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# What echocardiographic findings suggest a pericardial effusion is causing tamponade?

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## ABSTRACT

**Background:** Pericardial tamponade is neither a clinical nor an echocardiographic diagnosis alone. The echocardiogram carries diagnostic value and should be performed when there is suspicion for tamponade based on the history and physical exam. A pericardial effusion uncovered on point-of-care ultrasound (POCUS) may be mistaken for tamponade and thereby lead to inappropriate and invasive management with pericardiocentesis. **Objective:** This narrative review will summarize the echocardiographic findings and associated pathophysiology that support the diagnosis of pericardial tamponade. It will provide a succinct description of the core findings for which emergency physicians should evaluate at the bedside, along with potential pearls and pitfalls in this evaluation. Labeled images and video clips are included.

**Discussion:** The core echocardiographic findings of pericardial tamponade consist of: a pericardial effusion, diastolic right ventricular collapse (high specificity), systolic right atrial collapse (earliest sign), a plethoric inferior vena cava with minimal respiratory variation (high sensitivity), and exaggerated respiratory cycle changes in mitral and tricuspid valve in-flow velocities as a surrogate for pulsus paradoxus.

**Conclusion:** The emergency physician must recognize and understand the core echocardiographic findings and associated pathophysiology that suggest pericardial tamponade. Together with the history and clinical exam, these findings can help make the overall diagnosis and determine management.

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## 1. Introduction

Pericardial tamponade occurs when fluid within the pericardial sac impairs filling of the right-sided chambers, leading to a decrease in cardiac output and hemodynamic compromise. It cannot be diagnosed solely by echocardiography or clinical exam. Instead, the echocardiogram carries diagnostic value and should be performed when there is suspicion for tamponade based on the history and physical exam. Unfortunately, a pericardial effusion discovered on point-of-care ultrasound (POCUS) in the emergency department (ED) may be mistaken for tamponade and thereby lead to appropriate and invasive management in the form of pericardiocentesis.

This narrative review will summarize the echocardiographic findings and associated pathophysiology that may manifest as pericardial tamponade. It will provide a succinct description of the core findings for which emergency physicians should evaluate at the bedside. It will also present potential pearls and pitfalls in this point-of-care ultrasound (POCUS) evaluation.

## 2. Methods

This is a narrative review summarizing the physiology studies and echocardiographic descriptions of pericardial tamponade, with a discussion of the pearls and pitfalls in the POCUS evaluation. A literature review of the PubMed and Google Scholar databases was performed with search date 1960 to December 2017. Search terms included “pericardial tamponade”, “cardiac tamponade”, “pericardial effusion”, “[each of those terms] + echocardiography”, and “[each of those terms] + ultrasound.” The author included studies assessing the sensitivity, specificity, and accuracy of the echocardiographic findings. Experimental studies, case controls, cohort studies, and expert reviews were included, as determined by relevance to the narrative review. Commentaries and letters were excluded.

## 3. Discussion

### 3.1. Pericardial effusion

The pericardial cavity between the myocardium and pericardial sac normally contains <50 mL of lubricating fluid. With only 15–35 mL, separation of these layers can be visualized with high sensitivity and accuracy by point-of-care ultrasound (POCUS) [1,2] in both the medical and

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**Table 1**  
Classification of pericardial effusions by size.

Size	Volume	Myocardium-epicardium diameter in diastole	Descriptors
Trivial	<50 mL	Seen only in systole	Posterior atrioventricular groove; may be physiologic [8]
Small	50–100 mL	<10 mm	Seen throughout cardiac cycle
Moderate	100–500 mL	10–20 mm	Surrounds entire heart [9]
Large	>500 mL	>20 mm	

trauma settings [3,4]. With sufficient volume, the heart may be seen swinging within the pericardial sac (Video 1), though the sensitivity is low [5]. This is manifested on the electrocardiogram (ECG) as electrical alternans.

It is important to acquire multiple different views of the effusion for better characterization, because a single cardiac view can misrepresent the volume of the effusion. Although the size of the effusion can be estimated (Table 1) [6,7], the size of the effusion does not necessarily predict pericardial tamponade.

More important in predicting tamponade are the rate of rise of the effusion and the pericardial compliance [8]. This in turn determines the filling pressures within the pericardial space in relation to those within the cardiac chambers during diastole. Since the pericardial space is acutely non-compliant, even 50 mL of fluid can lead to hemodynamic compromise if it has accumulated rapidly [9]. Conversely, larger volumes can be accommodated without causing hemodynamic compromise by the stretching of a more compliant pericardium over longer periods of time. Eventually, however, the pericardium's compliance will reach a limit beyond which even a small increase in pericardial fluid volume will lead to the rise in intrapericardial pressure that causes tamponade (Fig. 1) [10].

Among others, there are two more common false-positives that may be mistaken for a pericardial effusion: a pleural effusion and a pericardial fat pad. To differentiate a pericardial from a pleural effusion, the descending aorta can be used as a landmark in the parasternal long axis (PLAX) view. A pericardial effusion will lie anterior to this structure, whereas a pleural effusion will lie adjacent or posterior (Fig. 2). In contrast to an anechoic pericardial effusion, a pericardial fat pad will appear echogenic and is often described as having a "stippled appearance." It will often be distributed in the anterior atrioventricular groove. Rather than seeming to compete with the cardiac chambers for space within the pericardial sac, the fat pad moves in concert with the myocardium during the cardiac cycle (Video 2).

A pericardial effusion can be visualized in any of the four main cardiac views: PLAX (Fig. 3), parasternal short axis (PSAX) (Fig. 4), apical 4-chamber (A4C) (Fig. 5), and subxiphoid (SX) (Fig. 6). The best views for detection of pericardial effusion are the PLAX and SX views. In the former view, a small effusion can easily be seen either anterior to the right ventricular outflow tract or posterior to the left ventricular posterior wall. In the latter view, an effusion can easily be seen in the space

between the liver and the right ventricle (RV). Due to liver tissue's homogeneity and inherently high propagation speed of sound wave transmission (1550 m/s), it serves as an excellent acoustic window for propagation of sound waves with little attenuation (attenuation coefficient of 90).

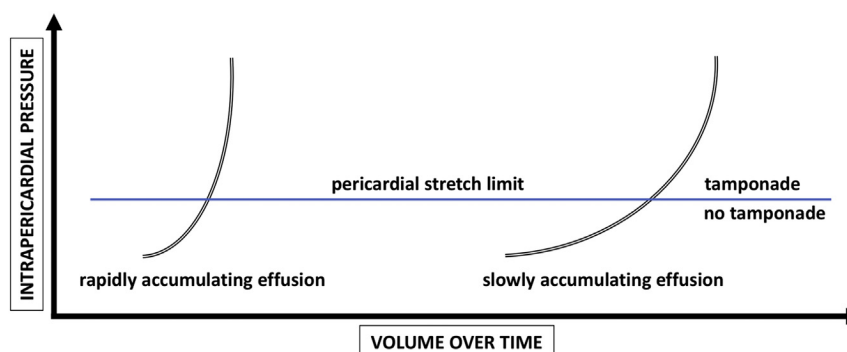
### 3.2. Diastolic right ventricular collapse

The RV collapses when the intrapericardial pressure exceeds the intracardiac pressure. The intrapericardial pressure is proportional to the pericardial fluid volume and the stiffness of the pericardial sac as follows [11]: Intrapericardial pressure (P) = Intrapericardial fluid volume (V) × Pericardial stiffness ( $\Delta P/\Delta V$ ). In other words, the intrapericardial pressure will be increased by: a larger pericardial effusion volume and/or a change in intrapericardial pressure that is greater than the corresponding change in intrapericardial volume.

The RV has a thinner, more compliant wall and lower pressure system than the left ventricle (LV). Its pressure is at its lowest in early diastole, so naturally, this is the point in the cardiac cycle at which an increase in intrapericardial pressure will cause the ventricle to bow inward. The severity of tamponade is correlated with the duration of the chamber's collapse [12], that is, the period of diastole over which the intrapericardial pressure exceeds the RV filling pressure. The outflow region collapses first, followed by the basal segment once tamponade progresses. Of note, in the rarer case of a loculated effusion, the focal effusion may cause its adjacent chamber to collapse during diastole, and not necessarily the RV.

Diastolic collapse of the RV carries a high specificity (75–90%), with a relatively lower sensitivity (48–60%) [13–16]. This is more specific but less sensitive than systolic right atrial (RA) collapse [17]. These values for sensitivity and specificity may be affected by changes in blood volume. Moreover, if the RV filling pressures are elevated at baseline, it follows that diastolic collapse will less likely occur. This may be the case with: acute or chronic cor pulmonale, pulmonary hypertension, severe LV failure, or other etiologies of RV hypertrophy [18–22]. Positive-pressure ventilation will exert this effect as well. In contrast, diastolic RV collapse may occur earlier if the filling pressure is lower at baseline, such as with hypovolemia [23].

Diastolic RV collapse can be visualized in all four cardiac views: PLAX (Video 3) (Fig. 7), PSAX (Video 5), A4C (Video 6), and SX views (Video



**Fig. 1.** Tamponade physiology is predicted by the rate of rise of the effusion and the pericardial compliance. At a certain threshold, the pericardium's compliance will reach a pericardial stretch limit, where even a small increase in pericardial fluid volume will lead to the rise in intrapericardial pressure that causes tamponade.



**Fig. 2.** A pericardial (anterior) and pleural effusion (posterior) in relation to the descending aorta in the PLAX view.

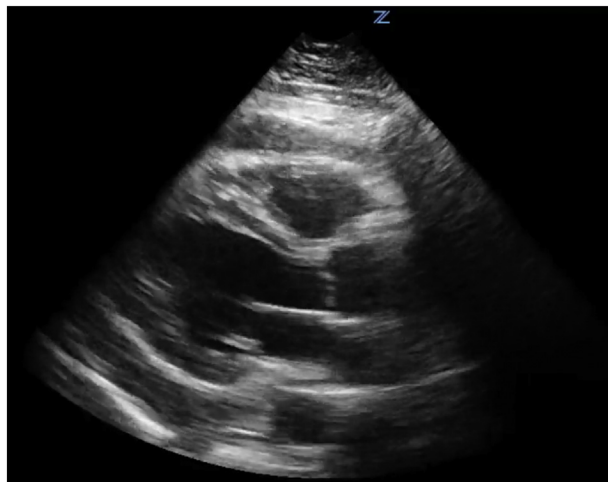
8). The best views for detection of diastolic RV collapse are the PLAX, A4C, and SX views. In the PLAX view, the diastolic phase of the cardiac cycle corresponds to the visualized opening of the mitral valve (MV), whereas both the MV and tricuspid valves (TV) can be seen opening in the A4C and SX views.

Tamponade physiology is less likely in instances where the RV does not clearly collapse during diastole: PLAX (Video 4), A4C (Video 7), and SX views (Video 9).

### 3.3. Systolic right atrial collapse

The RA is at its lowest pressure during systole, or more precisely, in late diastole at the onset of atrial relaxation. During this period, it is most susceptible to collapse from increased intrapericardial pressure. Its pressure during systole is lower than that of the RV in diastole, so systolic RA collapse is therefore the earliest echocardiographic sign of tamponade [24].

The specificity of systolic RA collapse varies for tamponade (33–100%) [16,25,26]. It increases when duration of chamber collapse lasts >1/3 of the cardiac cycle [14,27,28]. Otherwise, it may simply be mistaken for normal atrial systole [28,29]. The sensitivity for tamponade is higher, ranging from 50% in early tamponade to 100% with its



**Fig. 3.** Pericardial effusion in PLAX view.



**Fig. 4.** Pericardial effusion in PSAX view.

progression [16,26]. Altogether, the absence of any chamber collapse (RV or RA) has a 90% negative predictive value for tamponade [29].

Systolic RA collapse can be best visualized in the A4C view (Video 10). It can also be visualized in the SX view (Video 11).

### 3.4. Plethoric inferior vena cava with minimal respiratory variation

During normal inspiration in the spontaneously breathing patient, negative intrathoracic pressure leads to increased venous return from the inferior vena cava (IVC) to the RA. In pericardial tamponade, the RA cannot fully accommodate the incoming preload due to compression by increased intrapericardial pressure. As a result, the IVC remains dilated, or plethoric, with minimal respiratory variation. This is a very sensitive sign for tamponade (95–97%) [1,16,18,30,31] and is useful for its high negative predictive value [30,32]. It has much lower specificity (~40%) and can be caused by chronic lung disease as well as other cardiac conditions including congestive heart failure (CHF) and tricuspid regurgitation, among others [16,18].

A plethoric IVC has been defined as having diameter >2.1 cm with <50% inspiratory reduction [1,30,32,33]. However, given its high negative predictive value for tamponade, a visualized eyeball assessment of the IVC being plethoric (versus collapsible) may be sufficient for experienced sonographers.

A plethoric IVC can be visualized from the sagittal plane below the xiphoid process (Video 12). The diameter should be evaluated about 2–3 cm from the IVC-RA junction, usually around the level of the hepatic vein draining into the IVC (Fig. 8). Specific diameter measurements can be obtained in M-mode by placing the cursor through this point.



**Fig. 5.** Pericardial effusion in A4C view.

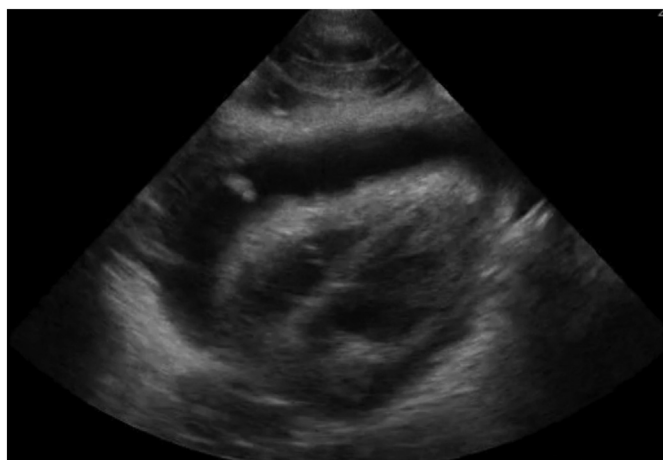


Fig. 6. Pericardial effusion in SX view.

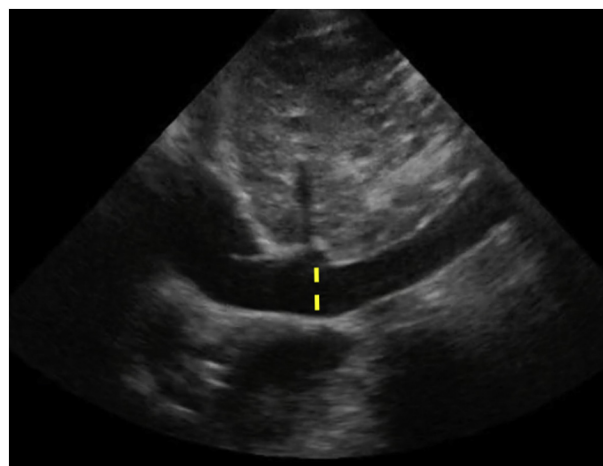


Fig. 8. Evaluation of the IVC diameter 2–3 cm from the IVC-RA junction, around the level of the hepatic vein drainage into the IVC.

3.5. Exaggerated respiratory cycle changes in mitral and tricuspid in-flow velocities as a surrogate for pulsus paradoxus

Pulsus paradoxus is the exaggeration of the normal respiratory variation in systolic blood pressure (SBP) that can occur with tamponade. During inspiration (negative intrathoracic pressure), air is pulled into the lungs. There is a drop in pulmonary vascular resistance, and blood flow through the right side of the heart (across the TV) increases. The two ventricles, and now the effusion as well, compete for space within the pericardial sac. The increased intrapericardial pressure from the effusion causes the RV and LV end-diastolic pressures to equalize. As a result, with inspiration, the intraventricular septum indents further toward the LV (versus without an effusion) in an exaggeration of ventricular interdependence. Less blood thereby flows through the left side of the heart (across the MV) and leads to a drop in LV stroke volume. This manifests as the abnormally large decrease in SBP > 20 mmHg seen with pulsus paradoxus.

A surrogate for the changes in blood flow through the mitral (i.e. left side of heart) and tricuspid (i.e. right side of heart) valves is the respective velocities of this blood flow through the valves [33,34]. These velocities and their changes during the respiratory cycle can be measured using Doppler echocardiography. This is achieved using the A4C view,

in which blood flow through both MV and TV is oriented roughly parallel to the direction of sound waves emitted from the transducer (Fig. 9).

Studies have shown variance in the percentages of change that define pulsus paradoxus [29,33–36]. This occurs due to imprecise and varying Doppler gate alignments, sample volumes, and patient positions during measurement. Tamponade physiology will produce an approximately 25% decrease in MV in-flow velocity and 40% increase in TV in-flow velocity with inspiration. Of note, this may also occur with marked dyspnea, severe chronic obstructive pulmonary disease, and pulmonary embolism [37]. Variation of in-flow velocities up to 10% can even occur without tamponade. For pericardial tamponade, pulsus paradoxus itself has a sensitivity of 82% (95% CI 72%–92%) [38], and in the presence of pericardial effusion, a positive likelihood ratio of 3.3 (95% CI 1.8–6.3) and negative likelihood ratio of 0.03 (95% CI 0.01–0.24) [39].

4. Conclusion

Bedside echocardiography can help diagnose pericardial tamponade when there is already a degree of clinical suspicion. The core findings

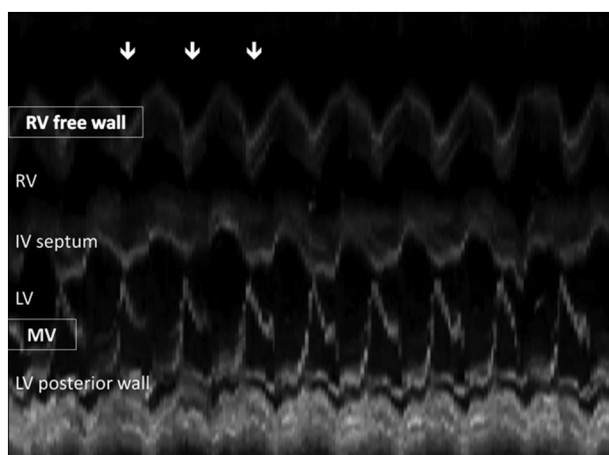


Fig. 7. M-mode view of diastolic collapse in PLAX view. In a tachycardic patient, the precise timing of the MV opening and RVOT anterior wall movement can be difficult to ascertain. In such cases, M-mode can be used to acquire a still image. This will demonstrate whether the RVOT anterior wall is bowing downward at the same time as the E-wave of the MV opening.

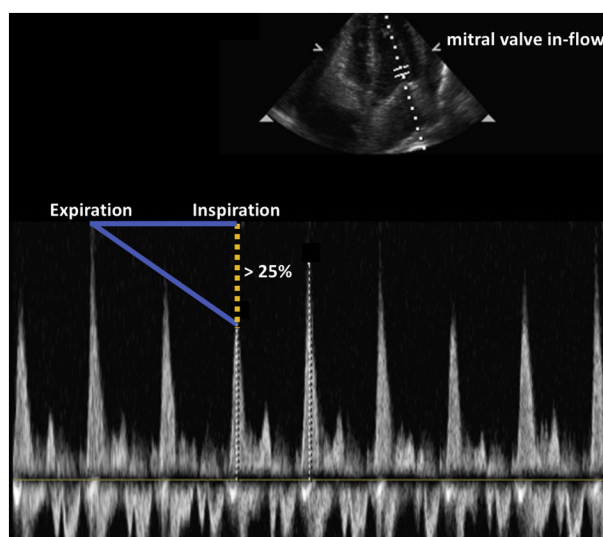


Fig. 9. Pulse wave Doppler measuring respiratory cycle changes in the MV in-flow velocity as a surrogate for pulsus paradoxus. In the A4C view, the gate is placed at the tips of the valve leaflets. The flow velocities should be measured at the first heartbeat after the change in respiratory phase. In other words, sample the tallest E-wave peak and compare it to the lowest.



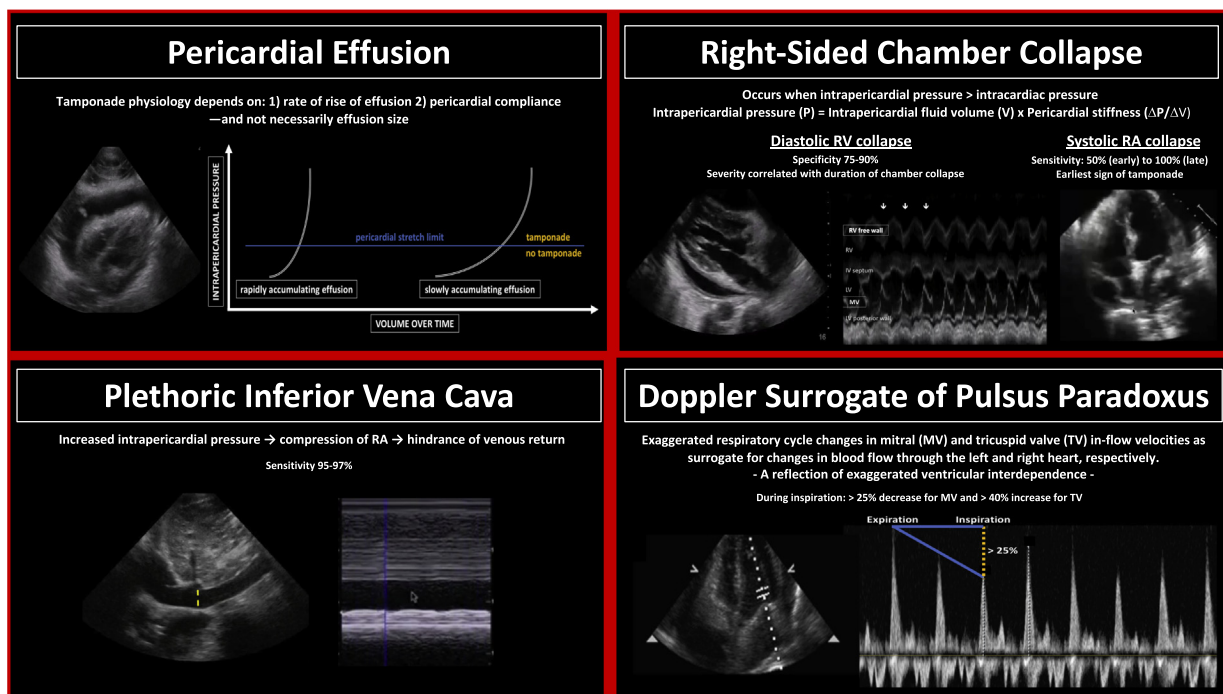


Fig. 10. A pericardial tamponade pocket primer.

include: a pericardial effusion, diastolic RV collapse (high specificity), systolic RA collapse (earliest sign), a plethoric IVC with minimal respiratory variation (high sensitivity), and exaggerated respiratory cycle changes in MV and TV in-flow velocities as a surrogate for pulsus paradoxus (Fig. 10). These findings can supplement the history and physical exam to determine the appropriate management, including whether an invasive pericardiocentesis is indicated.

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ajem.2018.11.004>.

### Sources of support

N/A.

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