

PERICARDIAL TAMPONADE

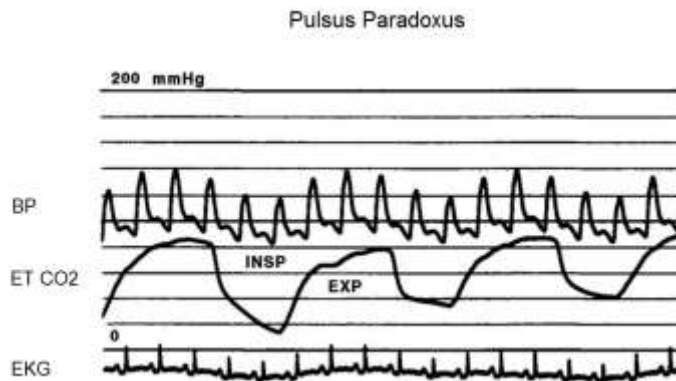
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Introduction

Tamponade is the accumulation of fluid in the pericardial sac that limits filling of the heart. It can occur in a variety of clinical conditions including malignant effusions, status post pericardiocentesis, idiopathic pericarditis, uremic pericarditis, and bleeding following cardiac surgery or cardiopulmonary bypass. Hemorrhagic tamponade is a surgical emergency and requires immediate decompression (pericardiocentesis, pericardial window, pericardiectomy) and mediastinal exploration to determine the bleeding source and stabilize hemodynamics.

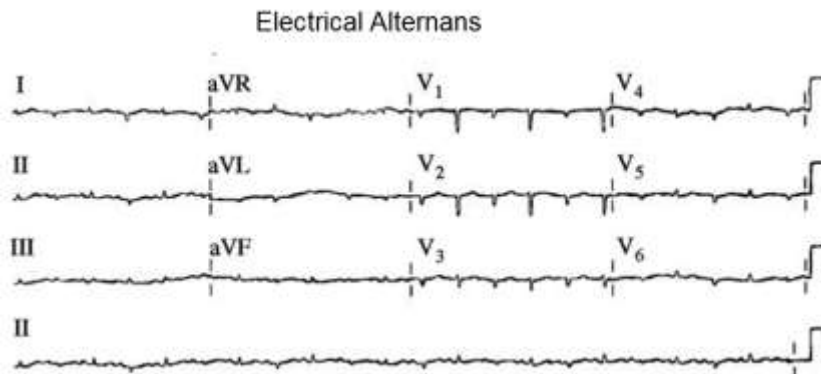
Clinical Presentation

The classic diagnostic triad includes **1) decreasing arterial pressure, 2) increasing venous pressure,** and **3) a small quiet heart.** Pulsus paradoxus may be present, defined as a fall in the SBP by more than 12 mmHg during inspiration caused by reduced LV stroke volume generated by increased filling of the right heart during inspiration (secondary to fixed volume of pericardial sac with fluid accumulation).



Kaplan, 2008.

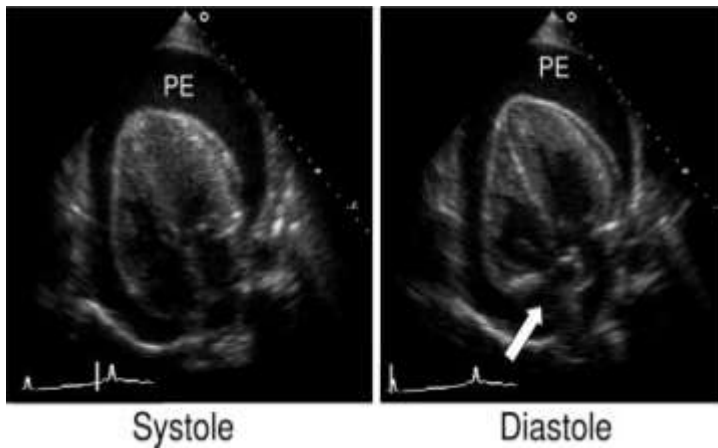
Pulsus paradoxus is not specific for tamponade, and may also be present in patients with COPD, RV infarction, and constrictive pericarditis. The ECG with tamponade is usually sinus rhythm, but findings may include a low-voltage QRS complex, electrical alternans, and T-wave abnormalities.



Kaplan, 2008.

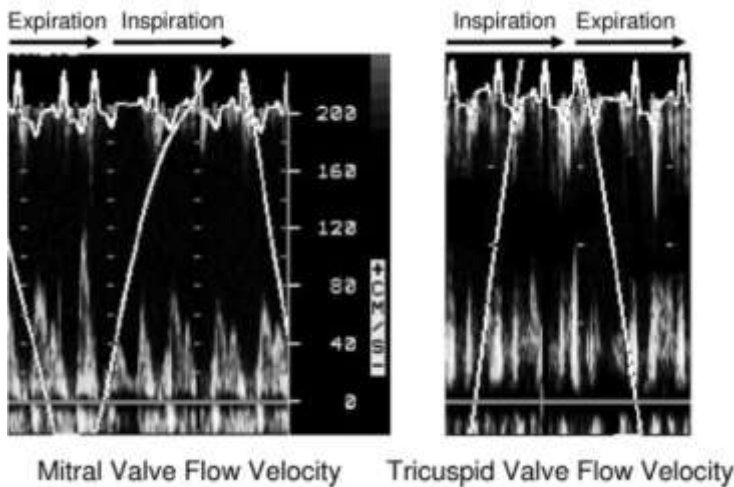
Echocardiography

While these clinical signs suggest the presence of tamponade, echocardiography is the most reliable noninvasive method to detect a pericardial effusion or exclude tamponade. Echocardiographic findings include exaggerated motion of the heart in the pericardial sac with atrial and/or ventricular collapse. Echocardiographic evidence of right atrial collapse at onset of systole and RV collapse in diastole are signs of hemodynamic compromise. Right atrial collapse is a sensitive sign of increased intrapericardial pressure. However, RV collapse is more specific for tamponade. Doppler flow studies have shown marked respiratory variation in transvalvular flow velocities, LV ejection, and LV isovolumetric times in patients with pericardial tamponade. (*Circulation*, 1997)



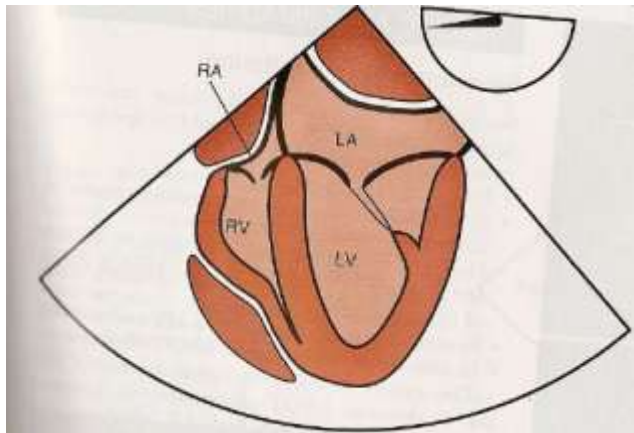
2D-echocardiogram in 4-chamber view from a patient with cardiac tamponade. There is a large pericardial effusion apparent as an echo-free space around the heart. In diastole, there is collapse of the right atrium (arrow).

Little WC, Freeman GL, 2006.



Doppler measurement of mitral valve and tricuspid flow velocities in a patient with cardiac tamponade. There is marked reciprocal respiratory variation: during inspiration, mitral valve flow velocity decreases, and tricuspid valve flow velocity increases.

Little WC, Freeman GL, 2006.



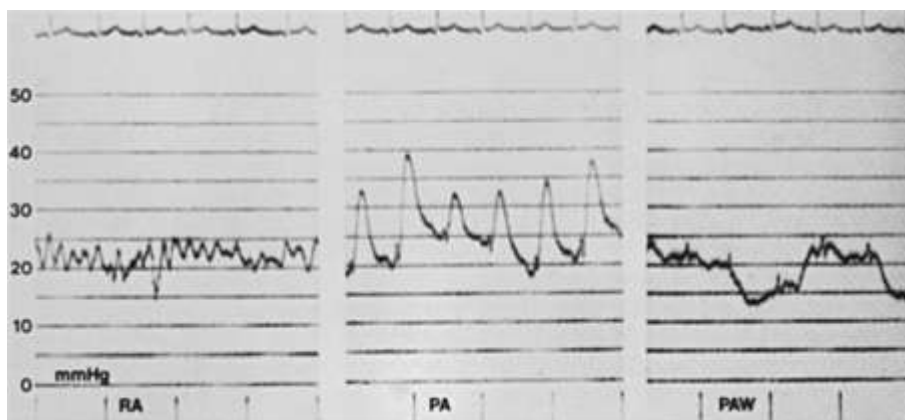
Three Common Sites of Pericardial Thrombus

- Behind the left atrium (oblique pericardial sinus)
- Posterolateral to the right ventricle
- Compressing the right atrium free wall

Sidebotham, 2011.

Pathophysiology

- Tamponade physiology exists when pericardial pressure exceeds that of one/more cardiac chambers
- Hemodynamic manifestations are mainly secondary to atrial compression, not ventricular collapse.
- Mild tamponade - diastolic filling is limited
 1. Reduction in stroke volume
 2. Stimulation of sympathetic reflexes
 3. Maintain cardiac output (CO) by increase in HR and contractility
- Rising right atrial pressure
 1. Reflex tachycardia and peripheral vasoconstriction
 2. CO begins to fall as pericardial fluid increases
 3. Equilibration of the right atrial, pulmonary artery diastolic, and pulmonary artery wedge pressures within 5 mmHg of one another
 4. Diastolic filling disappears, systemic blood pressure falls, resulting in coronary ischemia



Hemodynamic equilibration of right atrial pressure (RA), pulmonary artery diastolic pressure (PA), and pulmonary artery wedge pressure (PAW).

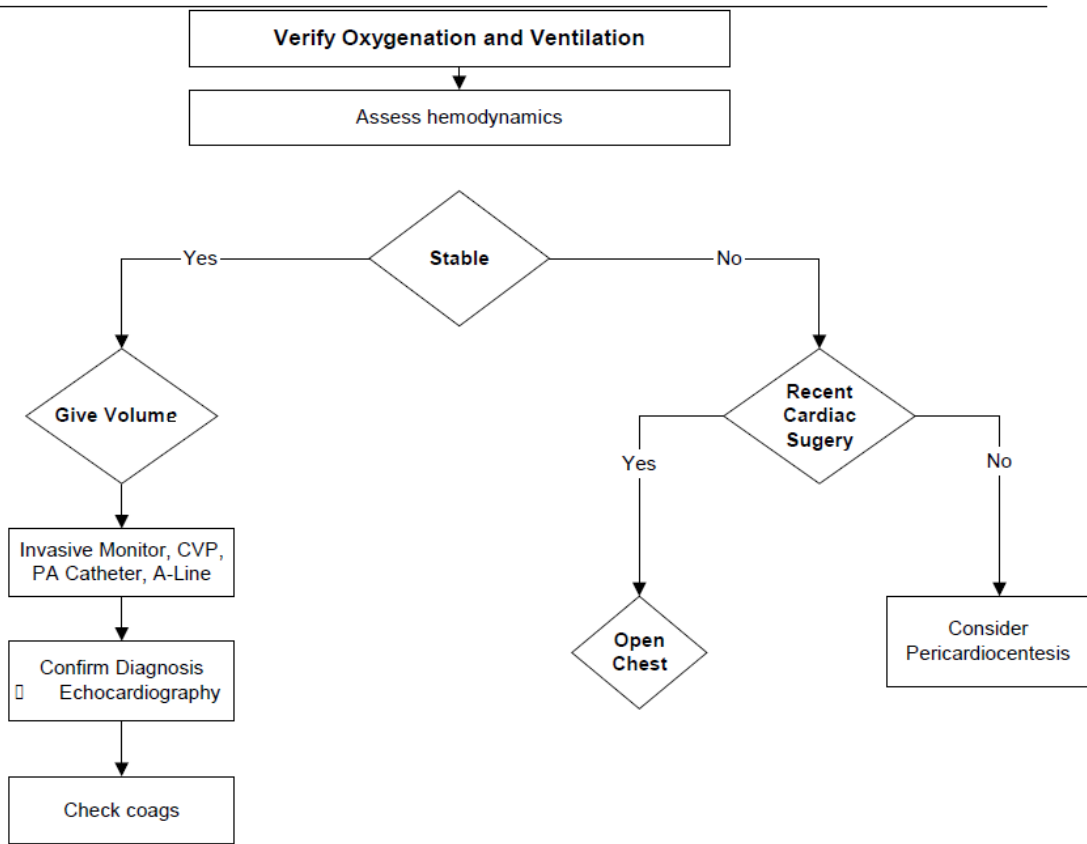
Kaplan, 2008

Organ System Manifestations

Cardiovascular	Systemic Hypotension Drop in coronary artery blood flow → myocardial ischemia/infarction Cardiovascular collapse
Nervous System	Hypotension → mental status changes
Genitourinary	Decreased renal perfusion → oliguria/anuria
Metabolic/Endocrine	Poor perfusion of vital organs and peripheral tissues can result in a metabolic lactic acidosis

Anesthesia Management

Monitoring	<ul style="list-style-type: none"> ▪ Pulse Oximetry ▪ ECG ▪ Intravenous central access, large-bore IVs ▪ Invasive arterial blood pressure monitoring
Resuscitation	<ul style="list-style-type: none"> ▪ Volume expansion to optimize preload ▪ Correct metabolic acidosis as it impairs myocardial function ▪ Catecholamine infusion to maintain CO (dobutamine, epinephrine, norepinephrine). This supports tissue oxygen delivery and delays the onset of lactic acidosis. <i>Caution with phenylephrine as stroke volume is fixed with tamponade physiology, and CO is severely impaired by bradycardia.</i>
Respiratory Support	<ul style="list-style-type: none"> ▪ Spontaneous respiration rather than positive-pressure ventilation supports CO more effectively until tamponade is relieved by decompression
Induction	<ul style="list-style-type: none"> ▪ Severe hypotension or cardiac arrest has followed induction of GA in patients with tamponade; caused by myocardial depression, sympatholysis, decreased venous return, bradycardia ▪ Pericardiocentesis or pericardiotomy via a subxiphoid incision may be performed under light sedation (Ketamine 0.5 mg/kg with 100% O₂) with local infiltration ▪ If intrapericardial injury is confirmed, GA may be induced after decompression of the pericardial space. Ketamine may be used as the induction agent as it minimizes myocardial depression, bradycardia, and reductions in venous return.



Adapted from O'Reilly M, 1999.

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