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Echocardiographic approach to cardiac tamponade in critically ill patients

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ABSTRACT

Cardiac tamponade should be considered in a critically ill patient in whom the cause of haemodynamic shock is unclear. When considering tamponade, transthoracic echocardiography plays an essential role and is the initial investigation of choice. Diagnostic sensitivity of transthoracic echocardiography is dependent on image quality, and in some cases a transoesophageal approach may be required to confirm the diagnosis. Knowledge of the pathophysiology and echocardiographic features of cardiac tamponade are essential for the practicing Intensivist. This review presents an approach to the recognition, diagnosis, and treatment of cardiac tamponade in critically ill patients.

Contents

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1. Introduction

Cardiac tamponade is a clinical syndrome which occurs due to accumulation of fluid in the pericardial space, thus resulting in reduced ventricular filling and subsequent haemodynamic compromise. Tamponade should be considered in a critically ill patient in whom the cause of haemodynamic shock is unclear. In order to confirm the diagnosis, echocardiography is the investigation of choice [1-3]. Occasionally tamponade is not suspected, either because hypotension is attributed to another cause or blood pressure is maintained despite a reduced cardiac output. In addition, transthoracic echocardiography (TTE) may not confirm the diagnosis, with sensitivity depending on the image quality. Knowledge of the pathophysiology and echocardiographic features of cardiac tamponade are essential for the practicing Intensivist. This review presents an approach to recognising, diagnosing, and treating cardiac tamponade in critically ill patients.

2. Causes of cardiac tamponade

Accumulation of fluid within the pericardial space is a common complication of pericardial disease [4]. An effusion of any aetiology has the potential to cause cardiac tamponade, with the fluid either being of sufficient volume or accumulating rapidly enough to result in haemodynamic compromise [3,5]. Table 1 shows a breakdown of the causes of cardiac tamponade relevant to intensive care medicine.

3. Clinical features

Pericardial effusions that develop slowly may be remarkably asymptomatic, while a rapidly developing effusion of much smaller size can present with tamponade [41]. Most clinical features of tamponade are non-specific [42], and the classic clinical signs may be difficult to appreciate in the critically ill patient. At the bedside the patient may be tachypnoeic if self-ventilating. Tachycardia, elevated central venous pressure (CVP), reduced peripheral pulses, and pulsus paradoxicus (an inspiratory fall > 10 mm Hg in systolic blood pressure) are often present, but are not specific to tamponade physiology [3,42] (Table 2). Tamponade should be considered when there are signs of a reduced cardiac output: hypotension, reduced peripheral perfusion, elevated serum lactate and decreased urine output. ECG changes may demonstrate reduced QRS and T wave voltage and electrical alternans [43]. In the presence of a large global pericardial effusion, plain chest radiology can demonstrate cardiomegaly. Computed tomography (CT) or cardiac magnetic resonance (CMR) can confirm the diagnosis of pericardial effusion in specific cases [44,45], but transport risks limit their use in unstable critically ill patients.

4. Pathophysiology of cardiac tamponade

The haemodynamics of cardiac tamponade are governed by complex interactions between the pericardium and the cardiac chambers and their relationship to each other throughout the respiratory and cardiac cycles. It is helpful to begin with an explanation of the following terms:

1. Pericardial restraint
   The pericardium is a thick fibrous membrane designed to accommodate 15–35 ml of pericardial fluid. As fluid collects in the pericardial space pericardial pressure rises and increases abruptly when the volume rises above 150–200 ml. The cardiac chambers start to become compressed as cardiac volume competes with pericardial fluid for space within the fixed parietal pericardium. In cardiac tamponade cardiac volume can only be maintained if there is a parallel rise in systemic and pulmonary venous pressure. As tamponade progresses the pericardial, right and left ventricular pressures equilibrate usually at 15–30 mm Hg [4,46].
2. Transmural pressure
   Transmural pressure is the effective pressure that distends or fills each cardiac chamber. It is defined as intracardiac pressure minus pericardial pressure. Normally there is a close relationship between intrapleural and intrapericardial pressures and both pressures fall equally during inspiration. In tamponade, because intrapericardial pressure has increased to the level of ventricular diastolic pressures it falls much less than intrapleural pressure during inspiration. Normally RA transmural pressure is positive (and higher than RA

Table 1
Causes of cardiac tamponade in critically ill patients [2, 6-40].

More common ICU causes	Less common
Chest trauma[a] – Blunt and penetrating injuries [14,37,38]	Infection[a] – Bacterial: Tuberculosis [17], *Coxiella burnetii* [26], others (rare) [22,24,34] – Viral: EBV, CMV, enteroviruses, HIV [2,25] – Fungal: very rare [27] – Parasitic: very rare [18]
Interventional cardiology procedures[a] – Permanent pacemaker insertion [10,35] – Temporary pacing wire insertion [13] – Epicardial pacing wire removal [30] – Percutaneous angiography/angioplasty/valve procedures [20,31] – Cardiac ablation procedures [20,31] – Cardiac biopsy [20]	Inflammatory and autoimmune – Systemic lupus erythematosus [36] – Rheumatoid arthritis [16] – Behcet syndrome [9,39]
Myocardial infarction – Ventricular free wall rupture [28]	Neoplastic[a] – Primary or secondary malignancy [11,31,33]
Aortic pathology[a] – Type A aortic dissection [7]	Metabolic – Uraemia [40] – Myxoedema [6]
Cardiac surgery[a] – Post-operative bleeding [32]	Idiopathic [31]
Central venous catheters[a] – Free wall perforation on Insertion [12,29] – Erosion of free wall (parenteral nutrition) [15]	Drugs – Warfarin and New oral anticoagulants (NOAC's) [8,21,23]
Extra-corporeal life support cannulation[a] [19]	
Other causes of tamponade-like physiology	
Effusive-constrictive pericarditis Pneumopericardium Extrinsic compression (eg mediastinal mass or haematoma, large pleural effusion)	

[a] May cause tamponade from localised chamber compression or global pericardial effusion.

Table 2
Conditions other than cardiac tamponade which can cause tachycardia, elevated central venous pressure, and pulsus paradoxicus in critically ill patients.

Cardiac causes	Non-cardiac causes
Cardiac tamponade	Severe asthma
Constrictive pericarditis	Tension pneumothorax
Restrictive cardiomyopathy	Superior vena cava obstruction
Acute right heart failure (eg pulmonary embolism)	Extreme obesity

pressure) which favours filling and expansion of the RA and RV [4]. The rising pericardial pressure in tamponade progressively reduces transmural pressure of the right and later the left sided chambers. In addition, in tamponade because of the relative uncoupling of intrapleural and intrapericardial pressures, there are phasic increases and decreases in transmural pressure during the respiratory cycle. Thus tamponade pathophysiology is characterised by increased filling of the right sided cardiac chambers during inspiration at the expense of filling of the left sided cardiac chambers. During expiration the opposite happens.

3. Parallel interventricular dependence (with septal shift)
   The distension of one ventricle alters the shape of the other ventricle as a result of marked shift of the interventricular septum. This is due, in part, to shared encircling muscle bands and an intact pericardium. Normally during spontaneous inspiration, right ventricular volume increases, slightly pushing the interventricular septum towards the left ventricle [47]. Under conditions of tamponade pathophysiology, this effect is exaggerated, and filling of the right sided chambers during inspiration is dependent on shifting of the interventricular septum into the LV. The consequence is decreased filling of the left side of the heart during inspiration. This diastolic interventricular interaction is manifest clinically as pulsus paradoxus [44,48].
4. Chamber compliance
   Chamber compliance is the relationship between atrial/ventricular volume and pressure, often described by pressure volume curves [46,49]. The increased filling pressures that develop in tamponade result in cardiac chambers operating on steep pressure volume curves, in effect a form of diastolic dysfunction. The structural properties of cardiac muscle influence chamber compliance and thus during tamponade, the thinner walled RA and RV demonstrate significant compression before the left ventricle does [46].

4.1. Normal respiratory cycle changes in intracardiac chambers and pericardium

During normal inspiration intrapleural and pericardial pressures fall to the same extent. This augments the positive transmural gradient and favours expansion and filling of the RA and RV.

4.2. Respiratory cycle changes in intracardiac chambers and pericardium in tamponade

In cardiac tamponade, the rising intrapericardial pressures do not follow intrapleural pressures so faithfully during inspiration. Transmural pressure, initially on the right and later the left side of the heart, progressively reduces and may become phasically negative. The compensatory increase in venous filling pressures equilibrates with pericardial pressure, reducing the transmural distending pressure of all chambers. Eventually, the phasic inspiratory increase in RA and RV filling and size, with simultaneous decrease in LV filling and size (interventricular shift) becomes a mainstay of cardiac filling and cardiac output. This is critical tamponade and is associated with a huge decrease in cardiac output.

4.3. Normal cardiac cycle changes in intracardiac chambers and pericardium

The normal pattern of right atrial filling is represented by an *a* wave (atrial contraction) and a *v* wave (ventricular contraction) on atrial pressure curves. The *x* descent following the *a* wave represents relaxation of the atrium and the downward pulling of the tricuspid annulus by right ventricular ejection. The *y* descent follows the *v* wave as the tricuspid valve opens and the RA empties into the RV. Normally one surge of systemic venous return occurs during the *x* descent and a second surge occurs during the *y* descent.

4.4. Cardiac cycle changes in intracardiac chambers and pericardium in tamponade

Cardiac tamponade alters the dynamics of cardiac filling, causing cardiac cycle related changes in intracardiac pressures and subsequent fluctuations in the transmural pressure gradient. During cardiac tamponade venous caval flow is reduced and ventricular filling pressures are increased. Right ventricular ejection reduces ventricular volume by expelling blood, causing a transient reduction in intrapericardial pressure and thereby transiently increasing transmural pressure. Atrial filling is simultaneously augmented by the descent of the tricuspid valve towards the ventricular apex. Thus there is a normal *x* descent in atrial pressure curve. However, the normal *y* descent of pressure drop in early diastole is usually absent as ventricular filling pressure is high, resisting ventricular filling [49]. As a result, right heart filling becomes monophasic. Right ventricular diastolic collapse occurs when intrapericardial pressure exceeds intracavitary pressure, causing a transient reversal in transmural pressure [49].

4.5. Compensatory mechanisms

Efforts to protect cardiac output and blood pressure include beta-adrenergically mediated tachycardia and sympathetic-mediated vasoconstriction. The renin-angiotension system is activated causing increased fluid retention. Elevated central venous pressures improves diastolic filling against intrapericardial pressure. There is no change in atrial naturetic peptide levels as the compressed heart does not stretch [49].

5. Cardiac tamponade in the Intensive Care Unit

5.1. Clinical features in critically ill patients

Cardiac tamponade can present as acute haemodynamic shock or more insidiously as organ failure due to reduced cardiac output e.g. renal failure, ischaemic hepatitis or mesenteric ischaemia. It is a clinical diagnosis which may be confirmed by imaging including echocardiography. History is important: for example, in the setting of recent pacemaker lead insertion, renal failure or penetrating chest trauma the presence of hypotension mandates a search for tamponade.

However, in the critically ill patient clinical assessment (including pulsus paradoxus, changes in CVP and cardiac output) can be confounded by the use of vasoactive medications and mechanical circulatory support devices (including intra-aortic balloon counter-pulsation, extracorporeal life support, and ventricular assist devices). Echocardiography may be inconclusive for features of tamponade, and the effects of other pathologies in critically ill patients (such as bronchospasm, significant pleural effusion, respiratory distress, and arrhythmias) can add to the difficulty in interpreting the Doppler findings of tamponade [66]. An alternative method for making the diagnosis is to demonstrate equalization of diastolic filling pressures and a low central venous oxygen saturation (ScvO2) with invasive haemodynamic monitoring (the pulmonary artery catheter).

5.2. Mechanical ventilation

Cardiovascular compromise can be worsened by mechanical ventilation (IPPV and PEEP) in cardiac tamponade. The positive changes in intrapleural pressures introduced by IPPV are transmitted to the pericardial cavity, further decreasing transmural pressure [50].

The characteristic respiratory variations in transmitral flow velocities seen with tamponade in a spontaneously breathing patient are markedly attenuated during IPPV [51]. However, consistent with the pattern seen in spontaneously breathing patients with tamponade, there will be an overall decrease in the amplitude of the flow velocities during IPPV compared to values obtained in individuals without tamponade.

Chest radiograph may reveal an enlarged cardiac silhouette with a globular heart shape. Sequential chest x-rays in critically ill patients with invasive lines, pacing wires, pulmonary artery catheter or extracorporeal membrane oxygenation (ECMO) cannulae in situ can be useful. The distance between the wire or catheter and the right heart border should be noted: an increase in this distance may be indicative of fluid or blood clot lying against RA/RV.

5.3. Localised tamponade

The absence of classic hemodynamic signs of tamponade when attributable to loculated pericardial haematoma, for example after open heart surgery with an open pericardium, is well described. Loculated clots are difficult to detect with TTE because acoustic access is limited and the clots often are located in the far field of the beam, reducing discrimination between haematoma and surrounding soft tissue. Transoesophageal echocardiography (TOE) provides an alternative acoustic window and superior image quality, making it ideal for detection of pericardial haematoma [52].

5.4. Extracorporeal membrane oxygenation (ECMO)

The use of systemic anticoagulation makes patients on ECMO susceptible to bleeding and potential pericardial collections. However, even in the presence of a pericardial effusion the clinical and echocardiographic diagnosis of tamponade remains challenging, especially during veno-arterial ECMO (VA ECMO) when the modification of RA and RV pressures may preclude appropriate analysis of chamber collapse. One clinical indicator of tamponade during peripheral VA ECMO will be a drop in the amplitude of the arterial waveform (signalling a reduction or loss of native cardiac ejection), and a paradoxical increase in PaO2 from a right sided arterial line as the pulmonary circulation becomes bypassed.

6. Echocardiographic features during cardiac tamponade

6.1. Pericardial effusion

Using 2-Dimensional (2-D) transthoracic echocardiography (TTE), a pericardial effusion appears as an echo-free space adjacent to the heart (Image 1). Parasternal long and short axis views usually allow adequate visualisation of a pericardial effusion. However, in the critically ill patient with haemodynamic collapse, the window of first choice is the subcostal view (Image 2). A normal pericardial cavity contains just 5–10 ml of fluid, acting as a physiological potential space. Separation of the two pericardial layers can be seen once pericardial fluid volume exceeds 15–35 ml [53]. The echocardiographic appearance of the effusion may point to its underlying cause (Images 3, 4, Video 1). In the parasternal long axis view, a pericardial fat pad may be seen anteriorly in some patients, and can be mistaken for an effusion. A hyper-echoic, granular appearance, and movement in synchrony with the external cardiac border suggests a fat pad rather than pericardial effusion.

6.2. Effusion size

A semi-quantitative evaluation of the effusion size and volume should be made. Although fluid volume cannot alone predict haemodynamic tolerance, documenting the size allows serial assessments and comparisons to be made. The diameter should be marked as the maximal diameter during diastole, using the 'leading edge to leading edge' principle. An effusion <10 mm can be graded as small (Images 5, 6), from 10–20 mm moderate (Images 7, 8), and >20 mm large (Images 9, 10, Videos 2–4) [5,54].

6.3. Two-dimensional features of cardiac tamponade

Right atrial free wall collapse or "inversion", an extremely sensitive marker of cardiac tamponade, is best visualised in the apical 4-chamber or subcostal view. In situations of tamponade RA wall inversion occurs in late diastole with a variable continuation into ventricular systole. Assessing the duration of RA inversion, using the right atrial inversion time index (the duration of inversion/cardiac cycle length), with a cut-off value of .34, increases specificity and predictive value without a loss of sensitivity [55-57]. RV wall collapse in early to mid-diastole is also a specific sign of cardiac tamponade [58,59]. It occurs initially at the RV outflow tract region, but with further increase in intra-pericardial pressure collapse of the basal RV develops. RV collapse can be seen in subcostal and parasternal long and short axis views [5]. Examples of RA and RV collapse in patients with cardiac tamponade can be seen in Images 11–14 and Videos 5–6.

Tamponade pathophysiology, as discussed above, will result in the characteristic septal motion signs due to heart-lung interaction. Septal shift towards the LV in inspiration, and towards the RV in expiration, may be seen [60,61]. This effect is best appreciated in the parasternal long axis view, and also using M-mode. With a large volume effusion causing tamponade, the heart may exhibit a classic 'swinging' motion within the pericardial cavity [62] (responsible for the ECG finding of electrical alternans), seen in the parasternal long axis, apical 4-chamber, and subcostal views.

Two-dimensional TTE assessment is not complete without examination of the inferior vena cava (IVC) using the subcostal window. Dilated IVC, with absence of normal IVC respiratory collapse (Image 15, Video 7), also termed IVC plethora, is a sensitive but not specific sign of tamponade in a spontaneously breathing patient [63]. In addition to the 2D features of IVC plethora, pulsed wave Doppler interrogation of the hepatic vein may demonstrate hepatic diastolic flow reversal in expiration.

6.4. Doppler echocardiography

Doppler features of cardiac tamponade are more sensitive than 2-D echo features [64,65]. Heart-lung interactions in cardiac tamponade lead to respiratory variations in trans-valvular flow. Pulsed wave Doppler measures tricuspid and mitral inflow during diastole (Images 16, 17). Tricuspid inflow velocity increases during spontaneous inspiration, reflected by an increase in Doppler velocity. The reverse occurs in expiration. With spontaneous respiration, an inspiratory rise in tricuspid peak E velocity greater than 40–50% is consistent with tamponade pathophysiology (Image 18) [64,65]. Decrease in mitral peak E velocity more than 25–40% with inspiration also indicates tamponade (Image 19) [64,65].

Doppler echocardiography does however require a higher level of expertise to perform and interpret appropriately. Its use in critical care may be limited by the technical specifications of the machine available, and the use of mechanical ventilation which renders Doppler interrogation inaccurate. In addition, the intercept angle and placement of the Doppler sample, crucial for accurate measurements, may be significantly displaced in a patient with respiratory distress.

7. Specific situations

7.1. Cardiac tamponade post-op cardiac surgery

When haemodynamic shock occurs in this patient group, a very high index of suspicion for tamponade should be maintained. Obtaining adequate transthoracic echo windows can be technically challenging, therefore the Intensivist should have a low threshold for performing trans-oesophageal echocardiography (TOE) [66]. Localised haematoma causing selective chamber compression is more common in post-sternotomy patients, meaning that haemodynamic effects can be variable. For example, haematoma causing ventricular compression might lead to more pronounced and rapid haemodynamic deterioration than a haematoma compressing the right or left atrium. In cases of localised atrial compression, the haemodynamic effects may present more gradually with elevation of filling pressures, and progressive increase in vasoactive supports despite adequate filling.

Mediastinal haematoma causing isolated compression of the superior vena cava (SVC) can also develop relatively insidiously following cardiac surgery [67]. An elevation of central venous pressure may initially be attributed to fluid overload or RV dysfunction due to the absence of significant haemodynamic deterioration. TOE is required to rule out a loculated haematoma external to the SVC, and avoid the potentially life-threatening formation of cerebral and laryngeal oedema similar to SVC syndrome [67].

It is important to note that posterior haematoma (for example retro-apical haematoma which classically occurs following cardiac surgery) may be missed by TTE even when image quality appears adequate, due to its location in the far field of the transthoracic ultrasound beam. For this reason, when there is a suspicion of tamponade in the cardiac surgery patient a negative TTE should prompt urgent TOE. [68] (Images 20–23, Videos 8–11).

7.2. Respiratory variation in Doppler inflow: other situations

Doppler features of cardiac tamponade have limitations. Respiratory variations in inflow patterns and stroke volume occur with several disease states including: obesity, severe hypovolaemia, chronic obstructive lung disease, pulmonary embolism, and right heart failure. In addition, Doppler echocardiography features of tamponade have been validated only in spontaneously breathing patients; they are not reliable in critically ill patients who are mechanically ventilated [50,69].

7.3. Large pleural effusions

Differentiating between a pericardial and pleural effusion is important, and in some patients the two may co-exist. The presence of large volume, bilateral pleural effusions has also been reported to produce features mimicking cardiac tamponade [70,71]. In the parasternal long axis view, a pericardial effusion can be seen as an echo-free space posterior to the heart, passing anterior to the descending thoracic aorta (Image 24). In contrast, a pleural effusion extends posterior to the descending aorta (Image 25). Pericardial fluid will usually, unless loculated or small volume, be circumferential. Fluid visible posterior to the left atrium is usually pleural, as the left atrium is largely an extra-pericardial structure. In the subcostal view, care should be taken not to mistake large volume ascites for a pericardial effusion.

Large left-sided pleural effusion may cause significant compression of cardiac chambers. Although not common, this can result in impaired cardiac filling and a tamponade-like pathophysiology [72,73]. Treatment involves urgent large volume thoracocentesis.

7.4. Chronic changes in right ventricular pressure and wall thickness

Reduction in RV free wall compliance, such as that occurring in chronic RV hypertrophy, means that the relatively stiff RV may resist diastolic collapse. When cardiac tamponade develops in patients with chronically elevated right heart pressures (eg pulmonary hypertension), collapse of right sided chambers may not occur. In these situations the LA and LV become the low pressure chambers, with resulting LV collapse [74].

8. Management of cardiac tamponade

8.1. Supportive

8.1.1. Fluid therapy

Fluid loading can help stabilise or delay haemodynamic collapse while definitive drainage is prepared [75]. Volume expansion will increase mean arterial pressure and cardiac output in patients who are hypovolaemic, but can be detrimental in others [76]. Fluid status should be assessed and fluid challenge administered if appropriate. Fluid infusion should be discontinued if haemodynamic deterioration occurs.

8.1.2. Vasopressor medication

Adrenergic stimulation with inotropic agents (such as adrenaline and dobutamine) in the setting of cardiac tamponade can be deleterious, causing tachycardia which further limits ventricular filling [77]. If vasoactive support is required, vasopressors (such as Noradrenaline) which increase systemic vascular resistance and thus mean arterial pressure, are the medication of choice pending pericardial drainage [78].

8.2. Definitive

8.2.1. Pericardiocentesis guided by echocardiography

Although previously done as a 'blind' procedure in emergent situations, bedside pericardiocentesis is now almost always performed under real-time image guidance. This may be done using transthoracic echocardiography [79], or alternatively using fluoroscopic techniques in the cardiac catheterisation laboratory [2]. Some centres may use a combination of both techniques. In resource poor settings, where TTE or fluoroscopy is not immediately available, aspiration may be guided using ECG connected to the pericardiocentesis needle. In this case an abrupt change in ECG (with an injury pattern such as ST elevation) signals myocardial contact, prompting slow needle withdrawal until the ECG returns to normal.

Echocardiography has the advantage of identifying the distribution and size of the effusion, therefore guiding the best approach for drainage (most commonly the subxiphoid or apical approach) [80]. When adopting a percutaneous approach to drainage, a pigtail catheter is inserted using a standard seldinger technique. Echo guidance allows direct vision of the needle within the pericardial space; at this point agitated saline may be injected to further confirm position [80]. Guidewire position can also be confirmed under direct vision prior to dilating. Windows used for subxiphoid and apical approaches to drainage are shown in Images 26 and 27, respectively.

8.2.2. Surgical decompression

When contra-indications to percutaneous drainage exist (for example coagulopathy) or when an effusion is loculated, then surgical decompression is indicated. Effusions that are not amenable to percutaneous drainage (posterior location) will also require a surgical approach. In the setting of trauma, a percutaneous approach may be considered as a temporising measure [81], but should not delay definitive surgical intervention.

Surgical approaches to pericardial drainage will depend on the underlying cause of effusion. For example, emergency or resuscitative thoracotomy may be required in the setting of penetrating chest trauma [82]; whereas re-sternotomy is the approach of choice in the setting of tamponade post-cardiac surgery [83].

9. Conclusion

Echocardiography is an essential tool for the evaluation of haemodynamically unstable critically ill patients. Intensivists should understand the pathophysiological mechanisms of cardiac tamponade, and should have some knowledge of its echocardiographic features. In most patients transthoracic echo can confirm the diagnosis, but in certain circumstances transoesophageal echo may be required. When tamponade is confirmed echo can guide the definitive treatment approach, either by pericardiocentesis or definitive surgical decompression.

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Competing interests

None.

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