

Risk

- Occurs rarely
- May be seen after open-heart surgery or PTCA.
- Blood and/or serous
- May be caused by infection: Viral, bacterial, or fungal
- May have a neoplastic etiology: Lymphoma, leukemia
- Can occur after acute MI, especially transmural
- Can be due to trauma
- Gender predominance: More common among men than women

Perioperative Risks

- If undiagnosed, tamponade leading to CV collapse is possible, with a low probability of determining the cause antemortem.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Tamponade limiting CO Hypotension Arrhythmias	Chest pain	Neck veins HR BP	TTE/TEE Equalization of all pressures in heart (cath) ECG
RESP	Decreased CO on institution of IPPB (mechanical ventilation)	Dyspnea Change in BP on institution of mechanical ventilation	Pulsus paradoxus: Large decrease in BP with inspiration Decreased O ₂ sat	Pulm artery, RA/LA pressures
METAB	Metabolic acidosis			ABG

Key References: Adler Y, Charron P, Imazio M, et al.: 2015 ESC Guidelines for the diagnosis and management of pericardial diseases, *Eur Heart J* 36(42):2921–2964, 2015; Grocott HP, Gulati H, Srinathan S, et al.: Anesthesia and the patient with pericardial disease, *Can J Anaesth* 58(10):952–966, 2011.

Perioperative Implications

Preoperative Preparation

- Appropriate monitoring before induction.
- Preoxygenation is not always effective.
- Hemodynamic goals: Full (fluid), fast (maintain or increase HR), tight (maintain or increase SVR).
- Consider draining transthoracically if hemodynamic compromise is severe.
- Consider prepping and draping prior to induction with surgeon ready.
- PPV may significantly worsen hypotension, resulting in shock and death.
- Consider placing external defibrillator patches prior to induction of anesthesia.

Monitoring

- Arterial line is indicated as BP may change suddenly; sampling of Hct for bleeding and acid-base status in state of low cardiac output is useful.

- Risk of CV collapse, especially with induction and institution of positive-pressure ventilation.

Worry About

- Hypovolemia
- Limited filling of cardiac chambers

Overview

- Effusion is found in the sac surrounding heart; if severe, it can restrict filling of the heart.
- Ventricular filling is depressed in both the RV and the LV.
- Fluid bolus and inotropes are beneficial but do little to improve CO.
- CO becomes more dependent on heart rate.

- For proper treatment, surgical drainage must be implemented.

Etiology

- Postsurgical and cath procedures
- During or after viral, bacterial, or fungal infection
- Postinflammatory process: Acute transmural inflammation, SLE, rheumatoid arthritis
- Uremia
- Neoplastic
- Trauma

Usual Treatment

- Drainage, either percutaneous or open.
- Medical management is generally ineffective.

- Consider a PA cath; useful for diagnosis and following surgical treatment. If pressures are not relieved by surgical drainage, question the original diagnosis.
- TEE is more useful than PA monitoring.

Induction/Maintenance

- Do not decrease preload or heart rate.
- Consider initiating invasive hemodynamic monitoring before induction.
- Monitor hemodynamics: If stable, slowly titrate small doses of propofol vs. etomidate.
- Consider inhalational induction if evidence of tamponade.
- Ketamine is advocated for new tamponade situations.
- Maintain spontaneous ventilation if possible; initiation of PPV may cause severe CV compromise due to decreased filling of RV and LV.

Treatment Approach

- For hemorrhage after open-heart surgery, reopen the sternum to explore for sources of bleeding, which is usually relieved by releasing the first few sutures.

- TTE-guided pericardiocentesis.
- Infections and/or neoplasia: Subxiphoid pericardial window.
- Insertion of a pericardioscope enables visualization of the pericardium and can serve to obtain biopsies.

Adjuvants

- Depend on etiology

Extubation

- Depending on etiology, consider awake extubation or postop mechanical ventilation.

Anticipated Problems/Concerns

- Many different causes, all with different sequelae.
- Hypotension on induction of anesthesia or positive pressure ventilation.

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Pericarditis, Constrictive

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Risk

- Dense changes in pericardium can be caused by scarring induced by a single episode of acute pericarditis or by prolonged exposure to an inflammatory process.
- 18% of pericardiectomies are attributed to previous cardiac surgery, which may explain the increase in number of cases of CP since the mid-1990s.

Perioperative Risks

- Heart failure, atrial arrhythmia, MI
- Abnormal drug metabolism secondary to liver failure
- Intraop major hemorrhage
- Postop respiratory failure

Worry About

- Hemodynamic instability due to limited filling or myocardial depression.
- When providing GA, be prepared for CPB.
- Right heart failure and volume overload.
- Differentiate from restrictive cardiomyopathy by various signs and symptoms as well as ECHO.

Overview

- CP is an inflammation of the pericardium, leading to impaired filling of the ventricles and reduced ventricular function.
- Restriction of the pericardium results in increased ventricular interdependence and a reciprocal relation between the filling of the left and right heart.

- During spontaneous ventilation, transtricuspid blood flow is increased, resulting in increased filling of the RV. This will lead the septum to shift to the left and to decrease LVEDV, with subsequent hypotension and pulsus paradoxus.
- During expiration, the septum is shifted to the right. Opposite changes take place during mechanical ventilation.
- Pts present with dyspnea, fatigue, orthopnea, and right heart failure with jugular venous congestion and chest pain, hepatomegaly, and ascites.
- Cardiac cath with hemodynamic assessment is considered the “gold standard.” However, comprehensive echocardiography with Doppler assessment is usually necessary to confirm CP and exclude restrictive cardiomyopathy.