Cardiac Tamponade

Mark J. Baskerville

Risk

- Overall incidence: 2 pts per 10,000 population
- 2% incidence due to trauma in penetrating injuries
- Post-cardiac surgery: Greater incidence after valve replacement (0.6%), compared to CABG (0.2%)

Perioperative Risks

- Early diagnosis and prompt treatment are crucial in mitigating mortality and morbidity.
- Effusion can irritate myocardium, causing atrial and ventricular dysrhythmias.
- Regional cardiac tamponade is more common after cardiac surgery, where a localized effusion or hematoma compresses a single chamber.
- Sudden death from cardiac tamponade typically presents as a PEA arrest.

Worry About

- Sudden deterioration in hemodynamics
- Catastrophic cardiac collapse upon anesthetic induction and/or mechanical ventilation
- Uncontrolled bleeding
- End-organ injury from poor perfusion
- Rebound hypertension after release of tamponade

Overview

- Pericardial effusion is the anatomic diagnosis, whereas tamponade is the pathophysiologic diagnosis resulting in obstructive shock.
- Pericardial sac normally contains ~20 mL of fluid. It is the duration of time that an effusion accumulates that determines the likelihood of an acute tamponade.
- The pericardial pressure-volume curve is exponential in that once the effusion exceeds the limit of pericardial stretch, small increments of fluid create a steep rise in pressure.
- Transmural pressure = P_{in}(chamber) – P_{out}(pericardial), such that when the transmural pressure becomes negative, the chamber collapses.
- A compensatory sympathetic response leads to tachycardia and systemic vasoconstriction in order to maintain cardiac output and BP. Loss of endogenous sympathetic tone (e.g., induction of anesthesia) can lead to cardiovascular collapse.
- As the stroke volume becomes fixed, cardiac output becomes dependent on heart rate.
- Ventricular interdependence occurs when the septum shifts during the respiratory cycle due to the external constraint of the tightening pericardial sac. During inspiration, the septum shifts to the left, decreasing the LV stroke volume. During expiration, the septum shifts to the right, decreasing RV filling. However, the opposite occurs in positive-pressure ventilation.
  - Pulsus paradoxus: An exaggerated drop in systolic BP (>10 mm Hg) with spontaneous inspiration.
  - Beck's triad: Hypotension/JVD/muffled heart tones.
  - CXR: Cardiomegaly with globular heart.
  - ECG: Sinus tachycardia, low voltage, PR depression, diffuse ST elevations, and electrical alternans.
  - CVP tracing: The y descent is abolished due to an increase in intrapericardial pressure, preventing diastolic filling of the ventricles.
  - PA catheter: Equalization of diastolic pressures across chambers.
  - ECHO: RV can collapse in early diastole and RA can collapse in late diastole. LA collapse is rare, but highly specific for tamponade. One may see the heart swinging within effusion. IVC dilation without respiratory variation correlates with elevated right atrial pressure in tamponade. Doppler study may demonstrate substantial variation in transvalvular flow velocities with respiratory cycle.
- Be suspicious of localized clot in post–cardiac surgery that may not be evident on transthoracic ECHO.

Etiology

- Post cardiac surgery (valves > vessels)
- Thoracic aortic dissection
- Traumatic mediastinal injury
- Pacemaker lead perforation
- Malignant effusion (especially breast and lung)
- Mediastinal radiation
- ESRD (uremic effusion)
- Post MI (Dressler syndrome, ventricular wall rupture)
- Infectious (viral, fungal, TB)
- Myxoedema
- Collagen vascular disease (lupus, rheumatic disease)

Usual Treatment

- Pericardiocentesis
- Percutaneous balloon pericardiotomy
- Pericardial subxiphoid window
- Mediastinal exploration

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Hypotension Tachycardia Poor perfusion	Lethargy Pleuritic chest pain	Distant heart tones JVD Friction rub	ECG ECHO
RESP	Dyspnea	Orthopnea Poor exercise tolerance	Rales Cyanosis	CXR
RENAL	Oliguria Metabolic acidosis	Weight gain	Edema	Creatinine Lactic acid

Key References: Spodick DH: Acute cardiac tamponade, *N Engl J Med* 349:684–690, 2003; O'Connor CJ, Tuman KJ: The intraoperative management of patients with pericardial tamponade, *Anesthesiology Clin* 28:87–96, 2010.

Perioperative Implications

Preoperative Preparation

- Ensure adequate preload with cautious volume administration.
- Vasopressors and inotropes readily available.

Monitoring

- Routine ASA monitors.
- Arterial line
- CVP +/– PA catheters advantageous, but not mandatory
- TEE ideal

Airway

- Full stomach precautions with emergent procedures

Preinduction/Induction

- "Full, Fast, and Tight."
- Ketamine is an ideal induction agent, since it increases heart rate, contractility, and systemic vascular resistance, while maintaining spontaneous ventilation.
- Maintain spontaneous ventilation; consider inhalational induction.
- Decompression of tamponade via subxiphoid cardiac window under local anesthesia prior to induction of general anesthesia may be necessary.

Maintenance

- If mechanical ventilation is necessary, use low tidal volumes and minimize PEEP until tamponade is relieved.
- May need to quickly deepen anesthetic to overcome sympathetic surge after pericardium is decompressed.
- Coagulopathy and anemia should be treated promptly with transfusion of blood products.

Extubation

- Low threshold to keep pt intubated until stability confirmed

Adjuvants

- Vasodilators and beta blockers readily available to treat residual sympathetic surge.

Postoperative Period

- Vigilance for recurrent effusion/tamponade.
- Postdrainage pulm edema more common after large-volume drainage.
- ICU care generally warranted.

Anticipated Problems/Concerns

- Sudden cardiovascular collapse with transition to positive pressure ventilation.
- Rebound hypertension/tachycardia after relief of tamponade.
- Atrial fibrillation may necessitate emergent cardioversion.
- Myocardial ischemia or stunned myocardium.