

Anaesthesia for patients with pericardial disease

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Introduction

Patients with pericardial disease can present for various diagnostic procedures or therapeutic interventions. Providing anaesthesia care for these patients may be challenging because of their primary pericardial pathology and significant comorbidities affecting other organ systems. A thorough understanding of the pathophysiology of the pericardial disease state and its effect on haemodynamics is necessary before formulating a perioperative care plan.

Normal Anatomy and Physiology

The normal pericardium is situated in the anterior mediastinum surrounding the heart and proximal portions of the great vessels.¹⁻⁶ It is composed of 2 layers: an inner visceral layer which is thin, adherent to and continuous with the epicardium of the heart and an outer parietal layer which is thicker and more fibrous. Normal pericardial thickness is 1–2 mm.⁴ The parietal and visceral layers are separated by a small amount of serous fluid.¹⁻⁶ In non-disease states, approximately 15–50 ml of fluid is contained within the pericardial sac.⁵ This fluid is produced by visceral mesothelium cells and is drained from the pericardial space via the lymphatic system into the right side of the heart. The pericardial fluid minimises friction exerted on the epicardium from normal heart movements during the cardiac cycle and serves to balance hydrostatic pressures over the surface of the heart.

The reflections of the pericardium surrounding the great vessels form two potential spaces called the oblique and transverse sinuses which can be visualised on echocardiographic imaging.³⁻⁷ The oblique sinus forms posteriorly between the left atrium and pulmonary veins. The transverse sinus also forms behind the left atrium, behind the aorta and pulmonary artery.⁴ Both sinuses are common sites for blood to collect post cardiac surgery.

Although normal cardiovascular function can occur in the absence of the pericardium, it does bestow certain physiological benefits.¹⁻⁶

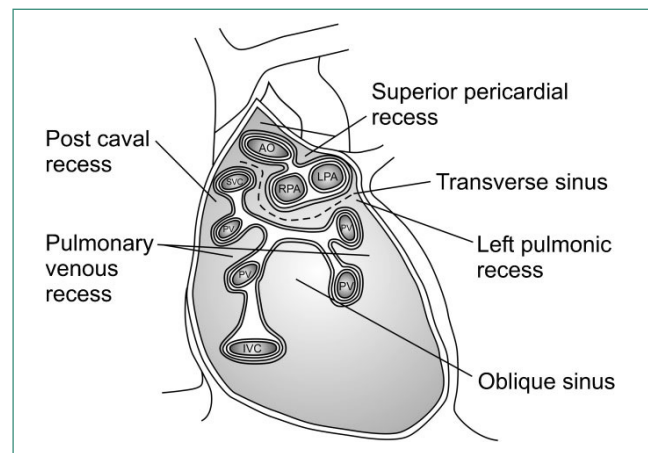


Figure 1. Line diagram demonstrating the posterior pericardial reflections which form the transverse and oblique sinuses and their associated recesses⁷

- Because of its relatively fixed ligamentous attachments, the pericardium stabilises the heart in its anatomic position and limits excessive movement within the chest cavity.
- The pressure exerted on the cardiac chambers by the pressure within the intrapericardial space prevents acute distention of the chambers and helps optimise atrial and ventricular coupling and filling.
- The pericardial sac serves as a physical barrier against the spread of infection or neoplasm within the mediastinum.
- Prostaglandins are secreted by mesothelial and endothelial cells of the pericardium that regulate autonomic cardiac reflexes, myocardial contractile function, and influence epicardial coronary artery tone.

Pathology

There are a number of pericardial pathologies which cause concern in the perioperative period.^{2,3,4,8}

- *Congenital defects*⁸
 - Usually associated with other cardiac, pulmonary and skeletal abnormalities
 - Often found at autopsy (incidence 1:10 000)

- Absence of the pericardium may be partial
 - left sided (70%) > right sided (17%)
 - left sided defects predispose to herniation of the heart which may become haemodynamically significant during induction of anaesthesia or cause prolonged ischaemia
 - right sided defects may cause significant compression of the vena cava.
- Total pericardial absence is rare
 - excessive cardiac motion and displacement predisposes to increased risk of traumatic aortic dissection.
- *Acute pericarditis*^{8,9,10}
 - May be self-limiting benign disease or the first presentation of underlying infectious or neoplastic disease.
 - Prudent to postpone elective surgery for diagnosis and specific treatment to be initiated.
 - Causes of acute pericarditis
 - Idiopathic
 - Infectious: viral (coxsackie B), bacterial, mycobacterial (tuberculosis)
 - Non-infectious: post cardiac surgery, trauma, malignancy, acute myocardial infarction, post-acute MI (Dressler's syndrome), uraemia (renal failure) or post radiation
 - Auto-immune: rheumatic fever, rheumatoid arthritis, systemic lupus erythematosus or drug induced (procainamide, dantrolene, heparin, warfarin).
 - Symptoms include a sharp left precordial or retrosternal chest pain which may be pleuritic in nature and varies with posture (decreased on sitting, increased on lying supine).
 - The pain may radiate to the trapezius ridge. This pain referral is due to involvement of the phrenic nerve which traverses the pericardium.
 - Often associated with prodromal symptoms of malaise, fever and generalised myalgia.
 - Tachycardia and tachypnoea are usually out of proportion to the low grade fever.
 - Triphasic friction rub corresponding to atrial systole, ventricular systole and rapid early filling during diastole may be present.
 - Most important is to differentiate between acute pericarditis and other causes:
 - differential diagnosis
 - acute coronary syndrome
 - aortic dissection
 - pulmonary embolism
 - May develop pericardial effusion +/- tamponade depending on the rate of fluid accumulation within the pericardial sac.
 - Increased risk of development of significant effusion and tamponade in the following patients:³
 - bacterial infections
 - fungal infections
 - malignancies
 - end stage renal disease
 - post cardiac surgery.
 - Special investigations:
 - ECG: sinus tachycardia, PR depression, diffuse concave upward ST segment elevation
 - ECHO: may show effusion and tamponade if present, other cardiac or para-cardiac disease.

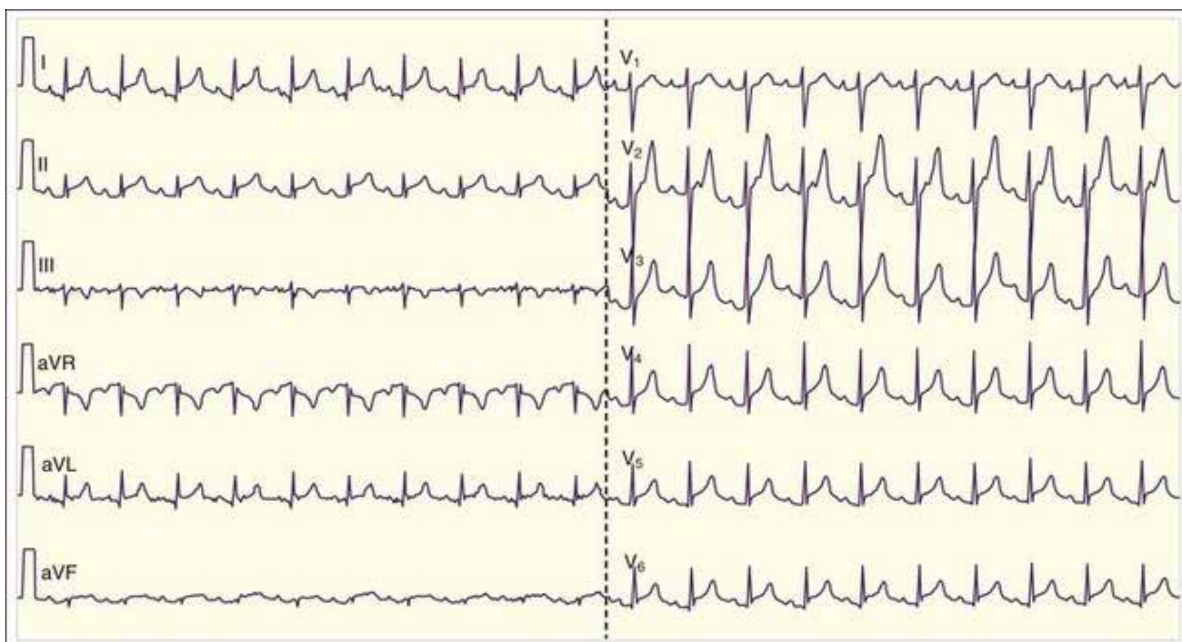


Figure 2: 12 lead ECG showing changes consistent with stage 1 of acute pericarditis⁹

- Treatment:
 - symptomatic: non-steroidal anti-inflammatory drugs, add colchicine to aid treatment and prevent recurrence¹¹
 - low dose corticosteroids if associated with autoimmune disease.¹²
- *Chronic pericarditis*⁸
 - Acute pericarditis may progress to chronic and recurrent/relapsing disease.
 - Perioperative management will depend on the haemodynamic influence of the disease process.
 - Differentiate between chronic pericarditis (ongoing inflammation, pain and fever), relapsing disease (periods of being symptom free) and chronic pericardial effusion.
 - Symptomatic treatment of pericarditis attacks with NSAIDs, colchicine and corticosteroids should be carried out before any elective surgery.
 - Pericardiectomy is only necessary in patients with frequent and severe symptoms that are unresponsive to medical therapy.
 - Moderate to large effusions, determined at echocardiography as being > 10 mm separation of the pericardial layers during diastole, should be drained before any elective surgery.
- *Pericardial effusions and Cardiac Tamponade*^{2-9,13}
 - Occurs when there is excessive fluid accumulation in the pericardial space.
 - The effusion may be transudative, exudative, haemorrhagic or purulent depending on the cause.
 - Causes for pericardial effusion are similar to those for pericarditis or may be caused by pericarditis itself.
 - Idiopathic, iatrogenic, trauma, malignancy, end stage renal disease, autoimmune, infectious (TB), aortic dissection and radiation
 - Patients most at risk for the development of tamponade are:
 - iatrogenic (post cardiac surgery or percutaneous coronary intervention, insertion of pacemaker, percutaneous valve repair)
 - Chest trauma
 - Malignancy
 - End stage renal disease^{3,14,15}
 - Incidence of pericarditis 2–21% and effusion with tamponade 14–56%
 - patients not on haemodialysis develop uraemic pericarditis and effusions that generally respond to aggressive haemodialysis
 - patients on haemodialysis develop dialysis pericarditis and effusions that more frequently require drainage via pericardial window
 - because of the chronicity of the disease process and the associated hypertension, significant haemodynamic compromise is rare in this group of patients and general anaesthesia for

pericardial window and drainage is usually well tolerated.³

- *Pathophysiology:*
 - The haemodynamic effects of the effusion and development of tamponade are dependent on^{5,13}:
 - The rate of fluid accumulation within the pericardial space.
 - The total volume of fluid that accumulates.
 - Type of fluid
 - exudative fluid may become fibrinous in nature
 - haemorrhagic fluid may contain clots causing isolated chamber compression
 - chronic effusions may become fibrotic.
 - The compliance of the pericardial tissues (decreased in mesothelioma and scarring from previous cardiac surgery).
- *Pericardial compliance:*
 - Because the pericardium is relatively stiff, it has a limited reserve volume
 - Small but rapidly accumulating effusions quickly exceed the compliance of the parietal pericardium.
 - once the limit of pericardial stretch is exceeded, even small increases in volume cause a steep increase in pericardial pressure causing external compression of the cardiac chambers (tamponade).
 - Chronic accumulation over a prolonged period allows for pericardial stretch and compensatory mechanisms, meaning that the intrapericardial pressure remains low despite a large amount of fluid within the space
 - even with additive amounts of fluid accumulation, the pericardial compliance curve remains less steep than in the acute setting, meaning that intrapericardial pressures rise more slowly relative to the volume of fluid within the space.

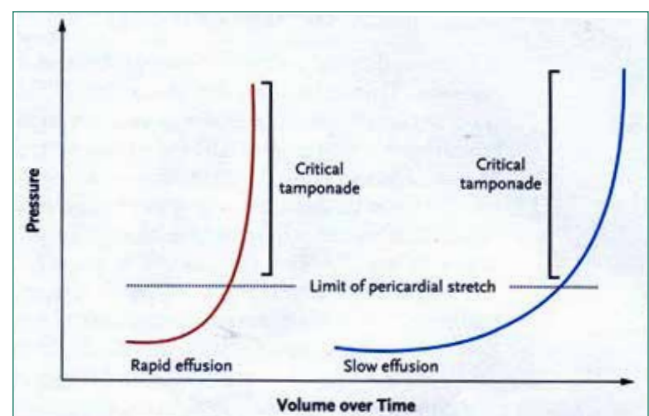


Figure 3: Cardiac Tamponade¹³ Pericardial pressure–volume (or strain–stress) curves are shown in which the volume increases slowly or rapidly over time. In the left-hand panel, rapidly increasing pericardial fluid first reaches the limit of the pericardial reserve volume (the initial flat segment) and then quickly exceeds the limit of parietal pericardial stretch, causing a steep rise in pressure, which becomes even steeper as smaller increments in fluid cause a disproportionate increase in the pericardial pressure. In the right-hand panel, a slower rate of pericardial filling takes longer to exceed the limit of pericardial stretch, because there is more time for the pericardium to stretch and for compensatory mechanisms to become activated.

- Once intrapericardial and intracardiac pressures increase beyond a certain limit, cardiac chamber filling and preload is reduced which causes a drop in stroke volume and cardiac output.
- This drop in cardiac output causes a reduction in organ perfusion which triggers compensatory mechanisms including activation of the sympathetic nervous system and the renin-angiotensin-aldosterone axis
 - the resultant tachycardia, peripheral vasoconstriction and fluid retention is an attempt to maintain systemic blood pressure, cardiac output and organ perfusion.
- **Tamponade**^{2,3,4,5,8,9,13,16,17,18}
 - Occurs when cardiac chambers exhibit compression due to an increase in intra-pericardial pressures.
 - Impaired filling of the cardiac chambers occurs during diastole (diastolic dysfunction).
 - Pericardial pressures are elevated throughout the cardiac cycle and cause compression of chambers for the duration of diastole
 - venous return pattern becomes unimodal and is confined to systole in severe tamponade.
 - Cardiac chamber filling pressures are dependent on the myocardial transmural pressure gradient
 - **transmural pressure = intrapericardial pressure - intracardiac pressure**
 - The intracardiac pressures are different for each chamber with right atrial pressures being the lowest to facilitate systemic venous return.
 - Based on Ohm's law ($V=IR$), in order to ensure filling and forward flow in a normal series circulation, there must be a pressure gradient (P_1-P_2) between the systemic venous return circulation and the aorta.

systemic venous return pressure ► right atrial pressure ► right ventricular end diastolic pressure ► right ventricular systolic pressure ► pulmonary capillary wedge pressure ► pulmonary venous return pressure ► left atrial pressure ► left ventricular end diastolic pressure ► left ventricular systolic pressure ► aorta

- As the intrapericardial pressure increases, extrinsic compression of the cardiac chambers occurs
 - compression causes the intra-cardiac chamber pressures to increase and the transmural pressures begin to equalise
 - the right sided filling (diastolic) pressures are lower than the left and are the first to be equalled and then exceeded by intra-pericardial pressures
 - the right atrium and ventricle are compressed, chamber filling is compromised and the right atrial pressure (CVP) increases to try and maintain right ventricular filling
 - this causes a significant decrease in the venous return to the right heart as right atrial pressures exceed systemic venous return pressures
 - impaired right ventricular filling leads to an underloaded ventricle which then functions at the

- lower end of the Frank-Starling curve resulting in a drastically reduced right ventricular stroke volume
- reductions in right ventricular stroke volume translate into decreased flow through the pulmonary circulation with decreased pulmonary venous return to the left atrium
- with continued increases in the intra-pericardial pressures, equalisation of left sided pressures will transpire
- this results in massive reductions in and eventual cessation of diastolic filling and forward flow of blood through the heart with dramatically reduced cardiac output
- a cycle of exhaustive physiological compensation follows with eventual cardiac arrest manifested as pulseless electrical activity
- although reductions in coronary blood flow do occur with the drop in cardiac output and aortic diastolic pressures, this is coupled with limited cardiac work and ischaemia is avoided
- *equalisation of transmural pressures can be a dynamic process influenced by extra-cardiac factors such as induction of general anaesthesia causing vasodilation, direct myocardial depression of anaesthetic agents and positive pressure ventilation*
 - *Patients at risk for the development of tamponade should have echocardiographic studies to confirm their preoperative haemodynamic state.*
 - **In the event of potential or imminent tamponade, all elective procedures should be postponed and the pericardium drained to relieve the increased intrapericardial pressures.**

- **Clinical signs and diagnosis:**

- Signs and symptoms
 - Dyspnoea, orthopnoea, diaphoresis, chest pain and tachycardia are all non-specific signs of pericardial effusion and tamponade
 - Muffled heart sounds, raised jugular venous pressure and hypotension are the features classically described as Beck's triad used in the diagnosis
 - *Pulsus paradoxus*
 - commonly found in tamponade
 - refers to the exaggeration of a normal physiological variation in systolic blood pressure during negative pressure ventilation
 - negative intrathoracic pressure generated on inspiration is transmitted to the heart and increases the transmural pressure gradient
 - this augments right atrial and ventricular filling during diastole as demonstrated by increased blood flow velocity across the tricuspid valve during inspiration on echocardiography¹⁸
 - the augmentation in right ventricular filling causes the intraventricular septum to shift to the

- left which transiently impacts on left ventricular filling ► interventricular dependence
- with normal pericardial pressures and compliance, compensation for this slight shift is maintained leading to minimal reductions in left ventricular end diastolic volume (stroke volume)
 - this results in a small decrease in systolic blood pressure during inspiration of not more than 10 mmHg
 - the augmented right ventricular stroke volume during inspiration translates into increased pulmonary venous return to the left atrium after several cardiac cycles and an increase in left ventricular stroke volume and systolic blood pressure is seen during expiration
 - in tamponade, this normal systolic pressure variation with respiration is exaggerated
 - with the decrease in pericardial compliance, the normal pericardial compensation for the left heart shift is lost
 - as the intraventricular septum shifts to the left, the left heart cannot expand and left ventricular filling and flow through the left ventricular outflow tract are affected to a greater degree ► *enhanced ventricular interdependence*
 - this translates into a decrease of systolic blood pressure of > 10 mmHg on inspiration.
 - Pulsus paradoxus is not very sensitive or specific for cardiac tamponade and may occur with other conditions or may be absent even when tamponade is present
 - Differential diagnosis
 - severe chronic obstructive pulmonary disease
 - asthma or severe bronchospasm
 - hypovolaemia
 - congestive cardiac failure
 - obesity
 - Conditions when pulsus is absent in the presence of tamponade
 - severe aortic regurgitation (left ventricular filling is maintained due to back flow from the aorta)
 - localised effusions/clot (compresses only a certain portion of the heart)
 - atrial septal defect (pressure effect on the right atrium is balanced by intracardiac shunt)
 - severe right ventricular hypertrophy and pulmonary hypertension (pressure effects from increased intrapericardial pressure on right heart less pronounced)
 - Pulsus paradoxus will be reversed in patients receiving *positive pressure ventilation* with increased systolic blood pressure noted on inspiration and a fall in systolic blood pressure on expiration⁵
 - this may be interpreted as systolic pressure variation in positively ventilated patients which may be interpreted as a sign of fluid responsiveness
 - positive pressure ventilation has 3 main effects on cardiac physiology
 - positive pressure ventilation increases intrathoracic pressure (alveolar pressures increase > pleural pressures) ► compression of pulmonary capillaries ► increased right ventricular afterload
 - compression of pulmonary capillaries also squeezes blood from the pulmonary bed into the left atrium augmenting left ventricular preload



Figure 4: Electrical Alternans. Electrocardiogram from a patient with pericardial tamponade demonstrating electrical alternans, a phenomenon caused by alternating voltage with each cardiac cycle due to anterior-posterior swinging of the heart in the non-constrained, fluid-filled pericardial sac. This process is observed with alternating heartbeats, such that the heart is physically closer to the chest wall with every other beat, resulting in taller r-waves.³

- positive inspiratory pressure augments left ventricular systolic function
- the augmentation of left ventricular stroke volume and systolic function during inspiration leads to increased systolic blood pressure
- pulmonary transit time is approximately 2 seconds and the drop in right heart cardiac output translates into a drop in left ventricular cardiac output and systolic blood pressure during expiration.
- **As mentioned above, the combination of decreased venous return (preload) to the right heart with induction of anaesthesia plus the increased right ventricular afterload effects of positive pressure ventilation and PEEP can severely compromise cardiac function in patients with tamponade.**
- **Special investigations**^{2,3,4,5,6,8,9,19}
 - Chest X-ray: enlarged globular bottle shaped cardiac shadow, widened mediastinum, right costo-phrenic angle < 90 degrees, clear lung fluids
 - May have an associated pleural effusion that requires concurrent drainage and management.
 - ECG³: sinus tachycardia, low QRS and T wave voltages, PR segment depression, non-specific ST-T wave changes, bundle branch block, electrical alternans
 - reflects excessive swinging movement of the heart within the large effusion.
 - Echocardiography^{2,16,17,18}:
 - diagnostic, sensitive and specific
 - grading system for size of the effusion based on echo free space in diastole
 - small < 10 mm
 - moderate 10–20 mm
 - large > 20 mm
 - RA collapse and invagination for > 30% of the cardiac cycle on ECHO is 100% sensitive for the diagnosis of tamponade¹⁶
 - distention of the IVC reflecting increased right atrial pressure

- increased respiratory variations in tricuspid and mitral transvalvular velocities and intra-ventricular septal shift (reflected clinically as pulsus paradoxus)¹⁶
- localised or loculated effusions showing isolated chamber compression, e.g. clot post cardiac surgery.

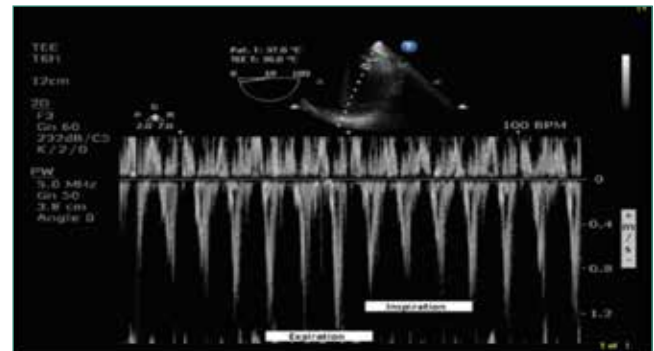


Figure 6: Pulsed wave doppler tracing using mid-oesophageal 4 chamber view on trans-oesophageal echocardiography to view trans-mitral flow velocities in a spontaneously breathing patient with tamponade. Note the reduction in velocities during inspiration highlighting pulsus paradoxus²

- Cardiac catheterisation: usually not performed as ECHO is less invasive and diagnostic, blunted Y descent^{19,20}
 - descent on CVP tracing after the V wave
 - indicates opening of the tricuspid valve and reflects early diastolic filling of the RV
 - attenuation of the Y descent reflects rapid equalisation of pressures between the right atrium and ventricle which leads to ineffective filling of the RV during early diastole in tamponade.²⁰

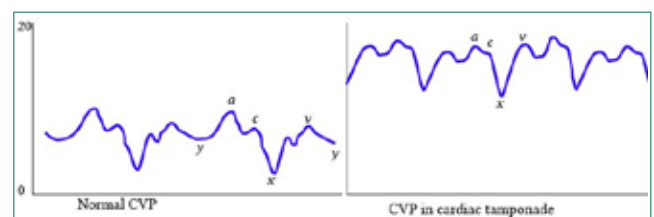


Figure 7: CVP tracing in cardiac tamponade reflecting attenuation of the Y descent. Anaesthetic management for effusions and tamponade^{2,3,5}

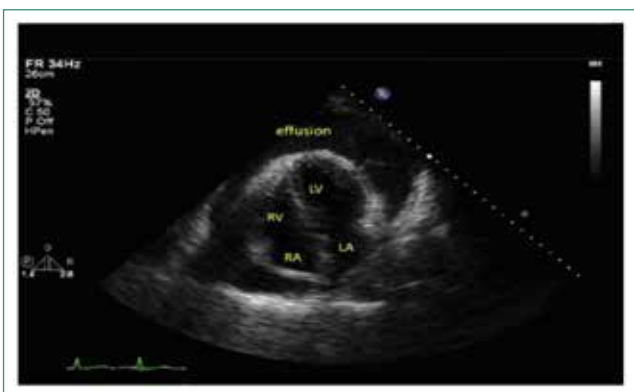


Figure 5: Trans-thoracic echocardiographic apical 4 chamber view showing large pericardial effusion. No evidence of tamponade²

- Accumulation of fluid in the pericardial space is the primary problem therefore definitive drainage and relief of raised intra-pericardial pressure is the required treatment.
- Type of drainage procedure will depend on the aetiology of the pericardial effusion and the clinical condition of the patient.
- A multi-disciplinary team approach needs to be taken.
- A thorough preoperative work up should be done if time allows
 - clinical history and examination should be carried out in every patient focusing on pertinent physiological symptoms and signs to identify the cause and degree of severity of the pathology.

- Patients fall broadly into 2 categories:
 - Pre-tamponade and haemodynamically stable
 - Tamponade is present with haemodynamic instability
 - the presence of orthopnoea and pulsus paradoxus are signs of a severely compromised patient
- If there is haemodynamic compromise, the safest and quickest drainage option needs to be selected
- Haemodynamic goals pre-drainage should follow the “A,B,C” approach bearing in mind the pathophysiology of tamponade as discussed above.
- Positive pressure ventilation should be avoided but if required, peak airway pressures and PEEP should be minimised to avoid precipitation of cardiovascular collapse.
- Large bore peripheral access is needed for fluid and blood product administration.
- Invasive arterial and central venous lines are needed for monitoring but should not delay drainage for patients in extremis.
- Optimisation of preload is advised preoperatively as hypovolaemia will worsen tamponade
 - Dynamic measures of fluid responsiveness should be used to guide fluid therapy
 - A single, individualised fluid challenge may be beneficial and outcome based measures should be used to assess response, e.g. patient reports relief of symptoms or markers of end organ perfusion improve
 - Caution needs to be exercised as too much fluid will worsen the ventricular interdependence and further compromise cardiac output
 - Volume loading pre-drainage may exacerbate volume overload post-drainage
 - Haemodynamic support with a vasopressor may be more appropriate.
- Inotropes and pressors need to be individualised in every case
 - Haemodynamic goals in tamponade
 - increase cardiac output
 - maintain heart rate
 - maintain afterload
 - decrease right atrial pressures
 - Although inotropes may facilitate reaching the above haemodynamic goals, inotropes also increase myocardial oxygen demand, decrease diastolic filling time and potentially worsen ischaemia
 - Noradrenaline with predominant pressor effects increases mean arterial pressures with minimal myocardial stimulation and no change in cardiac index which may be more appropriate choice.
- Types of drainage procedures:
 - Needle pericardiocentesis
 - percutaneous placement of catheter into the pericardial sac to facilitate external drainage of the effusion
 - use a Seldinger technique utilising specific anatomical landmarks and echo or fluoroscopic guidance to insert the catheter
 - can be done in an awake patient with supplemental local anaesthetic
 - diagnostic and therapeutic as drained fluid can be sent for testing
 - complications include puncture and rupture of myocardium or coronary vessels, arrhythmias, myocardial infarction, subsequent infection or damage to surrounding structures (stomach, liver and lung).
 - Surgical procedure: pericardial window
 - Sternotomy and possible cardio-pulmonary bypass
 - Anterolateral thoracotomy
 - Video assisted thoracoscopy (VATS)
 - surgical drainage allows for the formation of a pleuro-pericardial window which allows ongoing drainage of the effusion and decreases risk of recurrent tamponade.
 - Subxiphoid approach
 - small subxiphoid incision is made to relieve pressure and directly visualise parietal pericardium
 - advantages: effective, simple and can be done under local anaesthesia if needed for the patient nearing cardiovascular collapse.
- *Anaesthetic techniques:*
 - All patients require adequate peripheral access and standard ASA monitoring prior to any anaesthetic agent being administered
 - Depends on the clinical condition and haemodynamic stability of the patient, any concomitant co-morbidities, the aetiology of the effusion and the procedure being performed
 - Local anaesthetic infiltration with supplemental sedation using ketamine, midazolam or fentanyl may be sufficient for pericardiocentesis and subxiphoid windows in the less stable patients who are co-operative
 - If general anaesthetic is required, haemodynamic goals remain the same:
 - maintain and augment preload
 - maintain afterload
 - maintain and augment contractility
 - maintain heart rate
 - maintain sinus rhythm to preserve atrial contribution to ventricular filling
- Induction
 - Avoid respiratory depressant drugs and positive pressure ventilation if possible
 - spontaneous respiration with a volatile agent is ideal if tolerated
 - use infusion of vasopressor to maintain blood pressures while ensuring adequate depth of anaesthesia with volatile agent before manipulation of the airway
 - IV induction can be used for stable patients with no evidence of tamponade

- surgical preparation and draping to facilitate emergency drainage is advisable if IV induction is to be used
- ketamine and etomidate are recommended as they will have the least vasodilatory effects
- If endotracheal intubation and positive pressure ventilation is required
 - use the lowest possible inspiratory pressures and PEEP to maintain minute volume and oxygenation
 - choice of ETT will depend on the procedure: one lung ventilation may be needed to facilitate surgery in thoracotomy and VATS
 - the time taken to insert a double lumen ETT may be detrimental in an unstable patient
 - placing a single lumen ETT with a bronchial blocker may be advisable
 - if the patient is very unstable, performing a subxiphoid window initially to relieve the tamponade and then performing the general anaesthetic is another option
- Inotropic and vasopressor support should be anticipated and used as needed to maintain haemodynamic goals at induction of anaesthesia.
- Maintenance
 - IV opioids, propofol, ketamine and volatile agents can all be used if they are tolerated
 - muscle relaxants should only be used once the patient can tolerate positive pressure ventilation
 - continuous infusions of inotropes and pressors may be needed and require continuation depending on the case.
- Intra-operative arrhythmias are common because of surgical handling of pericardium and heart and should be anticipated
 - defibrillator and anti-arrhythmic drugs should be immediately available.
- Because intrinsic myocardial function is preserved in pericardial effusion and tamponade, once the tamponade is relieved, there is usually a dramatic improvement in haemodynamics and the patients do well post-operatively.
- Hypertension may present after drainage, this should be anticipated and controlled with IV agents especially in patients where increases in blood pressure will worsen intraoperative bleeding or outcome, e.g. chest trauma, coronary or myocardial perforation or aortic dissection.
- Occasionally, following drainage of large, chronic effusions, patients develop pulmonary oedema and global LV dysfunction
 - the cause is unknown but may be related to sudden increase in preload and ventricular filling with an abrupt increase in systolic wall stress, myocardial stunning, volume overload and extravasation of fluid into the alveoli and pulmonary interstitium
- Postoperative
 - Patients should be transferred to a high dependency unit or ICU
 - Ongoing care is necessary to monitor for recurrence of tamponade, ongoing bleeding and continuation of inotropic and vasopressor support
 - Extubation will depend on the patient's pre-op condition and intraoperative course.

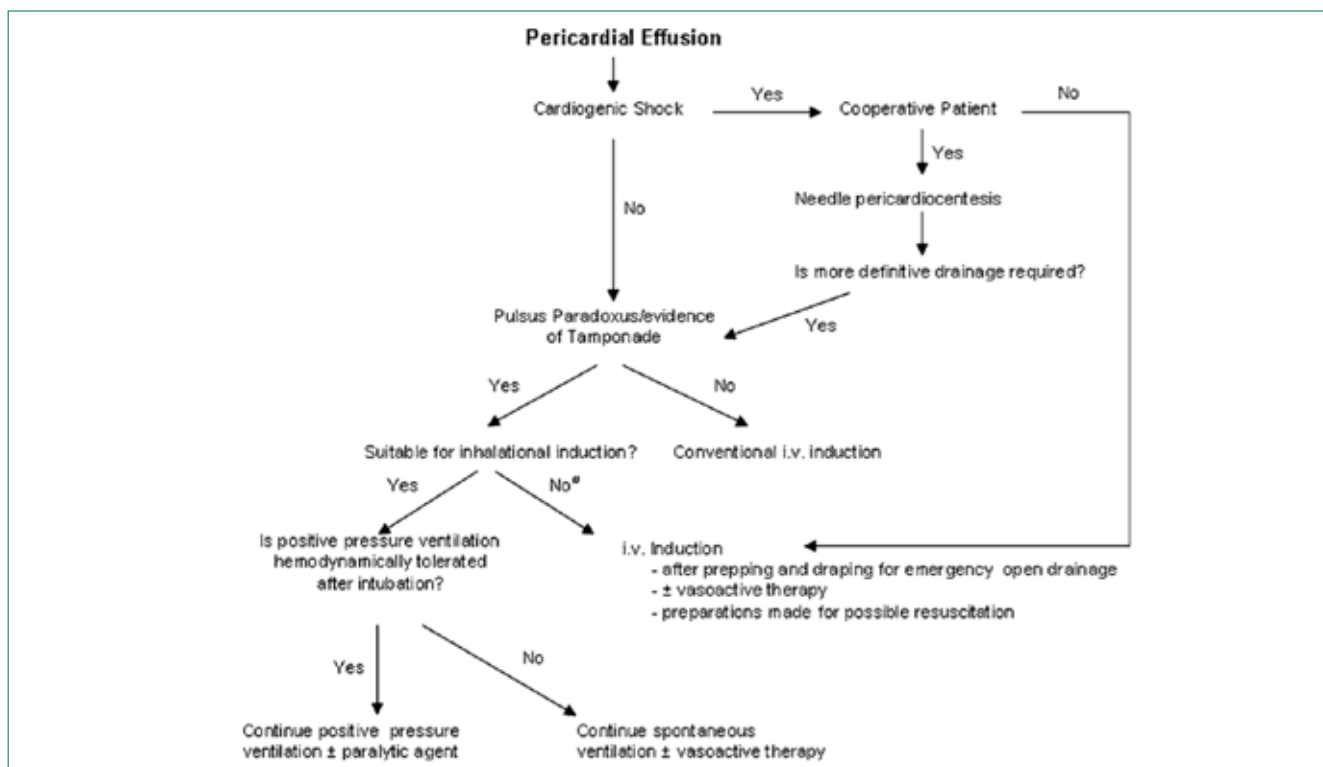


Figure 8: Proposed management strategy for patients with varying severity of pericardial effusion and cardiac tamponade. Conditions which may preclude inhalational induction include: aspiration risk, significant obesity, severe orthopnoea or an uncooperative patient²

- *Constrictive pericarditis (CP)*^{2,6,8,9,23,24,25,26,27}:
 - Chronic inflammation of the pericardium causes it to thicken, become non-compliant and rigid.
 - Restriction of cardiac chamber expansion during diastole causes impedance of cardiac filling and diastolic dysfunction.
 - The usual form is constriction without effusion but effusive-constrictive forms can occur.
 - Causes are similar to those for acute and chronic pericarditis:
 - Idiopathic, infectious, post cardiac surgery, following radiation to the mediastinum, trauma or auto-immune diseases.
- *Pathophysiology*^{2,8,23,26,27}:
 - Normally the pericardium can accommodate changes in cardiac volume.
 - A thickened, calcified and rigid pericardium encases the heart and limits cardiac chamber expansion
 - early diastolic filling is initially not affected
 - the atrial contribution to ventricular filling is mostly impeded during mid- to late diastole.
 - Diastolic filling is severely impaired and the total blood volume within the heart remains relatively constant during diastole leading to a fixed stroke volume state.
 - Any increases in tissue perfusion demands must be met by an increase in heart rate.
 - One of the most important features of CP is that changes in intrathoracic pressures during the respiratory cycle are not transmitted to the heart
 - this means that the normal augmentation of venous return to the right heart during inspiration does not occur
 - lack of inspiratory decline in the jugular venous pressure leads rather to an increase in CVP with inspiration = Kussmaul's sign (see below).
 - Respiratory variation in systolic blood pressures may still be seen as the pulmonary veins lie outside of the pericardium and thus changes in intrathoracic pressures will still cause changes in pulmonary vein flow
 - during negative pressure ventilation, inspiration will cause the pressure gradient between the pulmonary veins and the left ventricle to decrease resulting in a decrease in LV filling and a small decrease in systolic blood pressure
 - this effect, however is usually not enough to cause pulsus paradoxus.
 - There is a dramatic increase in ventricular interdependence in constrictive pericarditis²⁵
 - pressure changes in one ventricle will be transferred to the other which explains the observed equilibration of diastolic ventricular pressures
 - this increase in ventricular coupling means that there is little, if any, trans-septal pressure gradient during diastole
- abnormal shifting of the intra-ventricular septum has been observed in some studies but because the ventricles are operating at much higher baseline diastolic pressures, small changes in pressures caused by septal shift have very little effect on ventricular filling.
- Chronic constriction of the myocardium can lead to damage of the underlying muscle tissue
 - long-standing constriction causes the ventricles to operate at the lower end of the Frank Starling curve
 - myocardial atrophy may present with continued diastolic dysfunction coupled with significant systolic dysfunction even after successful pericardiectomy is performed and the myocardium is released
 - often these patients will require inotropic support in the peri-operative setting.
- *Clinical signs and diagnosis:*
 - Signs and symptoms:
 - Can be non-specific and similar to right ventricular failure
 - tachycardia is predominant sign because of the fixed stroke volume state
 - fluid overload ranging from peripheral oedema to anasarca due to venous hypertension, worsened by protein losing enteropathy in decompensated disease
 - decreased cardiac output with shortness of breath and fatigability
 - Ascites, hepatomegaly, pleural effusion and peripheral oedema can be misdiagnosed as chronic liver disease
 - pericardial knock
 - high pitched sound occurring in early diastole before the third heart sound
 - indicates rapid inflow during early diastole and then the abrupt cessation of ventricular filling at the end of the early diastole due to pericardial constriction
 - may have associated pleural effusions
 - may have cachexia associated (chronic disease process).
 - *Kussmaul's sign*
 - increase in jugular venous pressure on inspiration
 - increase in right atrial preload mediated by an increase in intra-abdominal pressure during inspiration independent of intrathoracic pressure changes
 - increase in preload cannot be accommodated because of the fixed pericardial constriction which is detected clinically as increased pressure in the jugular vein.
- *Differential diagnosis:*
 - restrictive cardiomyopathy
 - pulmonary embolus
 - right ventricular infarction
 - pleural effusion
 - chronic obstructive pulmonary disease

- *Special investigations:*

- Chest X-ray: ring of calcification around the heart, +/-cardiomegaly if associated with an effusion
- ECG: low voltages, non-specific upward sloping ST-T wave changes, atrial fibrillation, P mitrale (indicating chronic atrial changes)
- CT scan and MRI: confirm pericardial thickening and calcification
- ECHO: trans-oesophageal echo very sensitive,² increase in pericardial thickness > 2mm, abrupt inspiratory posterior motion of the intraventricular septum in diastole, non-pulsatile dilated IVC indicating venous hypertension
- Pulsed wave doppler (PWD) on ECHO:
 - trans-mitral flow velocity pattern very specific
 - increase in E wave with significant reductions in A wave
 - this indicates rapid early filling with premature equalisation of left atrial and left ventricular pressures from pericardial constriction
 - pulmonary vein flow changes
 - poor left atrial compliance and raised LA pressures redirects flow back into the pulmonary veins and results in decreased LA filling and decreased LV end diastolic volume
 - Cardiac catheterisation:
 - right ventricular trace = *square root sign*
 - RV pressures drop rapidly in early diastole = "dip" and a small amount of blood enters the RV
 - the limit of RV distensibility is quickly reached because of constriction by the rigid pericardium
 - rapid equalisation of RA and RV occurs and blood flow across the tricuspid valve ceases = plateau

Differentiating constrictive pericarditis (CP) from restrictive cardiomyopathy (RC)^{2,8:}

- Restrictive cardiomyopathy is an intrinsic myocardial disease resulting in impaired relaxation and reduced compliance of cardiac chambers with diastolic dysfunction.
- Many of the features of CP can also be found in RC making the diagnosis difficult.

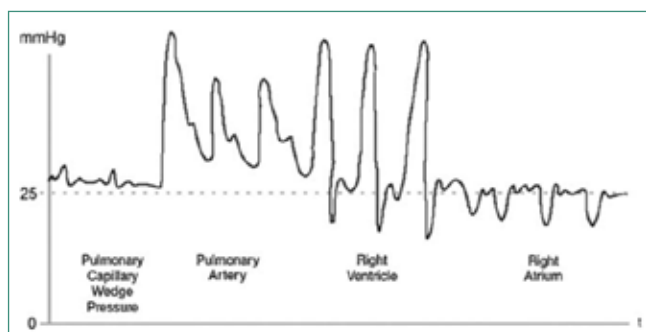


Figure 9: Right ventricular pressure tracings: pulmonary capillary wedge pressure, pulmonary artery pressure, right ventricular pressure and right atrial pressure with diastolic equalisation of pressures and sharp 'y-dip' in case of CP²⁶

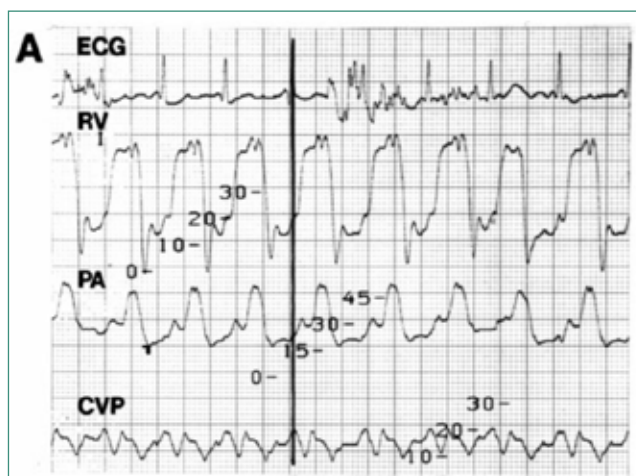


Figure 10: Intraoperative haemodynamic tracings before pericardiectomy. ECG: electrocardiogram, RV: right ventricular pressure, PA: pulmonary artery pressure, CVP: central venous pressure. Before pericardiectomy (A), the diastolic RV tracing demonstrates a "square root" sign, whereas the CVP shows "M" configuration with a plateau after the Y descent. The RV end-diastolic pressure is elevated at 20 mmHg.²⁴

- *Causes*

- Infiltrative disorders
 - amyloidosis
 - haemosiderosis
 - sarcoidosis
- Endomyocardial fibroelastosis
 - scleroderma
 - radiotherapy
 - idiopathic
- The most important differentiating features of CP are the uncoupling of intrathoracic and intracardiac pressures and the increased ventricular interdependence.
- On ECHO and doppler imaging:
 - E wave velocities (early diastolic filling) is normal in CP indicating preserved elastic recoil and compliance of the left ventricle
 - E wave velocities are reduced in RC because of the intrinsic myocardial disease
 - Respiratory variation in pulmonary vein flow and subsequent filling of the LA is present in CP but not in RC

Management for constrictive pericarditis^{2,8,26,27:}

- Medical management consists of diuretics, digoxin and beta-blockers to decrease venous congestion and tachyarrhythmias preoperatively.
- Anti-TB treatment is required for a minimum of 2 months before surgery in the case of TB pericarditis.
- Pericardiectomy with complete decortication is the only definitive treatment for established constriction
 - pericardial removal should always be as complete as technically feasible
 - perioperative mortality is highly dependent on the preoperative NYHA status

Clinical feature	Constrictive pericarditis	Restrictive cardiomyopathy
Early diastolic sound	Frequent	Occasional
Late diastolic sound	Rate	Frequent
Atrial enlargement	Mild or absent	Marked
Atrioventricular or intraventricular conduction defect	Rate	Frequent
QRS Voltage	Normal or low	Normal or high
Pulsus paradoxus	Frequent, but usually mild	Rare
Pericardium	Thickened	Normal
Left ventricular hypertrophy	Unusual	Frequent
Mitral or Tricuspid regurgitation	Rate	Frequent
Transmitral E wave velocity	Normal (or elevated)	Reduced
Transmitral flow velocity variation	Common: exhalation > inspiration ($\geq 25\%$)	Rare
Pulmonary venous flow velocities	Respiratory variation ($\geq 25\%$); S:D > 1	Little respiratory variation S:D < 1
Mitral annular velocities	E' > 8 cm sec ⁻¹	E' < 8 cm sec ⁻¹
CVP	No respiratory variation	Normal respiratory variation

S = systolic; D = diastolic; CVP = central venous pressure

Figure 11: Clinical and diagnostic features differentiating constrictive pericarditis from restrictive cardiomyopathy.² Clinical history and examination supported by special investigations will help differentiate the diagnosis.

- pericardial stripping is carried out via sternotomy or lateral thoracotomy with or without cardio-pulmonary bypass support
 - this is a technically challenging operation with significant risk:
 - *massive haemorrhage* should be anticipated with contingency plans in place
 - massive transfusion protocol activation pre-op
 - cardio-pulmonary bypass support immediately available with cannulation of femoral vessels pre-induction of anaesthesia in select patients
 - appropriate IV access and invasive monitoring
 - inotropes and vasopressor infusions drawn up and running
 - *damage to underlying epicardium, myocardium and coronary vessels* poses the most risk
 - overall mortality is around 12%
 - the underlying myocardium may also be affected by the disease process
 - myocardial atrophy after prolonged constriction, residual constriction from partial removal or an underlying myocardial process can lead to persistent congestive cardiac failure postoperatively
 - persistent constrictive physiology and abnormal diastolic filling patterns may be seen in a percentage of patients even after successful surgery.
 - Haemodynamic goals
 - maintain and augment preload
 - maintain afterload
 - maintain and augment contractility
 - maintain heart rate
 - maintain sinus rhythm to preserve atrial contribution to ventricular filling
 - fixed output state implies increased reliance on heart rate and SVR to maintain systolic blood pressures and organ perfusion
 - optimisation of preload and venous return pre-induction is important to compensate for the decreased filling of the cardiac chambers, bearing in mind the consequences of possible fluid overload
 - a balanced anaesthetic technique ensuring haemodynamic goals is necessary
 - anticipation for massive blood loss, both acute and ongoing, is of vital importance
 - the need for cardio-pulmonary bypass support will depend on the preoperative condition of the patient, aetiology of the CP, the underlying myocardial function and bleeding complications intra-operatively
 - the decision for the adjunctive use of bypass should be discussed and decided upon during the multidisciplinary team prior to surgery
 - Intra-operative arrhythmias are common and should be anticipated
 - defibrillator and anti-arrhythmic drugs should be immediately available.
- Predictors of outcome:
- An important predictor of long-term outcome is the aetiology of the pericardial disease.
 - Predictors of poor overall survival are previous radiation, impaired renal function, high pulmonary artery pressures, abnormal left ventricular systolic function, low serum sodium level and old age.

- Patients with mild or very advanced disease, renal failure or post-radiation constrictive pericarditis should be evaluated very carefully preoperatively as the risk of pericardiectomy may outweigh the benefits in these patients.

Conclusion

Pericardial disease can be challenging for the attending anaesthesiologist. An in-depth understanding of the pathophysiology of each disease state is necessary to provide optimal care for these patients. Careful pre-operative evaluation of the clinical history, examination and diagnostic investigations will allow adequate evaluation of risk and alert the anaesthesiologist to the necessary steps to be taken to manage these patients in the safest way possible.

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