintain systemic vascular resistance, which is high in patients with tamponade because of high sympathetic nervous activity. The compensatory cardiovascular mechanisms (tachycardia and raised systemic vascular resistance) must be maintained during induction of anesthesia. The usefulness of inotropic support is generally limited because endogenous adrenergic stimulation is already enhanced under tamponade conditions. Norepinephrine is commonly used to maintain systemic vascular resistance and increase arterial blood pressure when it is low.

## Key Points on cardiac tamponade

1. The pericardium protects the heart, limits excessive cardiac dilatation, and doesn't impede the transmission of intrathoracic pressures.
2. A pericardial effusion and tamponade should be understood as part of a continuum or progression of the same underlying condition, rather than as distinct pathologies. The clinical symptoms experienced will differ depending on the rate and volume of fluid accumulation.
3. Cardiac tamponade is classically considered "obstructive shock," although physiologically it involves restriction of cardiac chamber filling. The accumulation of fluid/blood in the pericardial space, increases PCP and limits diastolic filling of the heart which reduces stroke volume and cardiac output.
4. Hemodynamic signs are tachycardia, elevated RAP, hypotension, and pulsus paradoxus with a reduced pulse pressure.
5. Echocardiographic signs include the presence of pericardial fluid, atrial collapse, RV collapse, and Doppler indices of ventricular interdependence.
6. Anesthetic treatment includes volume administration, maintenance of increased systemic vascular resistance, and the avoidance of high PEEP and large tidal volumes during mechanical ventilation. The latter is critical during initiation of general anesthesia, since positive pressure ventilation reduces venous return and further limits diastolic filling of the heart. The initiation of mechanical ventilation can often lead to a sudden deterioration in the patient's clinical condition. In severe distress, a needle drainage of a small amount of pericardial fluid will improve the pericardial pressure-volume relationship (by moving to the flat part of pressure-volume curve), before the induction of anesthesia and mechanical ventilation.

## Conclusion

Differentiating between simple pericardial effusion and the critical condition of tamponade requires

an understanding of the underlying physiology. Pericardial effusion is initially asymptomatic, whereas cardiac tamponade has a distinct clinical presentation including tachycardia, dyspnea, jugular venous congestion, and pulsus paradoxus. Cardiac tamponade is a clinical diagnosis but echocardiography serves as an important adjunct for differential diagnosis, and as a follow-up examination. Although severe tamponade is a type of cardiogenic shock, the prognosis is good when the condition is treated promptly and adequately.

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