Chapter 9
Tamponade

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Case Presentation

The patient is a 47-year-old female with history of systemic lupus erythematosus (SLE) who presents with exertional dyspnea. She reports development of peripheral edema in the past month and has had onset of exertional dyspnea in the past few days.

Salient physical examination findings include a heart rate of 92, blood pressure of 136/72, elevated JVP at 16 mmHg, and grade I/VI early systolic murmur at left lower sternal border. The lung fields are clear. There is 2+ pitting lower extremity edema. Pulsus paradoxus is measured to be 8 mmHg.

A chest X-ray shows an enlarged cardiac silhouette. An EKG shows electrical alternans (Fig. 9.1). An urgent echocardiogram shows increased respirophasic variation across mitral and tricuspid valves (Fig. 9.2a, b).

The patient is then admitted to the hospital for further evaluation and management. Over the course of the admission, the patient becomes gradually confused. Laboratory evaluation reveals evidence of mild renal dysfunction and a mild transaminitis noted in her hepatic profile. She now has a heart rate of 108 and her blood pressure is 92/56. She is transferred to the Intensive Care Unit and a pulmonary artery catheter is placed with right atrial tracings showing blunted *y* descent (Fig. 9.3a). She subsequently receives a pericardiocentesis with improvement in hemodynamics (post-pericardiocentesis tracings shown in Fig. 9.3b). The patient improves clinically and subsequently is discharged on an intensified treatment regimen for SLE with follow-up echocardiogram scheduled.

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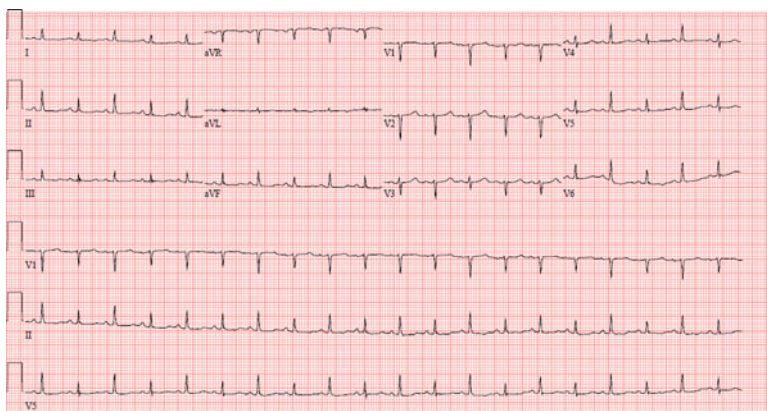
**Fig. 9.1** EKG demonstrating electrical alternans

Introduction

Anatomy

The myocardium is encased in a relatively rigid and noncompliant structure called the pericardium. The pericardium is formed by two layers. The outer sac, called the fibrous pericardium, is composed of fibrous tissue and encases the inner layer of pericardium and the heart. The inner sac has two layers—called the parietal and visceral pericardium. The parietal pericardium is attached to the internal surface of the fibrous pericardium and is contiguous with the visceral pericardium as it gets reflected on the cardiac surfaces. In between the visceral and the parietal pericardium lies the pericardial space. This space normally contains up to 50 mL of serous fluid to maintain a low friction environment for the myocardium as it contracts and relaxes within the pericardium throughout the cardiac cycle. The pericardium effectively restrains the cardiac chambers to a confined space and due to this pericardial constraint, any change in the volume of one chamber of the heart is reciprocated by changes in other chambers in the opposite direction [1]. It is this complex interaction of pericardial space with the intracardiac chambers that leads to the hemodynamic consequences of pericardial effusions.

Several diseases and complications of invasive procedures may lead to pathologic accumulation of fluid in the pericardial space, which may impact cardiac output. When the accumulation of pericardial fluid interferes with diastolic filling of the heart, hemodynamic consequences associated with varied clinical presentations including tamponade ensue.
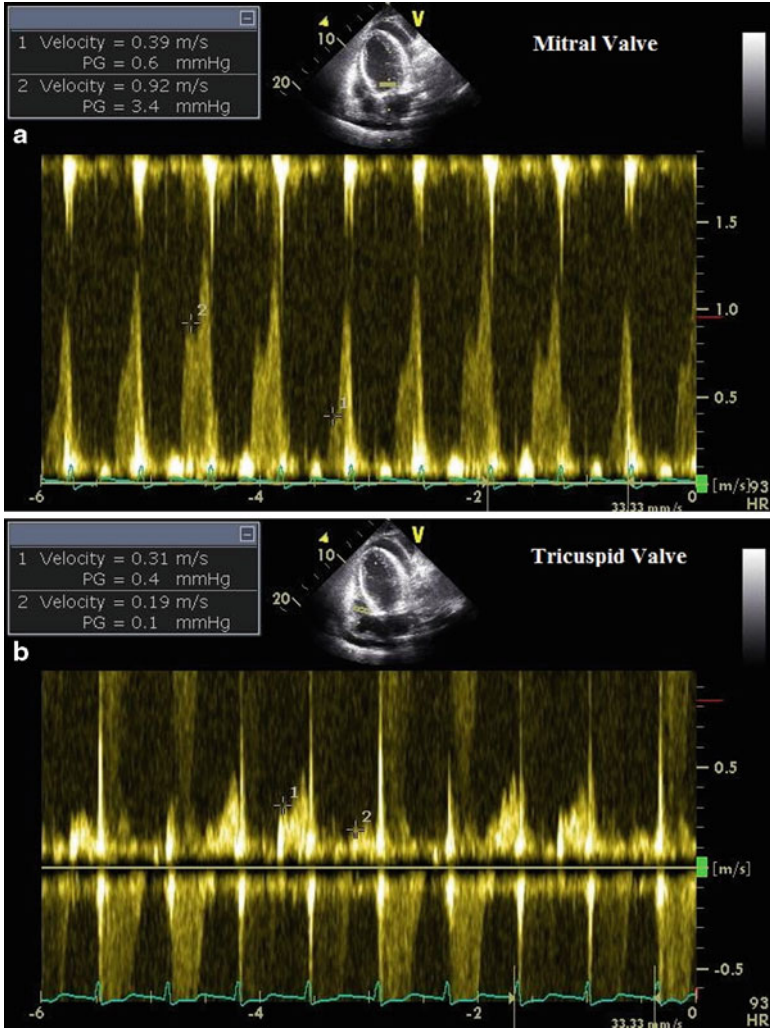
Fig. 9.2 Echocardiogram with Doppler evaluation demonstrating respiratory flow variation across mitral (**a**) and tricuspid valves (**b**)
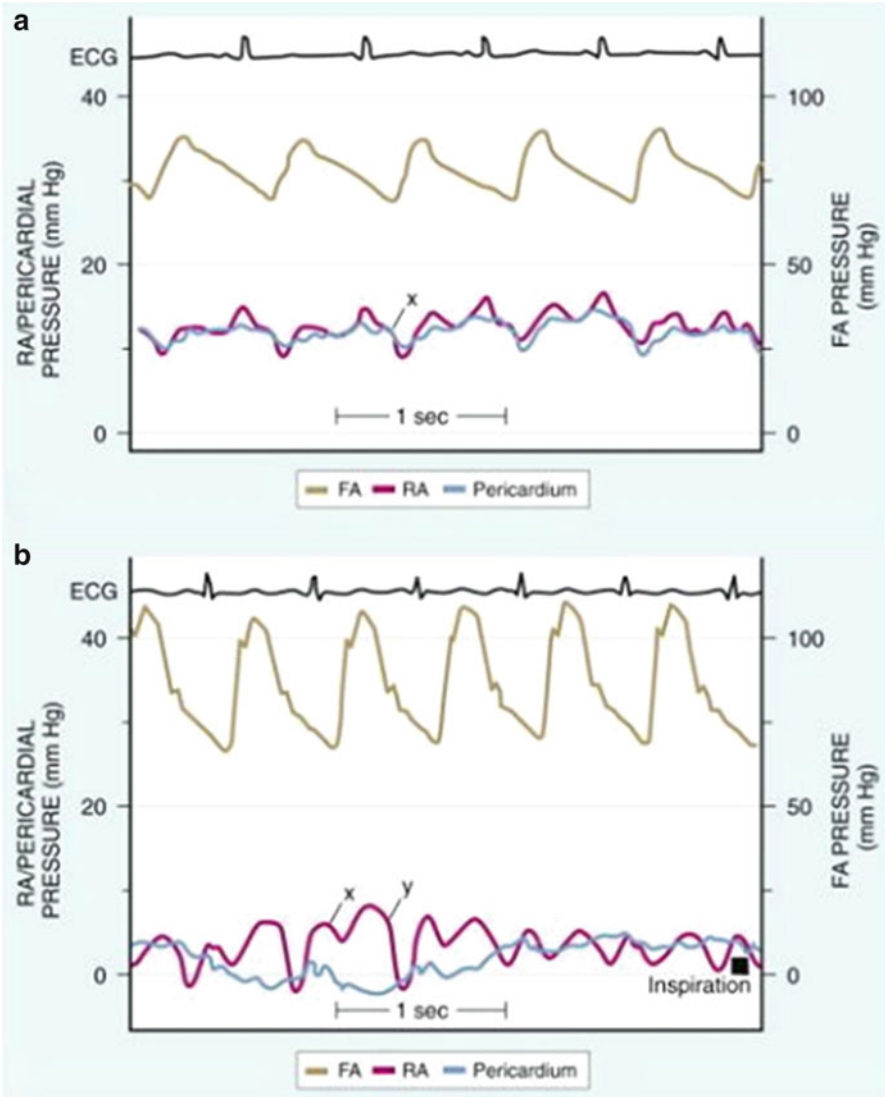
Fig. 9.3 Pre-pericardiocentesis—(**a**) and Post-pericardiocentesis—(**b**) (Reprinted from modification by LeWinter MM. Pericardial diseases: In: Braunwald's heart disease: a textbook of cardiovascular disease. Philadelphia: Saunders Elsevier; 2008. p. 1836 from original by "Lorell BH, Grossman W. Profiles in constrictive pericarditis, restrictive cardiomyopathy and cardiac tamponade. In: Baum DS, Grossman W, editors. Grossman's cardiac catheterization, angiography, and intervention. Philadelphia: Lippincott Williams & Wilkins; 2000. p. 840")

Pathophysiology of Tamponade

Progression of a pericardial effusion to tamponade depends on several factors. These include the rate of fluid accumulation, tensile properties of the pericardium, intracardiac pressures, and tensile properties of the myocardium. Ultimately, it is the intrapericardial pressure and its interaction with intracardiac pressures (transmural pressure) that determines the presence of tamponade.

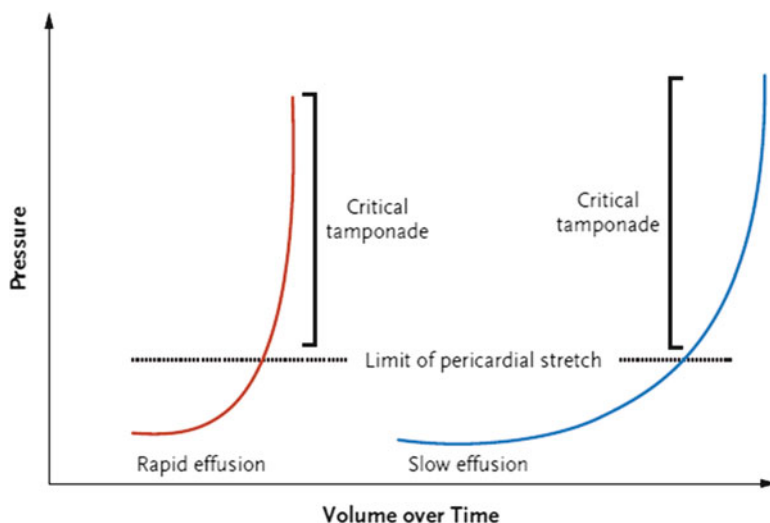$$\text{Transmural pressure} = \text{intracardiac pressure} - \text{intrapericardial pressure}$$

When the intrapericardial pressure equalizes or exceeds that of the intracardiac chambers, the transmural pressure is then <0 and impaired filling occurs. In patients with pericardial effusions, negative transmural pressure is more often due to increased pericardial pressure but decreased intracardiac pressures also contribute to the development of tamponade.

Determinants of intrapericardial pressure include the volume of pericardial fluid and the compliance of the pericardium. The relationship between size of the effusion and the associated increase in pressure is not linear and is modified by the compliance of the pericardium itself. As such, a larger but chronic effusion might be better tolerated than a smaller but rapidly accumulating one due to the higher compliance of the pericardium that develops in the chronic setting (Fig. 9.4). Therefore, size of the effusion should not be used as a sole criterion for the presence of tamponade.

Progression of a pericardial effusion to tamponade also depends on the intravascular volume status of the patient as well as the compliance of the myocardium. Patients who are intravascularly depleted are more likely to present with compromised hemodynamics due to diminished filling pressures in the cardiac chambers. On the contrary, those with ventricular hypertrophy or restrictive heart disease may not present with tamponade physiology for a given intrapericardial pressure as this pressure is not readily transmitted to the intracardiac chambers due to the diminished compliance of the myocardium.

Stages of Pericardial Tamponade

Pericardial tamponade was previously thought of as an "all or none" clinical disorder [2]. However, it is now recognized that patients might present at different stages along the spectrum of tamponade physiology (Table 9.1). The first stage (pretamponade) occurs when the pressure in the pericardium is less than right and left ventricular end diastolic pressures (transmural pressure still >0). In this stage, the effusion does not have a clinically significant hemodynamic impact; however there may be subtle changes in echocardiographic respiratory flow variation across mitral and tricuspid valves and minimally increased pulsus paradoxus pressure (<10 mmHg). In the second stage, the pericardial pressure equals the right ventricular diastolic pressure leading to collapse of the right ventricle during early

Fig. 9.4 Intrapericardial pressure that is critical in determining the transmural pressure is affected by the time course of fluid accumulation. Rapidly developing effusions reach the limits of the pericardial stretch sooner, whereas slowly developing effusions are better tolerated due to time allowed for the pericardium to adapt to the changes imposed by the effusion (adapted from Spodick DH. Acute cardiac tamponade. N Engl J Med. 2003;349(7):684–90)

Table 9.1 Three stages of tamponade

1. Pretamponade: No clinical evidence of tamponade as transmural pressure >0, however exaggerated tricuspid and mitral inflow patterns noted
2. Early tamponade: Right sided chambers are affected as the pericardial pressure increases. Clinical presentation might become apparent
3. Tamponade: Both right and left sided chambers are affected as there is equalization of pressures with pericardium. Clinical tamponade is evident

diastole. In this case, while there is no collapse in the left ventricle, clinical and hemodynamic evidence for tamponade is present with decrease in right ventricular stroke volume. Hypotension, compensatory tachycardia, and elevated pulsus paradoxus might be present. Finally, the most severe stage presents when left ventricular filling is also affected by the increased pericardial pressure. In this case, there is equilibration of pericardial, right ventricular and left ventricular diastolic pressures leading to severely impaired filling of the ventricles and low stroke volume (Fig. 9.5) [3]. Patients in the final stage of tamponade require expeditious diagnosis with direct intervention to drain the pericardial fluid and reestablish hemodynamic stability.

Pathophysiology of Cardiac Tamponade
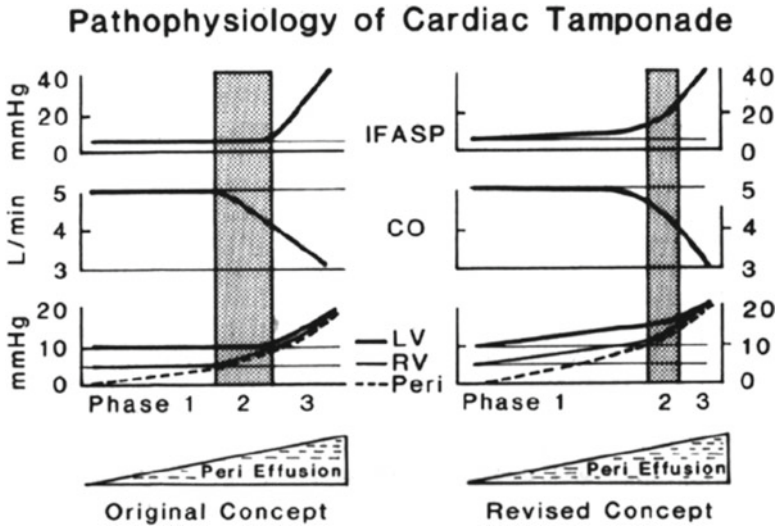
Fig. 9.5 Stages of pericardial tamponade showing pressures in right atrium, right ventricle, pulmonary capillary wedge pressure, and intrapericardium (*Peri* pericardium; *RV* right ventricle; *LV* left ventricle; *CO* cardiac output; *IFASP* inspiratory decrease in arterial systolic pressure). Initial concept and its revision with further data is shown below detailing changes in the pressures across stages of tamponade (adapted from Reddy et al. [3])

Low-Pressure Tamponade

This condition has been described as a form of tamponade in which relatively low intrapericardial pressure causes cardiac chamber compression in patients with low right sided pressures. Reported to be present in 20% of patients with tamponade physiology, this condition might be associated with hypovolemia, vasodilator, and diuretic use [4]. Overall, findings from pulmonary artery catheterization are similar with equalization of diastolic pressures albeit at lower pericardial and diastolic pressures. Given the low pressures, patients might not present with typical findings in physical examination (elevated JVD, tachycardia); however they benefit from pericardiocentesis with improved cardiac indices and arterial pressures [4]. When evaluating a patient with a pericardial effusion and hypotension, this clinical entity should be kept in mind.

Diagnosis

Physical Exam Findings

While physical exam might vary in patients with tamponade, there are certain classic findings as defined by Dr. Claude Beck in 1935: Low blood pressure, jugular venous distension, and muffled heart sounds. While all three might not be present

even in the most advanced cases of tamponade, hypotension and tachycardia with narrow pulse pressure should alert the clinician to its presence.

Pulsus Paradoxus

Originally described by Kussmaul in 1873, pulsus paradoxus is defined by a ≥10–12 mmHg or a >9% decline in arterial systemic pressure with inspiration [5]. While the etiology of this drop is debatable, the most widely accepted explanation has to do with intrathoracic pressure changes with inspiration and the fact that the total volume within the pericardium is fixed. Intrapericardial pressure normally ranges from −5 to +5 cm of water and fluctuates significantly with respiratory cycle. The drop in the intrathoracic pressure with inspiration is transmitted to the pericardium and the right atrium which results in increased venous return to the right side of the heart. The opposite occurs in the left heart where there is diminished filling of the left ventricle in early inspiration and slightly reduced systemic stroke volume. The reverse is true in expiration: As the intrathoracic pressure increases, the right sided filling diminishes resulting in improved filling of the left ventricle leading to a higher systemic stroke volume. In a normal individual, these respiratory changes result in a 3–4 mm change in systolic pressure as measured peripherally. When there is a hemodynamically significant tamponade however, there is interventricular dependence of LV and RV, which leads to exaggeration of the above described pressure changes with respiration. This is due to the fixed space that the myocardium is constrained with as the increased filling of the RV compromises the filling of the LV, which subsequently leads to the diminished systemic stroke volume with the next contraction. As the underfilled LV cannot generate the normal stroke volume, the blood pressure drops >10 mmHg, which is recognized as pulsus paradoxus on physical exam.

While checking for the presence of pulsus paradoxus allows for quick assessment of patient at the bedside, several coexisting conditions might diminish the accuracy of this test. In patients who have lung disease and shock, pulsus paradoxus might already be present without tamponade. Also, this finding might be absent despite a hemodynamically significant tamponade in the presence of coexisting LV dysfunction with elevated LVEDP, severe aortic insufficiency, pulmonary hypertension, and right ventricular hypertrophy [6, 7].

Echocardiographic Findings

Although tamponade is a clinical diagnosis, echocardiographic evaluation of the patient is a very useful adjunct as it provides direct visualization of the pericardium, the cardiac chambers, and early signs of tamponade physiology. Several echocardiographic parameters may be used to evaluate for evidence of tamponade including M-mode, Doppler interrogation, and 2D visualization. While 2D and M-mode provide visual confirmation of the effusion and the collapse of chambers, Doppler

Table 9.2 Evidence of tamponade on echocardiogram

| |
|---|
| Presence of an effusion. Size, while predictive, is not a reliable determinant of clinical tamponade |
| Early diastolic collapse of right ventricle |
| Late diastolic right atrial inversion |
| Dilated inferior vena cava (>2.5 cm) with failure to collapse of the vessel with inspiration >50% |
| Respiratory inflow variations across the tricuspid valve (>40%) and the mitral valve (>25%) |

Table 9.3 Typical order of findings in tamponade

Tricuspid inflow variation with respiration increased
Mitral inflow variation with respiration increased
Right atrial exaggerated late diastolic collapse (sensitive, nonspecific finding on echocardiogram)
Right ventricular outflow tract collapse (specific finding on echocardiogram)
Right ventricular free wall collapse
Left ventricular free wall collapse

evaluation can be useful in determining the above mentioned flow variations in tamponade (Table 9.2).

Exaggerated diastolic right atrial free wall collapse and diastolic right ventricular outflow tract collapse are early signs of elevated intrapericardial pressure. Brief inversion of the atrial wall can be seen during atrial contraction and does not represent tamponade physiology. More prolonged atrial wall inversion (>1/3 of cardiac cycle) is more indicative of increased intrapericardial pressures. Right ventricular outflow tract is the most easily compressible component of the right ventricle and may also be affected early from elevated intrapericardial pressure. As the intrapericardial pressure increases, the right ventricular free wall may also collapse in diastole signaling to the progression of disease. Collapse of these above right sided chambers is indirect evidence that the intrapericardial pressure have exceeded the intracardiac pressures leading to the inversion of the free walls [1]. As tamponade progresses, further collapse of the left ventricle can also be seen due to similar mechanisms (Table 9.3). Both 2D and M-mode techniques may be used to identify these anatomic manifestations of tamponade.

Doppler evaluation is very helpful in assessing the hemodynamic impact of the pericardial effusion. Outflow across aortic and pulmonary valve (Velocity Time Integral) and inflow across mitral and tricuspid valves (peak velocity) can be measured. In a normal patient, these parameters of flow demonstrate minimal respiratory variation with the pulmonic and aortic valve velocity time integral varying <10% and the mitral and tricuspid valve peak inflow velocities varying <15% and <25%, respectively. In patients with tamponade, however, respiratory variation across the mitral valve is usually >25% and that across the tricuspid valve >40% (Fig. 9.6). This exaggerated variation in flow across valves provides indirect evidence for ventricular interdependence and suggests a hemodynamically significant effusion.
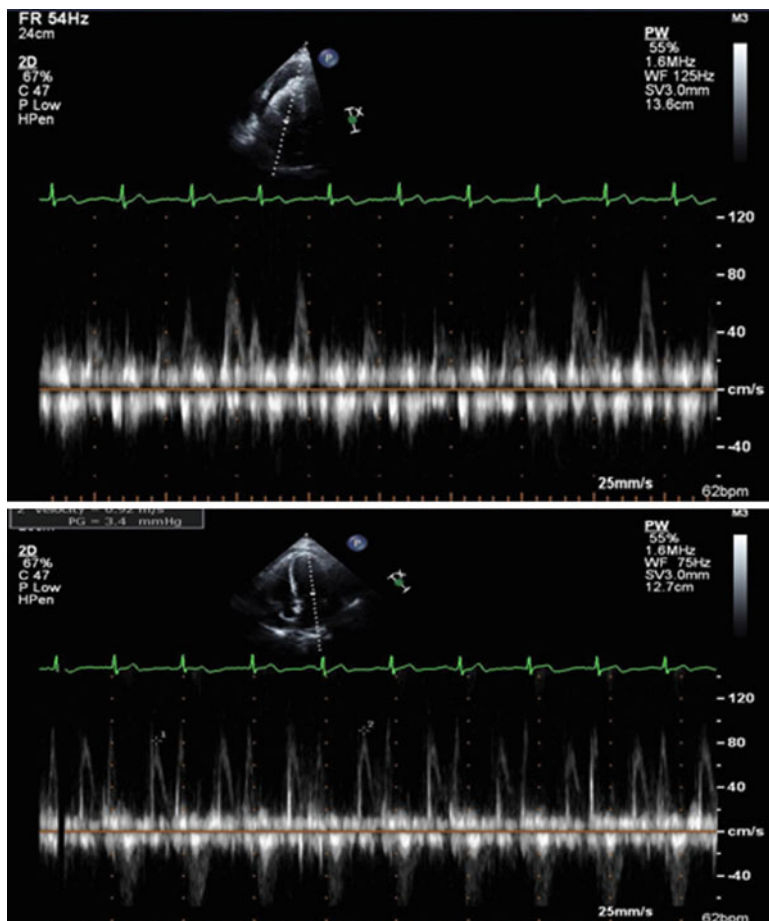
Fig. 9.6 *Top panel* showing tricuspid and *bottom panel* showing mitral valve inflow pattern

Also helpful, although often ignored, is the Doppler flow pattern of the hepatic vein. The hepatic vein flow pattern reflects the venous pressure. The normal hepatic vein flow has three major waves: positive systolic and diastolic flows (towards the atrium) and a negative flow corresponding to the atrial contraction (away from the atrium). The systolic flow wave corresponds to the x descent and the diastolic flow wave to the y descent. In patients with tamponade physiology, blood enters the right atrium only or mostly during ventricular systole. This is reflected in the hepatic vein flow as a predominant systolic wave with decreased or even absent diastolic flow wave (i.e., corresponding to blunted y descent on the RA tracing). After pericardiocentesis, the diastolic flow is reestablished (Fig. 9.7).
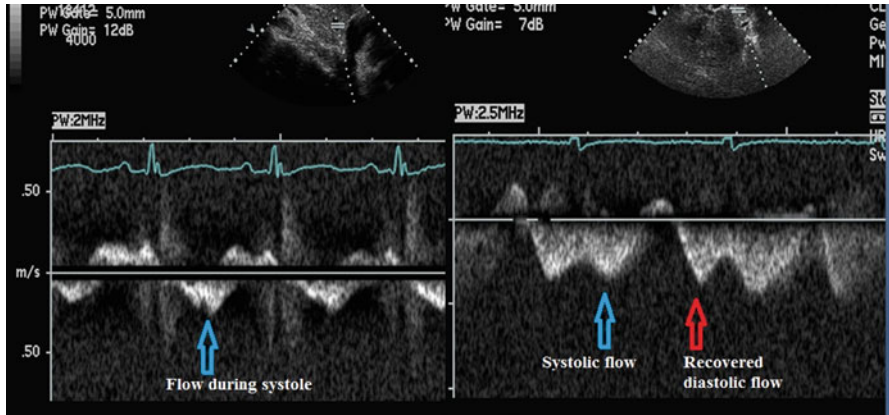
Fig. 9.7 Hepatic vein tracing showing flow to the atrium predominantly during systole and blunted return in diastole. After pericardiocentesis, flow to atrium during diastole is recovered

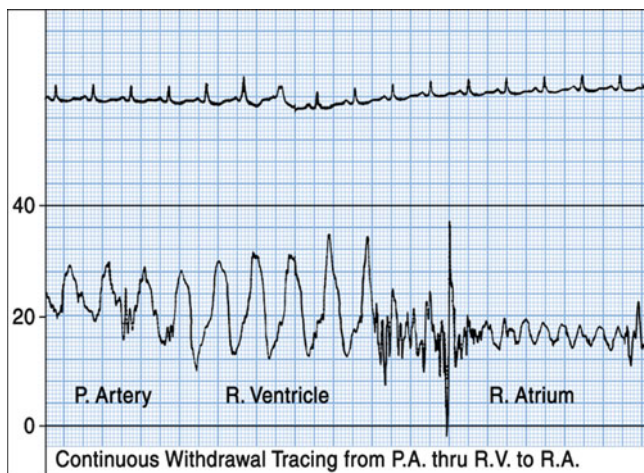## Pulmonary Artery Catheter Findings

While not routinely used for diagnosis, insertion of a pulmonary artery catheter may provide insight into the pathophysiology of tamponade as it helps determine the exact diagnosis and may demonstrate success of the pericardiocentesis.

The cardinal finding on PA catheterization during tamponade is diastolic equalization of pressures of the right atrium, RV diastolic pressure, pulmonary arterial diastolic pressure, and pulmonary capillary wedge pressure. A difference <5 mmHg suffices to establish this equivalency. This pressure is felt to be the passive pressure in the intracardiac chambers and equal to the intrapericardial pressure in severe tamponade (Fig. 9.8).

As mentioned above the other abnormal finding is blunted *y* descent following the *v* wave in the right atrial tracing. In tamponade, the *a* wave, *x* descent, and *v* wave remain unaffected as the contraction of the atrium, its relaxation, rapid filling remain unchanged. The *y* descent, which represents the rapid emptying of the atrium into the right ventricle, is blunted. This is due to the impaired RV relaxation in early diastole due to the effusion-related elevation in RV diastolic pressure. Thus, the rapid emptying of atrium to ventricle, i.e., the *y* descent is blunted (Fig. 9.9). This finding is readily reversed when pericardiocentesis is performed and the intrapericardial pressure is reduced. As the right ventricle is able to relax more efficiently, there is improved flow from the atrium to the ventricle hence a normalized *y* descent.

Distinction from Constriction

Intrathoracic pressures and changes during inspiration are readily transmitted to the pericardium and the intracardiac chambers in the case of a pericardial effusion with a relatively normal pericardium. In patients who have constrictive pericardium,

Fig. 9.8 Diastolic equalization of right atrial (mean), right ventricular end diastolic, pulmonary arterial diastolic pressures demonstrated as the PA catheter is pulled back from the pulmonary artery

Fig. 9.9 Right atrial pressure waveform with blunted *y* descent

these pressures are not transmitted, which as a result leads to differences in physical exam and invasive hemodynamic findings. Constriction is notable for lack of decreased right sided pressure with inspiration leading to the Kussmaul's sign, a finding not seen in tamponade. Furthermore, in patients with constriction, there is rapid ventricular filling in early diastole, which is recognized as a prominent *y*

descent with a "dip and plateau" appearance (square-root sign) on pulmonary artery catheterization. Such is not the case for tamponade where ventricular filling in early diastole is in fact impaired leading to a blunted *y* descent.

Computed Tomography and Magnetic Resonance Imaging

Advanced imaging such as CT and MRI may be useful to evaluate chronic pericardial effusions but should not be used in the setting of tamponade.

Pericardial effusion might be incidentally diagnosed with these imaging modalities. When this occurs, clinical assessment of the patient should be performed. In the event that there is a question of tamponade, hemodynamic assessment with a focused physical exam and echocardiography should not be delayed. MRI, in fact, can provide further hemodynamic information in these patients; however when tamponade is suspected, testing and treatment should not be delayed for performing this test.

Pericardial Tamponade in the Postsurgical Patient

While most effusions seen in clinical practice are circumferential, loculated effusions may be seen in postoperative settings and posttrauma. Chronic effusions though initially circumferential might organize into loculated effusions as well. In this setting, the aforementioned discussions about hemodynamics might not hold true. There may be focal compression of a cardiac chamber that might only be visible by 2D echocardiography. Doppler findings are also less helpful in postsurgical effusions; it is not uncommon to see exaggerated respiratory flow variation even in patients with small effusions. This may be due to increased respiratory effort secondary to pleural effusions or abnormal respiratory dynamics. When a case of focal tamponade is suspected, the clinical status of the patient and location of the effusion should be carefully assessed for further management.

Management

Patients who present with clinical tamponade should be treated emergently. While preparing the patient for removal of the pericardial fluid, intravenous administration of fluids to maintain adequate perfusion pressure is necessary. The administration of fluids improves the intracardiac filling and might stabilize the patient's hemodynamics by increasing the transmural pressure. Either a pericardiocentesis or a pericardial window might be necessary to resolve the compression of the cardiac chambers. Typically anterior, lateral, and apical effusions might be drained by a percutaneous approach; however surgical drainage is preferred for postoperative

patients, loculated, and posterior effusions. The risks and benefits of both approaches in conjunction with the clinical scenario should be taken into account.

Pearls of Assessment

- Pericardial tamponade is a clinical diagnosis.
- Tamponade occurs when transmural pressure is <0.
- When tamponade is in question, all measures for accurate diagnosis and stabilization of the patients should be undertaken.
- Echocardiography is the primary modality to establish the presence of an effusion and support the clinical diagnosis of tamponade.

  – Right atrial and/or ventricular wall inversion.
  – Increased respiratory variation in mitral and tricuspid flows.
  – IVC plethora—dilated IVC with blunted diastolic hepatic vein flow is the most sensitive early echocardiographic finding in tamponade. This may be absent in patients with low-pressure tamponade.

- Pulmonary artery catheterization might be useful to confirm the clinical suspicion in select cases.

Board Style Questions

1. A 72-year-old man presents with exertional dyspnea and work up including an echocardiogram shows a large pericardial effusion. Which of the following is more specific sign of tamponade?

   A. Systolic flow reversal in hepatic veins
   B. Early diastolic collapse of right atrium
   C. Diastolic collapse of right ventricle
   D. Hypotension in a patient with pericardial effusion
   E. Presence of a large pericardial effusion

2. A 67-year-old female with morbid obesity and rheumatoid arthritis presents with hypotension of unclear etiology. An echocardiogram is performed; however parasternal and apical windows are difficult to interpret due to body habitus, but there is a moderate circumferential pericardial effusion. Best Doppler hemodynamics are only obtained in the subcostal views. What's expected in the hepatic vein flow pattern in the setting of tamponade?

   A. Diminished forward flow during systole
   B. Diminished forward flow during diastole
   C. Flow reversal during systole
   D. Hepatic vein flow pattern is not expected to change with tamponade

E. Exaggerated forward flow during diastole

3. Which of the following is not a part of Beck's triad?

A. Muffled heart sounds
B. Hypotension
C. Elevated JVD
D. Kussmaul's sign

4. A 39-year-old female with a history of tuberculosis as a child presents with pitting peripheral edema and increased fatigue. Echocardiographic evaluation shows pericardial thickening with a moderate effusion. Her heart rate is 102 and blood pressure is 94/64. Which of the following can be helpful in distinguishing tamponade from constriction?

A. Presence of a pericardial effusion
B. Absence of Kussmaul's sign
C. Pericardial thickening seen on CT
D. Exaggerated flow across tricuspid valve during inspiration
E. None of the above

5. What's the initial treatment strategy in a hypotensive and tachycardic patient with a pericardial effusion if tamponade is suspected?

A. Pericardiocentesis
B. IV fluid infusion
C. Inotrope infusion
D. Consultation with cardiothoracic surgery for a pericardial window
E. Beta blocker administration

Answers to Board Style Questions

1. Correct Answer is C. Systolic flow reversal in hepatic veins is seen in severe tricuspid regurgitation. While options other than C can be seen in tamponade, they are not specific to tamponade.
2. Correct Answer is B. Diastolic forward flow is expected to be reduced in the hepatic veins due to elevated right ventricular pressures. Other options do not apply in tamponade.
3. Correct Answer is D. Beck's triad consists of muffled heart sounds, hypotension, and elevated neck veins.
4. Correct Answer is B. Other options can be seen with both conditions; however Kussmaul's sign is specific to constriction and is not seen with tamponade.
5. Correct Answer is B. An unstable patient in tamponade needs to be fluid repleted emergently in attempt to overcome the pericardial pressure. Further treatment modalities might include pericardiocentesis or a surgical window placement. Beta blockers are contraindicated in compensatory tachycardia.

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