Chapter

Anaesthesia for Patients with Pericardial Disease

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Abstract

Patients with pericardial disease can present to the anaesthesiologist for a variety of diagnostic procedures or therapeutic interventions. Providing safe anaesthesia care for these patients is challenging because of their primary pericardial pathology and significant co-morbidities affecting other organ systems. A thorough understanding of the pathophysiology of the pericardial disease state and its effect on overall haemodynamic variables is necessary before formulating a peri-operative care plan. A multidisciplinary approach involving cardiology, surgical, anaesthesia and critical care teams is needed to provide optimal peri-operative care. This chapter examines the basic pathophysiology of pericarditis, pericardial effusions, tamponade and constrictive pericarditis as these conditions pertain to the anaesthesiologist in the peri-operative period. Diagnostic tools, especially the role of echocardiography, that aid in the diagnosis and management of these high-risk patients are highlighted.

Keywords: anaesthesia, pericardial disease

1. Introduction

Patients with pericardial disease can present to the anaesthesiologist for a variety of diagnostic procedures or therapeutic interventions. Providing safe anaesthesia care for these patients is challenging because of their primary pericardial pathology and significant co-morbidities affecting other organ systems. A thorough understanding of the pathophysiology of the pericardial disease state and its effect on overall haemodynamic variables is necessary before formulating a peri-operative care plan. A multidisciplinary approach involving cardiology, surgical, anaesthesia and critical care teams is needed to provide optimal peri-operative care.

This chapter will examine the basic physical and physiological principles that govern the pathological states of pericarditis, pericardial effusion, tamponade and constrictive pericarditis. A thorough understanding of these principles is required prior to the induction of anaesthesia.

2. Normal anatomy and physiology

2.1 Normal anatomy

The normal pericardium is situated in the anterior mediastinum surrounding the heart and proximal portions of the great vessels [1–7]. It is composed of

two 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 [8]. The parietal and visceral layers are separated by a small amount of serous fluid [1–8]. In non-disease states, approximately 15–50 ml of fluid is contained within the pericardial sac. This fluid is produced by visceral mesothelium cells and is drained from the pericardial space via the lymphatic system to the right side of the heart.

The pericardium is fixed in its anatomical position to the diaphragm and sternum via the pericardio-phrenic and sterno-pericardial ligaments, respectively [4]. Posteriorly, the loosely bound connective tissue anchors it to structures contained within the posterior mediastinum.

Vascular supply is from the pericardio-phrenic artery which is a branch of the internal thoracic artery. Venous drainage is via the pericardio-phrenic veins which drain into the brachiocephalic veins bilaterally. The phrenic nerve provides sensory innervation and the sympathetic trunks' vasomotor innervation [4].

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 [4, 8]. The larger oblique sinus forms posteriorly between the left atrium and pulmonary veins. The transverse sinus too lies behind the left atrium, posterior to the aorta and pulmonary trunk [4]. Both sinuses are common sites for blood to collect post-cardiac surgery. Because of the formation of these two sinuses, the left atrium is not entirely an intra-pericardial structure.

2.2 Normal physiology and functions of the pericardium

Although normal cardiovascular function can occur in the absence of the pericardium, it does bestow certain physiological benefits [1–8]. Because of its relatively fixed ligamentous attachments, the pericardium stabilises the heart in its anatomic position and limits excessive movement within the chest cavity particularly with changes in body position. 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 pressure exerted on the cardiac chambers by the pressure within the intra-pericardial 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 neoplastic disease within the mediastinum. Prostaglandins secreted by mesothelial and endothelial cells of the pericardium regulate autonomic cardiac reflexes, modulate myocardial contractile function and influence epicardial coronary artery tone.

3. Pathology

There are a number of pericardial pathologies which can be a cause for concern in the peri-operative period [2–4, 9, 10].

3.1 Congenital defects

Congenital defects are rare, usually associated with other cardiac, pulmonary and skeletal abnormalities, and are often only found at autopsy with an incidence of 1:10,000 [9]. The absence of the pericardium is more commonly partial with the left side being affected about 70% of the time. Left-sided defects predispose to herniation of the heart which may become haemodynamically significant during

induction of anaesthesia or cause prolonged ischaemia due to compression of the coronary vessels. Right-sided defects may cause significant compression of the vena cava compromising venous return and cardiac output. Total pericardial absence is rare. Excessive cardiac motion and displacement associated with complete absence predispose to an increased risk of traumatic aortic dissection.

3.2 Acute pericarditis

Acute pericarditis is an inflammatory disease of the pericardium lasting less than 6 weeks and the most common pericardial pathology encountered in clinical practice [6, 9–16]. It may be a self-limiting benign condition or the first presentation of an underlying infectious or neoplastic disease process. In this setting, it is prudent to postpone elective surgery for diagnosis and initiation of appropriate treatment.

Causes of acute pericarditis vary widely and may be idiopathic, infectious, non-infectious or autoimmune. The most common causes encountered in the peri-operative period are non-infectious post-cardiac surgery, associated with trauma, uraemia in patients with chronic renal failure or post-myocardial infarction.

Symptoms include a sharp left precordial or retrosternal chest pain which may be pleuritic in nature and varies with posture, being decreased on sitting and increased on lying supine. The pain may radiate to the trapezius ridge. This pain referral is due to the involvement of the phrenic nerve which traverses the pericardium and supplies its sensory innervation. Often there are associated prodromal symptoms of malaise, fever and generalised myalgia. Tachycardia and tachypnoea are usually out of proportion to the low grade fever. A tri-phasic friction rub corresponding to atrial systole, ventricular systole and rapid early filling during diastole may be present on examination.

In the peri-operative period, differentiating between acute pericarditis and other causes of chest pain is of utmost importance. Careful clinical examination and special investigations should be carried out to make the correct diagnosis and allow for the institution of appropriate treatment. Three of the more important differential diagnoses are acute coronary syndrome, aortic dissection and pulmonary embolism.

Patients suffering from pericarditis associated with bacterial or fungal infections, malignancies, end-stage renal disease or post-cardiac surgery are at an increased risk to progress to pericardial effusion and tamponade [12].

Post-cardiac injury syndromes, also known as post-pericardiotomy syndromes, are being recognised as an important cause for pericardial disease [13, 14]. This clinical syndrome is characterised by a febrile illness associated with pleuritic chest pain and effusions of the pleura and pericardium. The initiating event is an injury to the pericardium, myocardium and pleura from ischaemia, post surgery or a non-iatrogenic traumatic event. In predisposed individuals, an autoimmune-mediated response is triggered that can vary from a simple, self-limiting pericarditis to a complicated pleuropericarditis, resulting in massive pericardial and pleural effusions with tamponade.

Special investigations would include a 12-lead electrocardiogram (ECG) which will show sinus tachycardia, PR interval depression and diffuse concave upward sloping ST-segment elevation. Transthoracic echocardiography (TTE) may show an associated pericardial effusion and tamponade as well as other cardiac or para-cardiac diseases.

Treatment for acute pericarditis remains symptomatic with non-steroidal anti-inflammatory drugs (NSAIDs) for pain and potentially adding colchicine as an adjunct to prevent recurrence [15]. Low-dose corticosteroids may also be introduced by the primary care team if there is an associated autoimmune disease or in the case of a post-cardiac injury syndrome [13, 14, 16].

3.3 Chronic pericarditis

Chronic pericarditis may be a result of the progression of acute disease or due to recurrent episodes of relapse [6, 9, 10]. The peri-operative management will depend on haemodynamic consequences the disease process has on patient physiological parameters.

Differentiation should be made between chronic pericarditis with ongoing inflammation, pain and fever, relapsing disease where patients have periods of being symptom free and chronic pericardial effusion with persistent fluid accumulation within the pericardial sac.

As with acute pericarditis, all elective surgery should be postponed to enable symptomatic treatment of attacks with NSAIDs, colchicine and corticosteroids. Pericardiectomy is indicated in patients with frequent and severe symptoms that are unresponsive to maximal medical therapy [6, 9].

Chronic inflammatory disease may be associated with a pericardial effusion. Moderate to large effusions, determined at echocardiography as being more than 10 mm separation of the pericardial layers during diastole, should be drained before any elective surgery takes place. Haemodynamic effects of the effusion should be assessed via echocardiographic studies and quantified prior to induction of anaesthesia as detailed below.

3.4 Pericardial effusions and tamponade

Pericardial effusion occurs when there is excessive fluid accumulation within the pericardial space [3–10, 12, 17–41]. The effusion may be transudative, exudative, haemorrhagic or purulent depending on the cause. Progressive accumulation of fluid within the pericardial sac may lead to compression of cardiac chambers, obstruction of cardiac filling and tamponade.

3.4.1 Causes for effusion

Causes of pericardial effusion are similar to those for pericarditis and may be due to the inflammatory process itself [6, 16, 18–21]. The most common causes of pericardial effusion seen in the peri-operative period are iatrogenic, associated with cardiac surgery or percutaneous coronary intervention (PCI); traumatic as seen in blunt or penetrating chest injuries; associated with malignancy; as a consequence of end-stage renal disease or dialysis; and those occurring from infectious diseases. In developing countries, especially where the prevalence of HIV is high, tuberculous pericarditis is common [6, 17].

There are a few groups of patients that require special mention as they frequently need intervention by the anaesthesiology team and present with pericardial effusions in the peri-operative period.

3.4.1.1 End-stage renal disease

Patients with end-stage renal disease (ESRD) frequently present to the anaesthesiologist for a number of diagnostic, therapeutic or vascular access procedures [6, 18–21]. The association with end-stage renal and cardiovascular disease is well established. Increased inflammatory processes, immune and autoimmune dysfunction, dyslipidaemia, endocrine abnormalities, oxidative stress and accumulation of toxic metabolites have all been suggested as causes for this association. This population is known to have an increased incidence of coronary artery disease, valvular pathology, arrhythmias and myocardial and pericardial diseases [18].

The incidence of pericarditis in ESRD is 2–21% with effusion and tamponade being present in up to 14–56% of patients. The aetiology differs between patients on haemodialysis and those who are not. Patients not yet on haemodialysis develop a uraemic pericarditis and effusions that generally respond well to aggressive dialysis and filtration. The incidence of uraemic pericarditis is decreasing because of more efficient dialyzer membranes and methods.

Patients on haemodialysis develop dialysis pericarditis with effusions that more frequently require drainage via a pericardial window procedure. This group of patients has a higher incidence of progressing to constrictive pericarditis possibly requiring pericardiectomy [18]. Progression from effusion to tamponade may be difficult to diagnose, but progressive right heart failure and hypotension in the setting of adequate diuresis is highly suggestive [18]. All patients with ESRD presenting for surgery should have pre-operative investigations to rule out significant pericardial disease that may impact on peri-operative management.

Indications for drainage of effusions in these patients are based on clinical and biochemical features. Dialysis pericarditis, tachypnoea >20 breaths/min, fever >39°C, low voltage complexes on 12-lead ECG, hypoalbuminaemia <31 g/l, leukocytosis and any signs of tamponade on TTE all indicate the need for drainage [18]. There are little data to support one drainage procedure over another [6, 18]. Pericardiocentesis and pericardial window are both acceptable methods for relief of raised intra-pericardial pressure.

Although these patients may exhibit signs of large effusions and even tamponade, significant haemodynamic compromise associated with anaesthesia is rare in this group of patients. Because of the chronicity of the disease process and the associated hypertension, careful titration of agents used for general anaesthesia to facilitate drainage procedures is usually well tolerated [21].

3.4.1.2 Post-cardiac surgery and post-operative pericardial effusions (PPE)

Patients post-cardiac surgery are another group whose management frequently requires the involvement of the anaesthesiology team [24, 32–38]. Accumulation of blood within the pericardial sac because of ongoing bleeding from either a surgical or medical cause peri-operatively will lead to the development of PPE and tamponade. PPE can be divided into early, presenting within 7 days, and late complications occurring more than 7 days post-procedure. An important cause of persistent PPE is the inflammatory response associated with post-cardiac injury syndrome [13, 14, 33].

The incidence for PPE has a wide range depending on the study quoted and is approximately 20% on the 20th post-operative day [33]. Most effusions are clinically insignificant, defined as less than 10 mm in diastole on TTE, and resolve spontaneously after reaching their maximum volume on day 10 post-operatively [33]. The incidence of significant pre-tamponade and tamponade requiring re-intervention is 1–2.6% [33].

Presenting symptoms are usually non-specific consisting of tachycardia, hypotension, tachypnoea, orthopnoea and decreased heart sounds [24, 32–34]. This low cardiac output state must be differentiated from other common causes of cardiogenic shock in the post-operative period. The differential diagnosis would include hypovolaemia, significant ischaemia, ventricular dysfunction or severe inflammatory response syndrome [32].

The following have been identified as independent risk factors for the development of post-operative effusions [33, 34, 37]: heart transplantation, pulmonary thromboembolism, aortic aneurysm surgery, increased body surface area, valve surgery, immunosuppression, urgent or emergent surgery, renal failure and

prolonged cardiopulmonary bypass (CPB) times. In other studies, female gender, pre-operative use of anticoagulants and early chest drain removal have been associated with increased incidence of PPE [33, 37].

In the setting of post-operative haemodynamic deterioration, critical tamponade may develop quickly with even small amounts of fluid accumulation causing significant haemodynamic effects. The diagnosis should be made immediately in order to facilitate life-saving intervention. TTE remains the diagnostic tool of choice because it is non-invasive and has a high sensitivity and specificity for detecting the signs of cardiac tamponade [6, 24, 32, 35, 38]. It may also be useful in confirming or ruling out other causes for post-operative haemodynamic deterioration. Because tamponade is a dynamic pathological process, especially in the post-operative period, serial or repeat echocardiographic examinations coupled with ongoing clinical evaluation are necessary to ensure appropriate and timeous management.

Although TTE remains the diagnostic tool of choice, detecting effusions and tamponade post-operatively may be challenging [31, 32, 38]. Patients who have altered anatomy are most likely to be on positive pressure ventilation with positive end expiratory pressure (PEEP), may have pneumopericardium (air trapped in the pericardium) and will have drains in situ. Effusions post cardiac surgery are often loculated causing regional compression of only one or some of the cardiac chambers. Blood and clots collect in the posterior sinuses making diagnosis via the traditionally accepted criteria on TTE difficult. Trans-oesophageal echocardiography (TOE) is well tolerated in sedated or anaesthetised patients but is more invasive and may not be readily available. Supplemental modalities such as CT scan and cardiac MRI have been suggested to aid in the diagnosis when TTE is inconclusive [6, 31, 38].

Indications for drainage of effusions are based on clinical and TTE findings of tamponade: left ventricular dysfunction in patients receiving intermittent positive pressure ventilation, loculated effusions or clots causing compression of the left atrium or left ventricle, documented collapse of right-sided chambers and if pericardial separation is >20 mm [3, 32, 35].

Anaesthetic management of post-operative effusions and tamponade remains the same as for an effusion from any other cause. The need for ongoing inotropic support and appropriate mechanical ventilation, massive fluid shifts, ongoing bleeding due to surgical causes, coagulopathy due to anticoagulant use, intrinsic clotting abnormalities, need for massive blood transfusion, renal dysfunction and the significant risks of re-sternotomy are just a few of the unique challenges for the anaesthesiologist to consider when taking a cardiac patient back to theatre for re-intervention. Drainage procedures are most often limited to pericardiocentesis under ultrasound- or video-assisted guidance [25, 32, 35]. A small percentage of patients may require sternotomy for the effective management of post-operative effusions [25, 32].

3.4.1.3 Effusions in patients undergoing percutaneous cardiac interventions

Percutaneous intracardiac intervention is a risk factor for the development of pericardial effusion and tamponade [39–41]. The exact incidence depends on the procedure performed, the extent of the intervention, whether it is diagnostic or therapeutic, and if anticoagulation is used in the peri-procedural period. The exact procedure-specific incidence is difficult to determine, and small to moderate effusions may occur much more frequently than studies suggest [39]. The incidence of tamponade ranges from <1 to 6% depending on the study being quoted. Some of the more commonly performed interventions include atrial fibrillation ablation associated with a 4% risk of tamponade, permanent pacemaker insertion which carries a 1.7% risk of cardiac perforation and life-threatening tamponade and left atrial

appendage (LAA) occlusion procedures which are associated with a 1.8–3% risk of tamponade, most commonly due to LAA perforation.

The overall incidence of tamponade following percutaneous coronary intervention (PCI) is 0.12% and is associated with high mortality [40, 41]. Coronary artery perforation following percutaneous coronary intervention is found in 0.3% of patients of which 10% went on to develop tamponade [41]. The risks were increased in patients requiring more complex interventions and if athero-ablative procedures were performed instead of angioplasty and stent placement.

Pericardial effusion and tamponade following PCI usually present acutely in the catheterisation lab but can have an insidious onset and are an important cause for late hypotension post intervention [40].

The diagnosis of tamponade post PCI remains predominately clinical and should be confirmed with TTE and other imaging techniques as soon as possible in order to facilitate life-saving treatment drainage procedures as described below.

3.4.2 Pathophysiology of pericardial effusions

The haemodynamic effects of the effusion and the development of tamponade are dependent on the rate of fluid accumulation within the pericardial space, the total volume of fluid that accumulates, the type of the fluid that accumulates and the intrinsic compliance of the pericardial tissue layers [4, 5, 7, 24].

Because the pericardium is relatively stiff, it has a limited reserve volume. Small but rapidly accumulating effusions quickly exceed the compliance of the parietal layer. 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 and tamponade.

Chronic accumulation over a prolonged period allows for pericardial stretch and compensatory mechanisms meaning that the intra-pericardial pressure remains low despite a large amount of fluid within the space. Even with additive amounts of fluid, the pericardial compliance curve remains less steep than in the acute setting resulting in slower increases in intra-pericardial pressures relative to the volume of fluid within the space.

The type of fluid that accumulates may influence the haemodynamic presentation. Exudative fluid may become fibrinous and cause constriction, haemorrhagic fluid may contain clots causing isolated chamber compression and chronic effusions may become fibrotic and confining.

The compliance of the pericardial tissues is also an important determining factor in the overall haemodynamic effects of an effusion. Pericardial compliance is decreased in mesothelioma, scarring from previous cardiac surgery and constrictive states.

Once intra-pericardial and intracardiac pressures increase beyond a certain limit, cardiac chamber filling and preload are reduced which causes a drop in stroke volume and cardiac output. This decrease 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 are an attempt to maintain systemic blood pressure and organ perfusion in the face of a low cardiac output state.

3.5 Tamponade

Tamponade physiology occurs when cardiac chambers exhibit compression due to an increase in intra-pericardial pressures and equalisation of transmural pressures [3–10, 14–17, 22–24].

3.5.1 Causes of tamponade

Patients most at risk for the development of tamponade are those with iatrogenic causes post-cardiac surgery, PCI, insertion of pacemaker or percutaneous valve repair, chest trauma where blunt injuries are five times more commonly associated with tamponade when compared with penetrating injuries [25], malignant disease which is the most common cause of cardiac tamponade accounting for up to 60% of cases [25] and ESRD. Uraemic disease accounts for 10–15% of cases, while the incidence of dialysis pericarditis is 12% in patients on long-term haemodialysis [18–21].

3.5.2 Pathophysiology of cardiac tamponade

Under normal circumstances, intra-pericardial pressures reflect intrathoracic pressures and are negative [3–10, 12, 21–31]. As fluid accumulates within the pericardial space, the intra-pericardial pressures increase. Pericardial pressures are elevated throughout the cardiac cycle and cause compression of chambers for the duration of diastole when intracardiac chamber pressures are lowest.

Impaired filling of the cardiac chambers leads to diastolic dysfunction and a type of obstructive shock [23]. The venous return pattern to the atria eventually becomes unimodal and is confined to ventricular systole. In severe tamponade, atrial filling only occurs when ventricular contraction forces the atrioventricular valvular apparatus towards the myocardial apex. This caudad motion of the tricuspid and mitral valves causes a decrease in atrial pressures and enhanced systemic and pulmonary venous return.

As tamponade physiology progresses, ventricular filling becomes highly dependent on atrial systole as the intra-pericardial pressures increase. The maintenance of a sinus cardiac rhythm becomes paramount to ensure adequate ventricular filling to maintain end-diastolic ventricular volumes which are equivalent to ventricular stroke volume.

Physiologically speaking, cardiac chamber filling pressures are dependent on the myocardial transmural pressure gradient expressed mathematically by the following:

transmural pressure = intra-pericardial 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 (P1–P2 or V) between the systemic venous return and the aorta. The pressure gradients below are often seen during diagnostic cardiac catheterisation procedures when venous and arterial pressures are obtained and documented:

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 intra-pericardial pressure increases, extrinsic compression of the cardiac chambers occurs. Compression causes the intracardiac chamber pressures to increase, and the transmural pressures begin to equalise. Right-sided filling pressures during diastole are lower than the left and are the first to be equalled and then exceeded by the increasing intra-pericardial pressures.

The right atrium and ventricle are compressed, right-sided chamber filling is compromised and the right atrial pressure, measured clinically as the central venous 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.

A decrease in right atrial venous return leads to impaired right ventricular filling, an underloaded right ventricle which functions at the lower end of the Frank-Starling curve and a resultant decrease in 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 eventually transpires. 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 usually avoided.

It is of utmost importance to note that the equalisation of transmural pressures found in cardiac tamponade is a dynamic process influenced by extra-cardiac factors. Induction of general anaesthesia causes a decrease in heart rate, vasodilation and direct myocardial depression. Coupled with the institution of positive pressure ventilation, massive decreases in venous return and cardiac output occur. Thus, induction of anaesthesia may precipitate complete cardiovascular collapse in patients with tamponade.

Patients at risk for the development of tamponade should therefore have echocardiographic studies to confirm their pre-operative haemodynamic state. In the event of potential or imminent tamponade, all elective procedures should be postponed and the pericardium drained via the most appropriate method to relieve transmural pressures before induction of anaesthesia is performed.

3.5.3 Classification of tamponade

Not all cases of cardiac tamponade are uniform, and patients can present with different forms [24, 25].

Low-pressure tamponade occurs at very low diastolic pressures of 6–12 mmHg and is present in patients with severe hypovolaemia. Patients do not present with the full clinical picture of tamponade until they have been adequately fluid resuscitated and are fluid replete. This form is often found in trauma patients with ongoing haemorrhage or post-cardiac surgery.

Regional tamponade occurs when only specific cardiac chambers are compressed from localised amassing of fluid. This is a common finding post-cardiac surgery or myocardial infarction. Typical clinical findings of acute tamponade are often absent necessitating multimodal diagnostic imaging to make the diagnosis.

Hypertensive tamponade is said to be present when there are classical features of tamponade but with elevated arterial pressures, sometimes over 200 mmHg. It is thought to be due to excessive beta-adrenergic drive in patients with pre-existing hypertension.

Effusive-constrictive pericarditis is present when there is decreased pericardial compliance with a reduced pericardial cavity associated with an effusion. Constrictive physiology is present, and tamponade can occur even with minimal amounts of fluid accumulation. A variety of haemodynamic and imaging signs

make for a mixed clinical picture. Drainage of the effusive fluid provides the best clue as symptoms of constriction persist in spite of appropriate drainage.

3.5.3.1 Clinical signs and diagnosis

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 is a clinical sign commonly found in tamponade and refers to the exaggeration of the normal physiological variation in systolic blood pressure of more than 10 mmHg during negative pressure ventilation. Under normal physiological conditions, the intra-pericardial pressure mirrors intrathoracic pressures. Negative intrathoracic pressure generated on inspiration is transmitted to the heart, and the intra-pericardial pressure decreases. This in turn increases the transmural pressure gradient. The increase in transmural pressure gradient augments right atrial and ventricular filling during diastole. This is demonstrated on Doppler echocardiography by increased blood flow velocity across the tricuspid valve during inspiration [28].

The augmentation in right ventricular filling causes the intra-ventricular septum to shift to the left which transiently impacts on left ventricular filling. This is known as *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. There is a small decrease in left ventricular stroke volume and cardiac output which 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. An increase in left ventricular stroke volume and systolic blood pressure is then seen during expiration.

In cardiac 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 intra-ventricular septum shifts to the left, the left heart cannot expand because of the increased intra-pericardial pressures. Left ventricular filling and flow through the left ventricular outflow tract are affected to a greater degree resulting in *enhanced ventricular interdependence.* This translates into a decrease of systolic blood pressure of more than 10 mmHg during inspiration.

Pulsus paradoxus is not very sensitive or specific for cardiac tamponade and may be absent even when tamponade is present or be present with conditions other than tamponade [22, 24, 25].

Pulsus may be absent in the presence of tamponade with severe aortic regurgitation where left ventricular filling is maintained due to back flow from the aorta; with localised effusions or clot that compress only certain chambers of the heart; with an atrial septal defect where the pressure effect on the right atrium is balanced by the intra-cardiac shunt; and with severe right ventricular hypertrophy with pulmonary hypertension where the pressure effects from increased intra-pericardial pressure on right heart are less pronounced.

Pulsus paradoxus may exist in the absence of tamponade in severe chronic obstructive pulmonary disease, asthma or severe bronchospasm, hypovolaemia, congestive cardiac failure and obesity.

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 expiration [22, 25]. This can be interpreted as systolic pressure variation in patients receiving mechanical ventilation which is often used clinically as a sign of fluid responsiveness.

Mechanical ventilation with PEEP has three main effects on cardiac physiology. (1) With the increase in intra-thoracic pressure, alveolar pressures increase more than pleural pressures which causes compression of pulmonary capillaries and increased right ventricular afterload. (2) Compression of pulmonary capillaries squeezes blood from the pulmonary bed into the left atrium augmenting left ventricular preload. (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 pressures. Pulmonary transit time is approximately 2 s, and the drop in right heart cardiac output from positive intrathoracic pressures translates into a drop in left ventricular cardiac output and systolic blood pressure during expiration. A reversed pattern of pulsus paradoxus is seen.

As mentioned above, the combination of decreased venous return or preload to the right heart with induction of anaesthesia plus the increased right ventricular afterload effects of mechanical ventilation can severely compromise cardiac function in patients with tamponade.

3.5.3.2 Special investigations

Chest X-ray will show an enlarged globular bottle-shaped cardiac shadow if the pericardial fluid volume is >200 ml, a widened mediastinum with a right costophrenic angle <90° and clear lung fluids [7–10, 12, 21–29]. There may be an associated pleural effusion that requires concurrent drainage and management.

ECG may show a sinus tachycardia which is the most common finding. Other findings may include low QRS and T wave voltages, PR segment depression, nonspecific ST-T wave changes and bundle branch block. Electrical alternans is the change in QRS voltage with each beat and reflects excessive swinging movement of the heart within a large effusion [21].

TTE is the investigation of choice to diagnose and evaluate pericardial disease [3–6, 12, 23–28, 30, 31]. It is a class 1 recommendation with a high sensitivity and specificity for the detection of effusions and tamponade. TTE in addition allows for the evaluation of myocardial and endocardial structures and function, aiding to rule out other causes for the clinical picture. A grading system for the size of the effusion is based on echo-free space in diastole with small effusions being <10 mm in size, moderate effusions 10–20 mm and large effusions >20 mm. Right atrial collapse and invagination for >30% of the cardiac cycle on ECHO is 100% sensitive for the diagnosis of tamponade [26].

Other TTE signs of tamponade include distention and loss of respiratory variation in the size of the IVC which reflects increased right atrial pressures, increased respiratory variations in tricuspid and mitral trans-valvular inflow velocities on Doppler studies, intra-ventricular septal shift reflected clinically as pulsus paradoxus [26], localised or loculated effusions showing isolated chamber compression and regional tamponade commonly encountered post-cardiac surgery [32].

Cardiac catheterisation is usually not performed in acute tamponade as TTE is less invasive, less time-consuming and diagnostic. If catheterisation is performed, a blunted Y descent may be demonstrated [29]. The Y descent on the CVP tracing after the v wave indicates opening of the tricuspid valve and reflects early diastolic filling of the right ventricle. Attenuation of the y descent reflects rapid equalisation of pressures between the right atrium and ventricle which leads to ineffective ventricular filling during early diastole in tamponade (**Figure 1**).

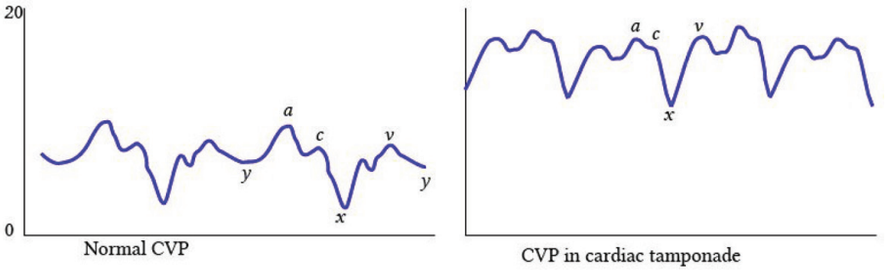

Figure 1.
CVP tracing in cardiac tamponade reflecting higher baseline pressures and attenuation of the y descent.

3.5.4 Medical management of pericardial effusions and tamponade

In about 60% of cases of pericardial effusion, an underlying disease process is known, and medical management would therefore be targeted at the aetiology [6]. In the absence of significant associated inflammation, NSAIDs, colchicine and steroids are generally ineffective, leaving no conservative management options for isolated effusions. In these instances, surgical drainage is indicated.

3.6 Constrictive pericarditis (CP)

Chronic inflammation of the pericardium causes it to thicken and become non-compliant and rigid [4, 6, 7, 9, 10, 12, 25, 31, 43–49]. This thickening may be concentric or regional depending on the cause. The pericardial space becomes obliterated as the visceral and parietal layers change and stiffen. Restriction of cardiac chamber expansion during diastole causes impedance of cardiac filling and diastolic dysfunction at both the atrial and ventricular levels. The usual form is constriction without effusion but effusive-constrictive forms can occur.

3.6.1 Causes of constrictive pericarditis

Causes are similar to those for acute and chronic pericarditis including infectious disease, radiation to the mediastinum, trauma and autoimmune conditions [4, 13, 14, 48, 49]. In the developing world, tuberculosis remains the most common cause with a prevalence of >90% in places where the HIV infection rates are high [49].

Post-cardiac surgery is the most common cause of CP in the peri-operative period. The usual presentation is constrictive pathophysiology with an increase in pericardial membrane thickness. However, constrictive pathophysiology may present in the absence of pericardial membrane thickening [4, 48].

Post surgery, blood in the pericardial space causes a post-pericardiotomy-type picture with inflammation and adhesions [4, 13, 14]. These adhesions diminish the pericardial space and cause a constrictive pathophysiological picture in spite of normal pericardial thickness. This is termed *normal thickness constrictive pericarditis*. In a Mayo Clinic study from 2003, 18% of patients with proven constriction who underwent pericardiectomy had a normal pericardial thickness of <2 mm on pre-operative imaging [48]. Disease of the pericardium in the normal thickness group consisted of mild focal areas of inflammation, calcification and fibrosis. The majority of patients were post-cardiac surgery and post chest irradiation or had suffered previous infective or idiopathic disease. Pericardiectomy was equally effective in relieving the disease symptoms in these patients compared with those who had a pericardial thickness > 2 mm. It would therefore seem reasonable to consider pericardiectomy in patients with a normal pericardial thickness if the endocardial biopsy is negative for restrictive cardiomyopathy [4, 48].

3.6.2 Pathophysiology of CP

Under normal circumstances, the pericardium can accommodate changes in cardiac volume [4, 6, 9, 12, 25, 43, 46, 47, 49]. With ongoing inflammation, the pericardial compliance decreases. The now thickened, calcified and rigid pericardium encases the heart and limits cardiac chamber expansion during diastole. Initially, early diastolic filling is not affected, and the atrial contribution to ventricular filling is mostly impeded during mid- to late diastole.

Over time, diastolic filling becomes 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 therefore 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 is because the pericardial space has been destroyed by the inflammatory process and intrapericardial pressure no longer mirrors intrathoracic pressures [25]. 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 which is known as Kussmaul's sign.

Respiratory variation in systolic blood pressures may still be seen clinically as the pulmonary veins lie outside of the pericardium and the left atrium is only partially within the pericardial sac. Changes in intrathoracic pressures will therefore still cause changes in pulmonary vein flow despite the uncoupling of intra-pericardial and intrathoracic pressures [4]. 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 not enough to cause pulsus paradoxus.

Another important pathophysiological feature of CP is the dramatic increase in ventricular interdependence [45]. 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 intraventricular 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 overall ventricular filling.

Chronic constriction of the myocardium can lead to damage of the underlying muscle tissue. Constriction causes the ventricles to operate at the lower end of the Frank-Starling curve which causes a kind of disuse atrophy within the myocardium. 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 ongoing inotropic support in the peri-operative setting.

3.6.2.1 Clinical signs and diagnosis

Signs and symptoms are usually non-specific and mimic right ventricular failure making diagnosis difficult [4, 6, 12, 25]. Tachycardia is the predominant sign because of the fixed stroke volume state. Arrhythmias, especially atrial fibrillation, are not uncommon. Fluid overload ranging from peripheral oedema to anasarca is a usual finding. This is due to venous hypertension which is often worsened by protein-losing enteropathy in decompensated disease. Patients may have associated pleural effusions and cachexia, indicating a chronic and insidious disease process.

Decreased cardiac output with shortness of breath and fatiguability are almost always elicited on thorough enquiry. Ascites, hepatomegaly, pleural effusion and peripheral oedema can be misdiagnosed as chronic liver disease.

Classical clinical findings in CP include a pericardial knock and Kussmaul's sign. A pericardial knock is a high-pitched sound occurring in early diastole before the third heart sound. It indicates the cessation of ventricular filling at the end of the early diastole due to the pericardial constriction.

Kussmaul's sign is a paradoxical increase in jugular venous pressure on inspiration. It reflects an increase in right atrial preload from an increase in intra-abdominal pressure during inspiration. This increased venous return from the intra-abdominal veins is independent of intrathoracic pressure changes. The increase in preload cannot be accommodated because of the fixed pericardial constriction, and the back pressure is transmitted to the systemic venous system. This is detected clinically as increased pressure in the jugular vein.

The differential diagnosis for constrictive pericarditis is important to consider as clinical signs can be confusing. Restrictive cardiomyopathy, pulmonary embolus, right ventricular infarction, pleural effusion and chronic obstructive pulmonary disease can all have a similar clinical presentation.

3.6.2.2 Special investigations

The diagnosis of constrictive pericarditis remains challenging, and the clinical picture of unexplained venous congestion should be clarified with multiple imaging modalities to ensure accurate and efficient diagnosis [4, 6, 12, 25, 31].

Chest X-ray may show a ring of calcification around the heart and cardiomegaly if there is an associated effusion.

ECG may show low voltages, non-specific upward sloping ST-T wave changes, atrial fibrillation and P mitrale, indicating chronic atrial hypertension.

CT scan and MRI are both useful to confirm pericardial thickening and calcification [4, 31]. CT scan can delineate and quantify the degree of calcification based on CT scan score, and MRI has a 93% accuracy for differentiating CP from restrictive cardiomyopathy based on a pericardial thickness of >4 mm.

TOE has a high sensitivity for the detection of features consistent with the diagnosis [12]. An increase in pericardial thickness > 2 mm, abrupt inspiratory posterior motion of the intra-ventricular septum in diastole and a non-pulsatile dilated IVC indicating venous hypertension are all expected echocardiographic features.

Pulsed wave Doppler on echocardiography can be used to assess the mitral trans-valvular inflow velocity pattern. Classically, an increase in the E wave velocity represents rapid early diastolic filling. Significant reductions in the A wave velocity represents decreased flow in mid- to late diastole. This indicates rapid early filling with premature equalisation of left atrial and left ventricular pressures from pericardial constriction.

Reverse pulmonary vein flow changes will be detectable as the poor left atrial compliance and raised left atrial pressures redirect flow back into the pulmonary veins. This results in decreased left atrial filling and decreased left ventricular end-diastolic volume.

Cardiac catheterisation may reveal a classical square root sign pattern on the right ventricular trace. This is because right ventricular pressures drop rapidly in early diastole which causes a "dip" in the trace as a small amount of blood enters the right ventricle. The limit of RV distensibility is quickly reached because of constriction by the rigid pericardium. Rapid equalisation of right atrial, right ventricular and pulmonary wedge pressures occurs, and blood flow across the tricuspid valve ceases abruptly causing a "plateau" in the pressure tracing (**Figure 2**).

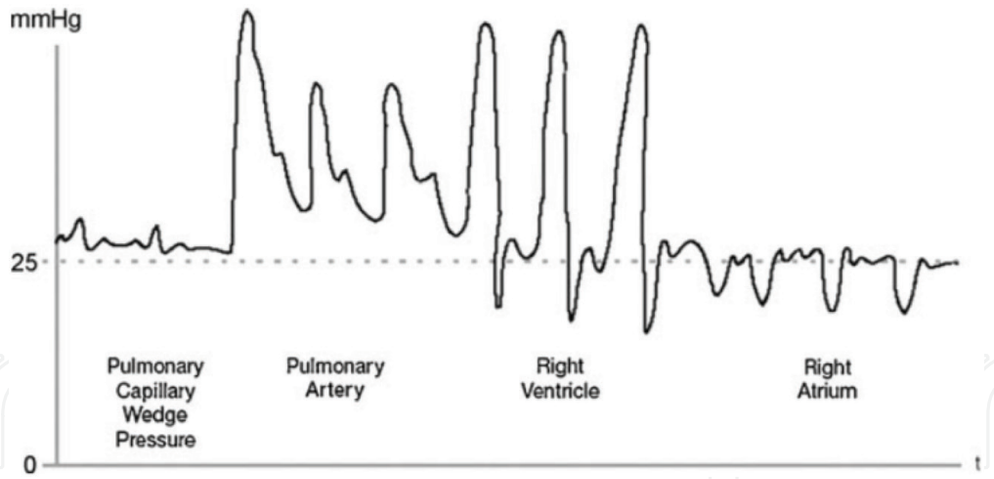

Figure 2.
Right ventricular pressure tracings: pulmonary capillary wedge pressure, pulmonary artery pressure, right ventricular pressure and right atrial pressure with diastolic equalisation of pressures and sharp "y-dip" in the case of CP [46].

Kussmaul and Vaitkus were the first to delineate criteria for correctly diagnosing CP in patients on whom cardiac catheterisations were performed. They identified three criteria that could be used in the diagnosis: a left and right ventricular end-diastolic pressure difference of <5 mmHg, a ratio of right ventricular end-diastolic pressures to right ventricular systolic pressures of >1.3 and a right ventricular systolic pressure of <50 mmHg. If all three criteria are present and patients have clinical features in keeping with the condition, CP will be correctly diagnosed in >90% of patients [49].

3.6.3 Medical management for constrictive pericarditis

Medical management is focused on the underlying cause of the pathology and may include diuretics, digoxin and beta-blockers to decrease venous congestion and tachyarrhythmias pre-operatively [9, 12, 46, 47, 49]. In the cases of TB pericarditis, anti-tuberculous chemotherapeutic treatment is required for a minimum of 2 months before surgery but can be continued for up to 12 months pre-operatively.

3.6.4 Differentiating constrictive pericarditis (CP) from restrictive cardiomyopathy (RC)

Many of the features of CP can also be found in RC making the diagnosis difficult [4, 6, 9, 12]. Restrictive cardiomyopathy is an intrinsic myocardial disease resulting in impaired relaxation and reduced compliance of cardiac chambers with severe diastolic dysfunction and atrial hypertension. Causes include infiltrative disorders such as amyloidosis, haemosiderosis and sarcoidosis or endomyocardial fibroelastosis found in scleroderma, radiotherapy or idiopathic disease.

The most important differentiating features in CP are the uncoupling of intrathoracic and intracardiac pressures and the increased ventricular interdependence. In RC, normal respiratory variation will be found on the CVP tracing. Conduction defects are also more common in patients with RC.

Differentiating features can also be elicited on TTE investigation [12]. E wave velocities reflecting early diastolic filling are reduced in RC because the intrinsic myocardial disease affects ventricular filling throughout the whole of diastole. Transmitral inflow velocity variation and respiratory variation in pulmonary vein flows are rarely found in RC. Atrial enlargement is marked reflecting severe atrial hypertension. Left ventricular hypertrophy is a common finding, and pericardial thickness will be normal. Pericardial calcifications will be absent in RC.

4. Anaesthetic management

4.1 Pericardial effusions and tamponade

4.1.1 Pre-operatively

Accumulation of fluid in the pericardial space is the primary problem, and therefore definitive drainage and relief of the raised intra-pericardial pressure are required [9, 12, 21, 22, 32]. The type of drainage procedure will depend on the aetiology of the pericardial effusion and the clinical condition of the patient. A multidisciplinary team approach is needed, and a thorough pre-operative workup should be done if time allows. Clinical history and examination should be carried out focusing on pertinent physiological symptoms and signs to identify the cause and severity of the pathology.

Patients fall broadly into two categories: pericardial effusion with pre-tamponade physiology and a haemodynamically stable clinical picture or features of tamponade are present on TTE with haemodynamic consequences and instability. The presence of orthopnoea and pulsus paradoxus is a sign 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 "Airway, Breathing, Circulation" approach bearing in mind the pathophysiology of tamponade as discussed above. If required, a definitive airway management strategy should be employed and supplemental oxygen administered in order to optimise the delivery of oxygen to tissues during the decreased cardiac output state. 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 and should be secured before any pharmacological agents are administered. Invasive arterial and central venous lines are needed for monitoring but should not delay drainage for patients in extremis.

Optimisation of preload is advised pre-operatively as hypovolaemia will worsen tamponade physiology. 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. Improvement will be if the 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 also exacerbate volume overload post-drainage. Temporary haemodynamic support with a vasopressor agent may be more appropriate than excessive fluid administration to maintain blood pressure in the pre-operative period.

4.1.2 Types of drainage procedures

Needle pericardiocentesis consists of percutaneous placement of a catheter into the pericardial sac to facilitate external drainage of the effusion [24, 25]. Specific anatomical landmarks with echocardiographic and/or fluoroscopic guidance are used to insert the catheter using a Seldinger technique. This can be done in an awake patient with supplemental local anaesthetic and is the treatment of choice for an iatrogenic tamponade caused in the interventional suite [38]. The subcostal approach is safest for a blind procedure in the emergency setting where TTE may not be available. The apical and parasternal approaches may also be used. Pericardiocentesis is

diagnostic and therapeutic as drained fluid can be sent for testing. It is the preferred method of drainage in hypotensive patients.

Percutaneous balloon pericardiotomy is performed in a similar manner to TTE-guided pericardiocentesis and is effective in patients with malignant effusions. A balloon is used to generate a pericardial window to prevent recurrence of the tamponade.

Surgical procedures include a pericardial window performed via a substernal approach. Anterolateral thoracotomy and video-assisted thoracoscopic approaches may also be used. Surgical drainage allows for the formation of a pleuropericardial window which allows ongoing drainage of the effusion and decreases the risk of recurrent tamponade. Surgical drainage is preferred for haemopericardium, purulent effusions and recurrent disease. The sub-xiphoid approach consists of a small sub-xiphoid incision made to relieve pressure and directly visualise the parietal pericardium. The advantages of this approach include its effectiveness and simplicity and that it can be performed under local anaesthesia if needed for the patient nearing cardiovascular collapse.

4.1.3 Complications of drainage procedures

Needle pericardiocentesis under ultrasound guidance is considered a life-saving procedure with morbidity rates of 1–3% and a mortality rate of <1% [21, 41, 42, 50].

Puncture and/or rupture of myocardium or coronary vessels; arrhythmias; myocardial infarction; subsequent infection or damage to surrounding structures such as the stomach, liver or lung; and re-accumulation are some of the rare complications.

In the case of a loculated effusion, the pericardiocentesis may not be effective in relieving the chamber compression, and more definitive surgery may be needed.

Paradoxical haemodynamic deterioration following relief of pericardial tamponade is sometimes encountered because of vasovagal effects with decreases in blood pressure and heart rate.

Pericardial decompression syndrome [21, 50] is defined as an unexpected decline in haemodynamic parameters associated with pulmonary oedema and global LV dysfunction. It usually occurs following surgical drainage of large, chronic effusions, but the incidence is rare <5%, and in most patients, there is no discernible reason for the ventricular dysfunction observed. The cause is unknown but may be related to increased interventricular interdependence. The sudden increase in preload and ventricular filling causes an abrupt increase in systolic wall stress, myocardial stunning and volume overload with acute left ventricular failure. Sudden increases in left ventricular end-diastolic pressures are transmitted to the pulmonary circulation with increases in pulmonary pressures and extravasation of fluid into the alveoli leading to pulmonary oedema.

4.1.4 Anaesthetic techniques

All patients require adequate peripheral access and standard ASA monitoring prior to any anaesthetic agent being administered [12, 21, 22, 32, 49, 51–54]. It may also be necessary to institute invasive monitoring via arterial and central venous lines when appropriate, ensuring that insertion of the lines does not delay definitive treatment.

The anaesthetic technique chosen will depend on the clinical condition and haemodynamic stability of the patient, any concomitant co-morbidities, the aetiology of the effusion and the procedure being performed. There are some advantages to performing general anaesthesia over local anaesthesia and sedation. Patient comfort

is improved, surgical and operating conditions are optimised and TOE may be employed to guide surgery and ensure adequate evacuation of the effusion [21].

Regardless of the technique chosen, the following haemodynamic goals should be adhered to increase cardiac output, maintain heart rate, maintain sinus rhythm, maintain systemic vascular resistance and left ventricular afterload as tamponade is a fixed output state and ensure an appropriate ventilatory strategy to decrease right atrial and pulmonary pressures. This will augment right atrial filling and decrease right ventricular afterload to promote forward flow through the lungs and maintain left-sided filling pressures from the pulmonary veins.

Local anaesthetic infiltration with supplemental sedation using ketamine, midazolam or fentanyl may be sufficient for pericardiocentesis and sub-xiphoid windows in the less stable patients who are co-operative. Midazolam is a potent, short-acting benzodiazepine that may be used to facilitate drainage procedures under local anaesthesia. Doses of 0.01–0.03 mg/kg may be given slowly and titrated to effect. Respiratory depressant effects are exaggerated with concomitant administration of opioids and other central nervous system depressant drugs.

If general anaesthetic is required, haemodynamic goals remain the same as above. Supplemental administration of fluid and/or blood products with vasopressor or inotropic infusions may be required to support cardiac output.

4.1.4.1 Intraoperatively

Induction of anaesthesia may be associated with severe hypotension and subsequent cardiovascular collapse. The suppression of sympathetic drive from induction agents such as propofol or thiopentone causes vasodilation and bradycardia. Coupled with the deleterious effects of mechanical ventilation as described above, induction of anaesthesia may cause cardiovascular collapse in patients at risk.

The avoidance of respiratory depressant drugs and positive pressure ventilation should be the technique of choice if possible. Spontaneous respiration with a volatile agent such as sevoflurane is ideal if tolerated by the patient. Conditions which may preclude inhalational induction include significant aspiration risk, morbid obesity, severe orthopnoea or an unco-operative patient [12].

Sevoflurane is a fluorinated methyl isopropyl ether anaesthetic agent that is non-pungent, causes minimal airway irritation and is well tolerated by awake, spontaneously breathing patients. It causes a dose-dependent decrease in mean arterial pressure by causing vasodilation and reducing systemic vascular resistance. At concentrations of more than 1.5 minimum alveolar concentration (MAC), increases in heart rate may occur. Nominal changes in cardiac output and stroke volume occur with careful titration. Infusions of vasopressor agents may be required to maintain blood pressures while acquiring adequate depth of anaesthesia with a volatile agent. Adequate depth of anaesthesia should be confirmed before any attempts at manipulation of the airway.

IV induction can be used for more stable patients with no evidence of tamponade. Surgical preparation and draping to facilitate emergency drainage is advisable if IV induction is to be used. IV induction agents need to be carefully selected. Ketamine and etomidate are recommended agents of choice as they will have the least vasodilatory effects.

Ketamine is a phencyclidine derivative that induces a dissociative anaesthetic state. The cardiovascular effects mimic those of sympathetic nervous system stimulation. Systemic blood pressure, heart rate and cardiac output are all increased after intravenous induction doses of 1–2 mg/kg which satisfy the haemodynamic goals required in patients with tamponade pathophysiology. Although there is a theoretical concern about increases in right ventricular afterload with ketamine, it

has been used safely in patients for both adjunctive sedation and general anaesthesia. Critically ill patients may respond with unexpected decreases in blood pressure and cardiac output, reflecting a depletion of catecholamine stores and the unmasking of ketamine's direct myocardial depressant effects. Caution must therefore be exercised with these patients. In this instance, it may be more appropriate to use local anaesthesia with supplemental sedation doses of ketamine instead.

Etomidate is a unique carboxylated imidazole-containing compound that exerts its anaesthetic effect by binding to the $GABA_A$ receptor and enhancing GABA binding. Myocardial depressant effects are minimal and contractility is preserved. Nominal changes in heart rate, stroke volume and cardiac output are noted after induction doses of 0.3 mg/kg. This superior cardiovascular stability profile makes etomidate the recommended IV induction agent for patients with little or no cardiovascular reserve.

Propofol is a substituted isopropyl phenol which causes rapid onset of anaesthesia at induction doses of 1.5–2.5 mg/kg when administered intravenously. Its mechanism of action is to modulate GABA receptors causing hyperpolarisation and inhibition of the postsynaptic cell membrane. Propofol causes decreases in systemic vascular resistance, negative inotropy and bradycardia. Vasodilation is mediated by the activation of protein kinase C, increased concentrations of nitric oxide and calcium channel-blocking effects at a cellular level. This effect is present on both sides of the vascular system with arterial- and veno-dilation causing decreases in preload and afterload. Propofol has a depressant effect on the heart rate by decreasing the baroreceptor response to a drop in blood pressure [51]. These effects are exaggerated in patients with tamponade, and the use of propofol at induction doses is not recommended.

Potent opioid agonists, such as fentanyl, are recommended for use in varying doses for both sedation and anaesthesia in patients with cardiac tamponade coming for drainage procedures. They exhibit superior cardiac stability when compared with morphine and are useful adjuncts to provide analgesia and reduce the dose of narcotics required.

Inotropic and vasopressor support should be anticipated, immediately available and used as needed to maintain haemodynamic goals at induction of anaesthesia. The positive inotropic effects of catecholamine-enhancing drugs may be limited in patients with tamponade [32]. This may be because patients are already operating at maximum sympathetic stimulation as a compensatory mechanism for the low cardiac output state. The choice of inotropic agent will depend on the patient's clinical condition. Those agents with inodilator properties may improve coronary perfusion initially, but no increase in other organ perfusion has been shown. Caution should be exercised and drugs titrated in at low doses with continuous assessment of haemodynamic response. Vasopressor agents are useful to maintain afterload but should be avoided in patients with normal blood pressure as their use may cause further decreases in cardiac output.

4.1.5 Airway management

Securing a definitive airway early and in the safest way possible is recommended. Endotracheal intubation allows for adequate control of ventilation and oxygenation. Mechanical ventilation may increase right ventricular afterload and decrease forward flow through the right ventricular outflow tract. This will in turn increase right ventricular end-diastolic pressures, worsen interventricular septal shift, further compromise left ventricular filling and negatively impact cardiac output. With an appropriate ventilatory strategy, however, the effect of mechanical ventilation can be minimised. The lowest possible inspiratory pressures and PEEP to maintain minute volume and oxygenation should be used [21].

The choice of endotracheal tube will depend on the procedure. For example, one-lung ventilation may be needed to facilitate surgery in thoracotomy and video-assisted thoracoscopic surgery (VATS). The time taken to insert a double-lumen tube and check its position may be detrimental in an unstable patient. Under these circumstances, placing a single-lumen tube with a bronchial blocker may be more appropriate.

If the patient is very unstable, the safest option may be to perform a sub-xiphoid window initially under local anaesthetic to relieve the tamponade. General anaesthesia may then be more safely induced.

4.1.6 Maintenance of anaesthesia

General anaesthesia care including intraoperative acid–base management, correction of electrolyte abnormalities, temperature and glucose control is required as for all high-risk patients coming for a procedure. These patients may present in a decompensated state which negates pre-operative optimisation of their condition. In the case of any emergency surgery, ongoing resuscitation is required even after the patient has been safely anaesthetised.

IV opioids, propofol, ketamine and volatile agents can all be used if they are tolerated and the haemodynamics remain stable. Careful titration of agents rather than specified doses should be employed. Muscle relaxants should only be used once the patient can tolerate positive pressure ventilation. The choice of neuromuscular-blocking agent will depend on the onset and duration of action required. The newer agents from both the aminosteroid and benzylisoquinoline groups of intermediate-acting neuromuscular-blocking agents are appropriate as they do not exhibit significant cardiovascular effects.

Continuous infusions of inotropes and pressors may be needed and must be continued on a case-by-case basis.

Because of surgical handling of the heart, intraoperative arrhythmias are common and should be anticipated. A defibrillator and antiarrhythmic drugs should be immediately available and standard Advanced Cardiac Life Support (ACLS) algorithms employed to manage peri-operative arrhythmias.

Transfusion triggers and the need for intraoperative administration of blood and clotting factors should be discussed and decided by the multidisciplinary team before induction. Ongoing fluid and blood product resuscitation may be required. Adequate haemoglobin levels to ensure optimal oxygen-carrying capacity is needed and should be individualised per case. Peri-operative monitoring of the patient's coagulation status with thromboelastography (TEG) is advised in order to guide targeted administration of plasma products and pharmacological agents that may be required.

Because intrinsic myocardial function is generally preserved in pericardial effusion and tamponade, once the tamponade is relieved, there is usually a dramatic improvement in haemodynamics. At this point, vasopressor and inotropic infusions should be reviewed and weaned if appropriate. The use of vasodilator therapy is controversial and only indicated if there is associated systemic hypertension and elevated systemic vascular resistance [32].

4.1.6.1 Post-operatively

Patients should be transferred to a high-dependency unit for continuation of care. Ongoing monitoring is necessary to assess for recurrence of tamponade, ongoing bleeding and continuation of cardiovascular and ventilatory support.

4.2 Pericardiectomy for constrictive pericarditis

Pericardiectomy is the only definitive treatment for established constriction, and most studies suggest that resection should be as complete as technically feasible [49, 54–57]. A retrospective review by Nozohoor et al. showed that radical pericardiectomy was associated with improved functional status and 10-year survival rates of 94% compared to 55% with subtotal pericardiectomy [55]. Bozbuga et al. also showed that performing early radical resection in combination with appropriate chemotherapeutic agents translated into the best outcomes in patients with tuberculous disease [56].

4.2.1 Types of pericardiectomy

Pericardial stripping is carried out via sternotomy or lateral thoracotomy with or without cardiopulmonary bypass support [49]. Total resection is extremely difficult to perform via a thoracotomy, and median sternotomy is the preferred approach to provide definitive resection and optimise benefit [49, 55].

Partial pericardiectomy is defined as incomplete decortication of one or both ventricles because of severe myo-pericardial adhesions.

Total pericardiectomy is when wide excision of the pericardium is performed. The anatomical borders consist of the phrenic nerves posteriorly, the great vessels and intra-pericardial portion of the superior vena cava/right atrial junction superiorly and the diaphragm and inferior vena cava/right atrial junction inferiorly.

Radical resection is the removal of the pericardium including the anterolateral and diaphragmatic surfaces of the left and right ventricles with careful dissection posterior to the phrenic nerves to leave the left and right phrenic pedicles intact.

Epicardial involvement may be missed at surgery leading to persistent constrictive physiology post-operatively. Reoperation to remove the diseased epicardium or visceral pericardium may be required [49].

The need for cardiopulmonary bypass (CPB) should be individualised per case. Studies suggest that mortality rates are higher in patients who required CPB, but data may be skewed because its use is usually reserved for patients with a poor pre-operative status. Other indications include any coexisting cardiac conditions that may require intervention, previous cardiac surgery or partial pericardiectomy, a heavily calcified pericardial "cocoon" encasing all four cardiac chambers, post mediastinal irradiation and unintentional surgical damage to cardiac structures intraoperatively [49].

Persistent constrictive physiology and abnormal diastolic filling patterns may be seen in a percentage of patients even after successful surgery. In conjunction with symptomatic improvement, normalisation of pressure-volume loops and echocardiographic findings post pericardiectomy are used as markers of successful resection [49, 58].

4.2.2 Complications associated with pericardiectomy

Pericardiectomy is a very technically challenging operation with significant morbidity and mortality performed in high-risk patients [49, 54–57, 59]. Intraoperative complications may contribute significantly to poor outcomes. Sufficient pre-operative preparation prior to induction of anaesthesia is paramount to ensuring patient safety, and a multidisciplinary team approach should be used.

Massive haemorrhage should be anticipated with contingency plans in place before the administration of any anaesthetic agents. Timeous activation of a massive transfusion protocol is necessary. Cardiopulmonary bypass support should be immediately available with cannulation of femoral vessels under local anaesthesia

in select patients with severe or decompensated disease. Appropriate IV access and invasive monitoring should be established, and inotropic or vasopressor infusions drawn up and running at the time of induction of anaesthesia. Damage to underlying epicardium, myocardium and coronary vessels poses the most risk to patients contributing to an overall mortality of about 7–18% [4]. Arrhythmias are common and prompt management as per the latest ACLS guidelines.

Low cardiac output syndrome, defined as a cardiac index of <2.2 l/min/m, will result in a subset of patients with chronic constrictive pericarditis [49, 60]. This is a form of acute cardiac failure, and there are a number of suggested mechanisms. The underlying myocardium may be affected by prolonged constriction causing myocardial disuse atrophy; residual constriction from partial or incomplete removal of the epicardial layer results in persistent constriction; the disease process itself may affect the myocardium; prolonged abnormal diastolic filling leads to architectural changes with remodelling of the ventricles; worsening of tricuspid regurgitation with progressive right ventricular dysfunction causes volume overload and right ventricular failure; and elongation of the papillary muscles leads to significant mitral regurgitation and left ventricular failure post relief of the constriction.

The incidence of low cardiac output syndrome post pericardiectomy is 28% with an associated mortality rate of up to 70% [60, 61]. Patients demonstrating prolonged symptomatic disease are at greatest risk [60]. This complication should be anticipated peri-operatively and managed with the appropriate haemodynamic and ventilatory support. Some studies suggest that levosimendan may be a pharmacological agent of choice [60].

4.2.3 Anaesthetic management

Intraoperative haemodynamic goals are to maintain heart rate, maintain systemic vascular resistance, optimise venous return and support myocardial contractility [12, 21, 22, 32, 49, 51–54, 60]. General anaesthesia care including intraoperative acid–base management, correction of electrolyte abnormalities, temperature and glucose control is required and will apply to these patients as mentioned above for pericardial tamponade.

Because CP is a fixed output state, patients have an increased reliance on heart rate and systemic vascular resistance to maintain systolic blood pressures and organ perfusion. Optimisation of preload and venous return preinduction are important to compensate for the decreased filling of the cardiac chambers. The consequences of possible fluid overload must always be considered, and these patients require dynamic assessment of fluid status as they too operate at the lower end of the Frank-Starling curve. This places them at increased risk for volume overload and low cardiac output syndrome, especially after relief of the constriction.

A balanced anaesthetic technique ensuring haemodynamic goals is necessary to avoid sudden decompensation at induction of anaesthesia [54, 60]. Ketamine is often recommended as the anaesthetic induction agent of choice because of its sympathomimetic effects on the cardiovascular system. Midazolam, etomidate and potent opioids such as fentanyl have also been safely used for induction of anaesthesia. Securing a definitive airway is recommended, and the use of muscle relaxants can be used early on, bearing in mind the possible deleterious effects of mechanical ventilation. The institution of positive pressure ventilation may compromise right heart chamber filling, and appropriate settings are needed to optimise ventilation, oxygenation and haemodynamics.

It is highly recommended that inotropic and/or vasopressor infusions are drawn up and running at the time of induction. These should be continued and titrated

throughout surgery and continued into the post-operative period in anticipation for a persistent low cardiac output state, especially in at-risk patients.

Anticipation for massive blood loss, both acute and ongoing, is of vital importance as mentioned above. Monitoring with heightened intraoperative vigilance, regular blood gas and TEG analysis will guide transfusion of blood, clotting factors and pharmacological agents that may be required.

Damage to cardiac structures may lead to massive sudden haemorrhage, myocardial ischaemia or significant arrhythmias. Appropriate management must be instituted immediately and may include the need for cardiopulmonary bypass if conventional surgical and medical management fail.

The need for elective cardiopulmonary bypass support will depend on the pre-operative condition of the patient, the aetiology of the CP and the underlying myocardial function. The decision for the adjunctive use of bypass should be discussed and decided upon during the multidisciplinary team discussion prior to surgery. If deemed necessary, the femoral vessels should be prepped and cannulated under local anaesthesia prior to induction of anaesthesia.

Post-operatively, patients should be transferred to a high-dependency unit for continuation of care. Ongoing monitoring for persistent bleeding and low cardiac output syndrome is necessary as well as ongoing haemodynamic and ventilatory support.

4.2.4 Predictors of outcome following pericardiectomy

Early post-operative mortality rates are in the region of 18% [46, 49, 56, 57, 59]. Peri-operative mortality is highly dependent on the patient's pre-operative NYHA status [56].

Predictors of poor long-term outcome include the aetiology of the disease, previous radiation, renal dysfunction, hyponatraemia, old age, low pre-operative left ventricular ejection fraction, right ventricular dilatation, elevated pulmonary pressures and the severity of tricuspid regurgitation [46, 49, 57, 59].

Because of the high peri-operative morbidity and mortality, patients with mild or very advanced disease, renal failure or post-radiation constrictive pericarditis should be evaluated very carefully pre-operatively as the risk of pericardiectomy may outweigh the benefits.

5. Conclusion

Patients with 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. 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 high-risk patients in the safest way possible.

An appreciation of how the chosen anaesthetic technique may influence the patient's haemodynamic state is necessary to avoid unwanted anaesthetic complications. This includes a thorough understanding of the effect of pharmacological agents as well as how mechanical ventilation may influence haemodynamics. The potential risk and complications of the surgical procedure should be examined and discussed with the multidisciplinary team pre-operatively in order to ensure adequate preparation for any potentially catastrophic events. These patients are best cared for by experienced peri-operative teams who have this appreciation.

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