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IMAGES IN ANESTHESIA

Perioperative cardiovascular rounds: pericardial tamponade

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Echocardiography plays a significant role in the diagnosis of pericardial effusion and its related complications. To illustrate this issue, we were involved in the care of a 55-year-old male with liver cirrhosis who presented with pleuritic chest discomfort of four days' duration. His electrocardiogram (ECG) showed diffuse ST segment elevation and a shortened PR interval (Fig. 1). Echocardiography showed a pericardial effusion of 18–20 mm without compression of the right ventricle but with slight compression of the right atrium. The left ventricular ejection fraction was normal. Initially, he was suspected as having viral pericarditis. However, on the evening of admission, the patient developed hypotension and shock and was transferred to the intensive care unit. Fluid resuscitation was initiated, and broad-spectrum antibiotics and vasopressors were administered. Blood cultures grew coagulase-negative staphylococci.

Over the ensuing days, the patient's condition deteriorated as he developed progressive respiratory failure, renal failure, and liver dysfunction. Repeat echocardiograms showed persistent pericardial effusion with more significant right atrial compression. A pericardial drain was introduced under echocardiographic guidance, and 500 ml of thick bloody fluid were removed. Vasopressor requirements decreased significantly after the procedure. Pericardial fluid showed numerous polymorphonuclear leukocytes and grew coagulase-negative staphylococci. Despite continued pericardial drainage, the patient's condition further deteriorated. Based on his moribund condition and poor prognosis, a surgical approach was contraindicated; he died 14 days after admission. Autopsy findings are illustrated in Fig. 2.

Purulent pericarditis is a rare entity that is encountered more often in children and immunocompromised patients.[1] Infection of the pericardium occurs via either contiguous infection or hematogenous spread. *Staphylococcus aureus* is the most frequently encountered organism. The diagnosis is often delayed, as it can only be confirmed by pericardiocentesis, which is most safely accomplished under echocardiographic guidance. Treatment includes intravenous antibiotics and adequate drainage of the pericardium. Reports suggest a role for the infusion of intra-pericardial streptokinase to facilitate drainage, but a thoracotomy with a pericardial window should be attempted if drainage is incomplete. Mortality remains high (40%) even in treated patients.[2]

Infected pericardial effusion can lead to acute hemodynamic instability through several mechanisms, including obstruction, ventricular dysfunction, and vasoplegia. The most common cause of related hemodynamic instability is obstruction causing tamponade (Fig. 3).[3] Tamponade occurs when the pericardial pressure rises

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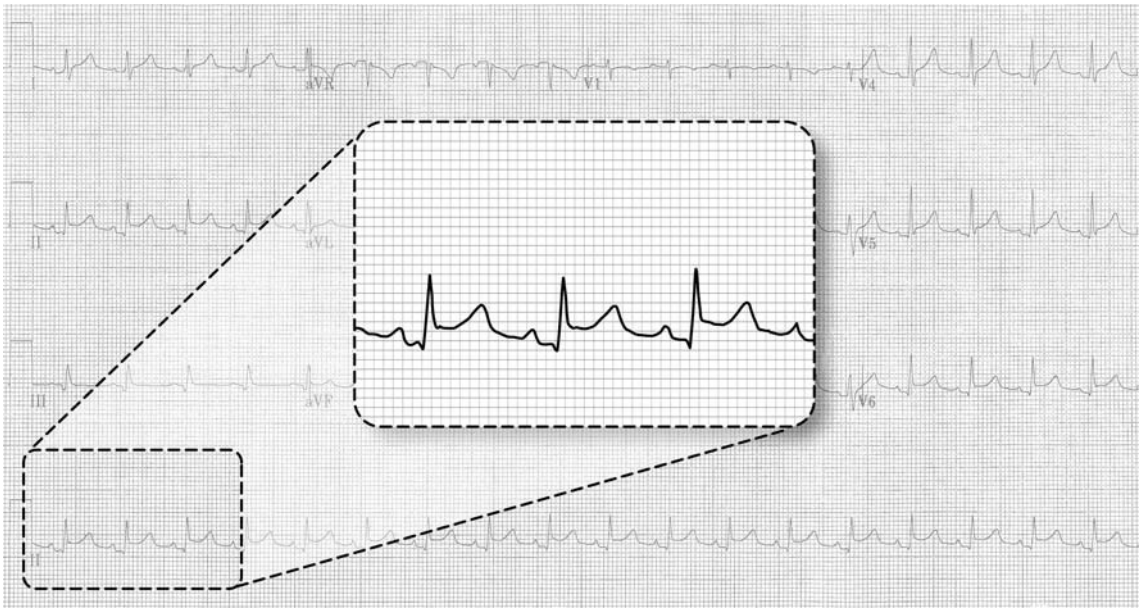

Fig. 1 Electrocardiogram showing diffuse ST segment elevation and depressed PR

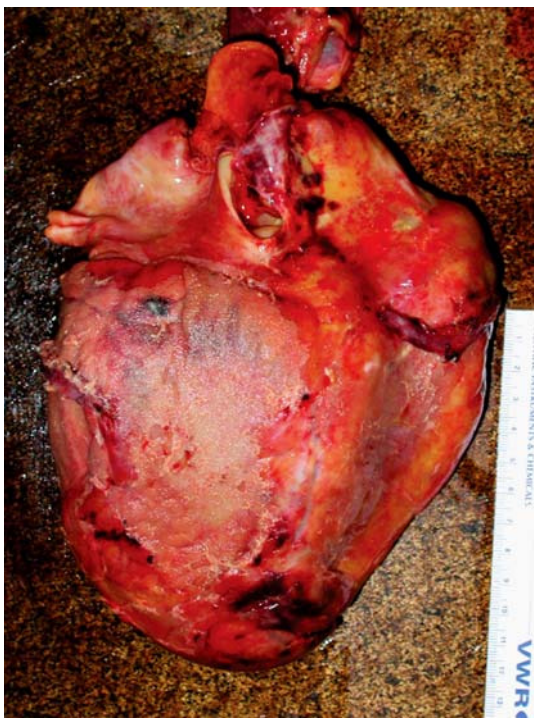

Fig. 2 Post-mortem specimen showing signs of chronic epicardial inflammation, scarring, and lysed adhesions. (Courtesy of Dr. Tack Ki Leung)

above the filling pressure and impairs filling of the ventricles.[4] If pericardial fluid accumulates rapidly, a volume as low as 100 ml can lead to tamponade. In contrast, if the fluid accumulates gradually, more than 1 l can be accommodated before the onset of symptoms in adults. In the classical presentation of cardiac tamponade, fluid accumulates across the pericardium. The right atrium, having the lowest pressure, will be the first cardiac chamber to collapse in diastole, followed by the right ventricle and the left atrium in diastole. This can be easily diagnosed using transthoracic (Fig. 4) or transesophageal echocardiography (Fig. 5). After cardiac surgery, however, localized tamponade can occur with regional compression of any of the cardiac chambers. In that situation, transesophageal echocardiography is mandatory to rule out the diagnosis (Figs. 6, 7). As tamponade progresses and shock worsens, coronary perfusion pressure is compromised leading to additional myocardial dysfunction.[6] Less frequently, and as illustrated in this case, pericardial effusions can be a source of sepsis in patients with fever of unknown origin. In patients with tamponade, venous return will be maintained as long as mean systemic pressure (Pms) is maintained. Therefore, volume expansion and vasopressors such as noradrenaline and phenylephrine can be used. Inotropes can also be used to lower right atrial pressure; however, they can reduce Pms and worsen the condition. The definitive treatment of tamponade is the reduction of the surrounding pressure either percutaneously or surgically. Our approach to the diagnosis and management of pericardial effusion and tamponade is summarized in Fig. 8.

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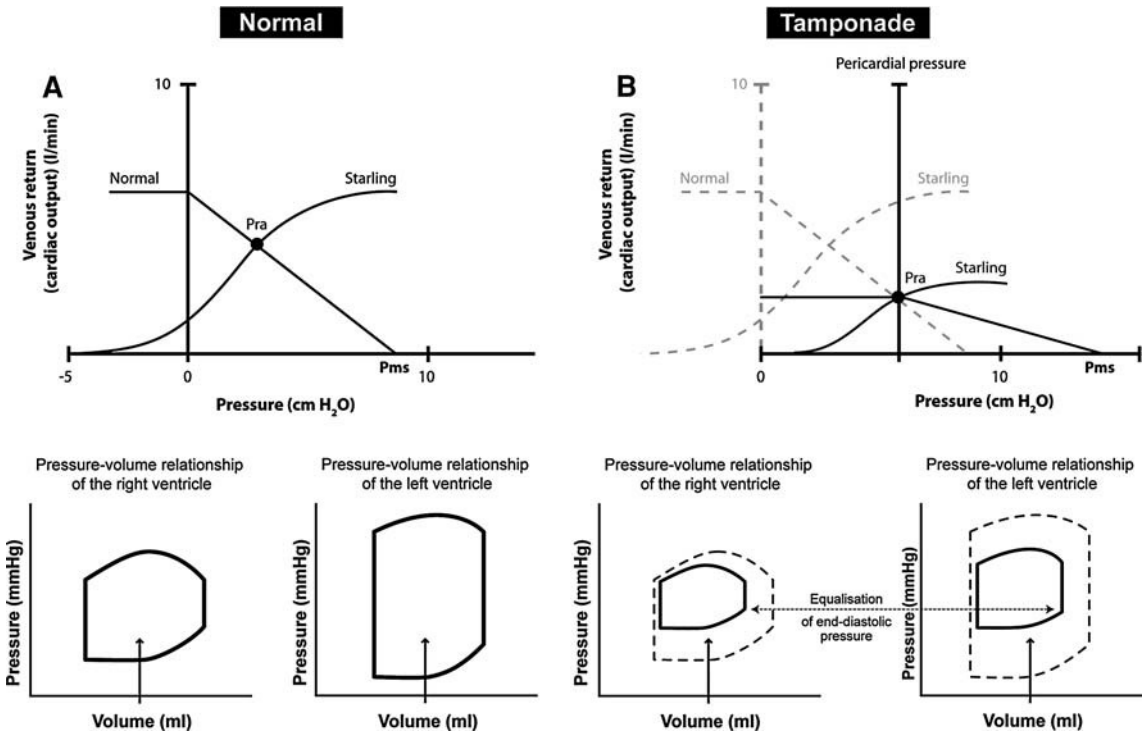

Fig. 3 Mechanism of pericardial tamponade. **a** The normal venous return (*VR*) curve and cardiac output (*CO*) using the Starling relationship. Both *VR* and *CO* are equal at a specific right atrial pressure (*Pra*). The venous return is equal to the difference between the mean systemic pressure (*Pms*) and the *Pra* divided by the resistance to *VR*. The resistance to *VR* is a function of the slope of the *VR*. At a certain point on this curve, *VR* cannot increase because *Pra* becomes sub-atmospheric. This leads to collapse of the great vessels entering the intra-thoracic cavity. The corresponding normal pressure–volume relationship of the right and left ventricles are shown below. **b** *Top* In tamponade, *VR* and *CO* are reduced and *Pra* is increased. This is secondary to the rise in pericardial pressure. In addition, *VR* will now be limited by the pericardial pressure, not by the sub-atmospheric pressure. Therefore, *VR* is now equal to the difference between Pms and pericardial pressure divided by the resistance to *VR*. The *VR* slope is reduced by an increase in the resistance to *VR*. A normal compensatory increase in mean systemic pressure (*Pms*) will also be observed secondary to the activation of the autonomic nervous system. *Bottom* Bi-ventricular pressure–volume relationships in pericardial tamponade. The increase in pericardial pressure will be transmitted to both ventricles. Therefore, an upward shift of the horizontal part of the pressure–volume relationship will be observed. This is typically associated with equalization of end-diastolic pressures. As pericardial pressure increases and tamponade develops, bi-ventricular volumes will be further reduced. Consequently, left ventricular pressure and systemic pressure will be reduced

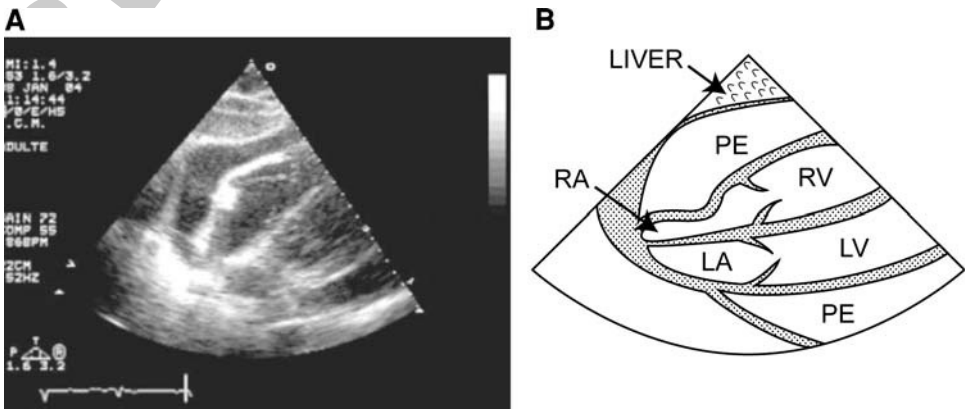

Fig. 4 Classical tamponade diagnosed using transthoracic echocardiography from a subcostal view. The compression of the right atrium (*RA*) is seen (*LA* left atrium, *LV* left ventricle, *PE* pericardial effusion, *RV* right ventricle) (Video 1, see Electronic supplementary material) (with permission of Denault et al.[5])

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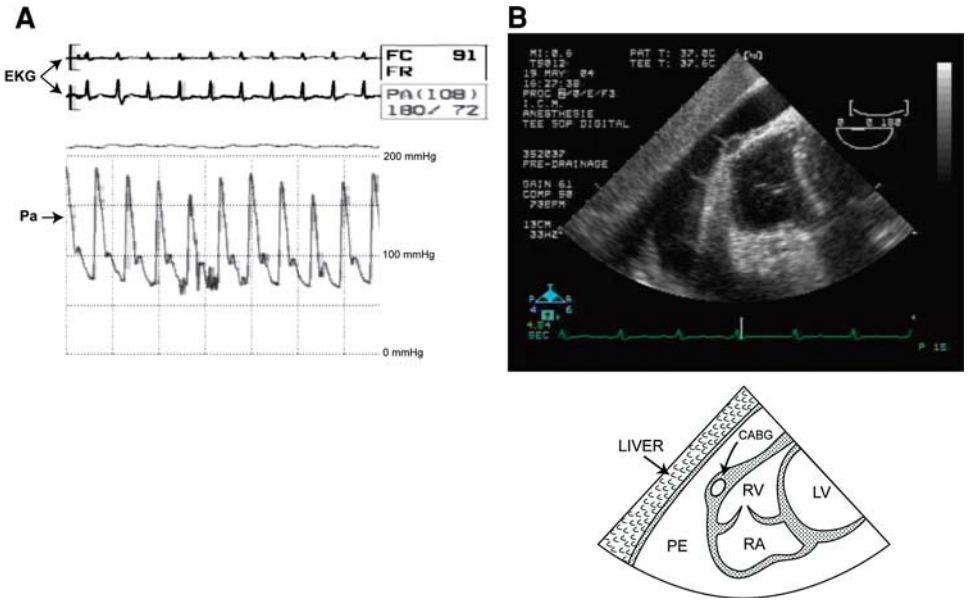

Fig. 5 Classical tamponade diagnosed using transesophageal echocardiography in a patient developing after surgical coronary revascularization from a deep transgastric view. **a** The arterial pressure (*Pa*) waveform shows the typical respiratory variation of pulsus paradoxus. The patient was on a significantly high dose of noradrenaline. **b** The intermittent compression of the right atrium (*RA*) can be visualized in Video 2 (see Electronic supplementary material) (*CABG* coronary artery bypass graft, *LV* left ventricle, *PE* pericardial effusion)

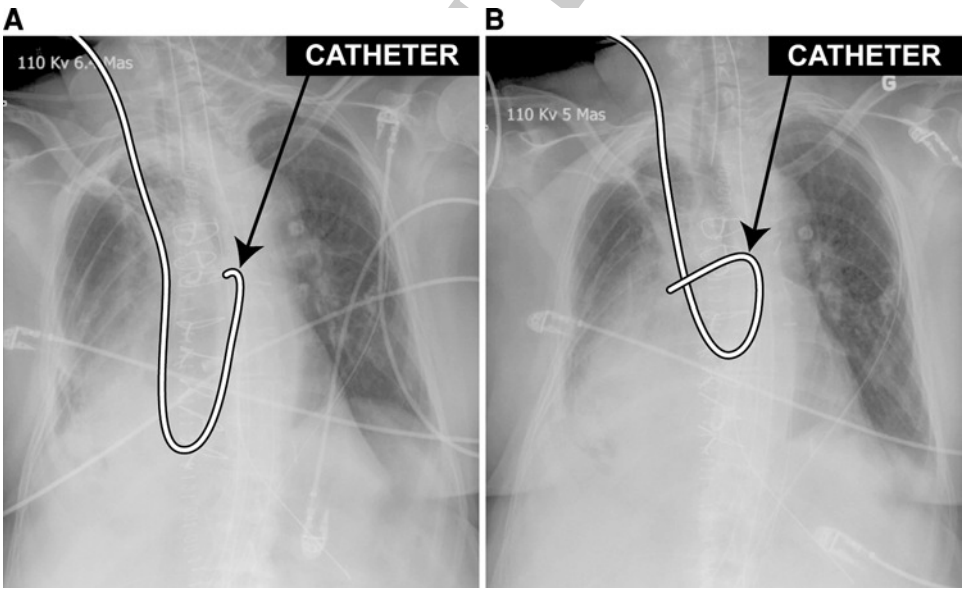

Fig. 6 **a** Chest radiograph in a critically ill patient showing the normal position of the pulmonary artery catheter (line drawn on top of the catheter). **b** In regional cardiac tamponade, the catheter is spontaneously pushed upward and further into the right pulmonary artery. This classical displacement is consistent with regional tamponade of the right atrium and ventricle

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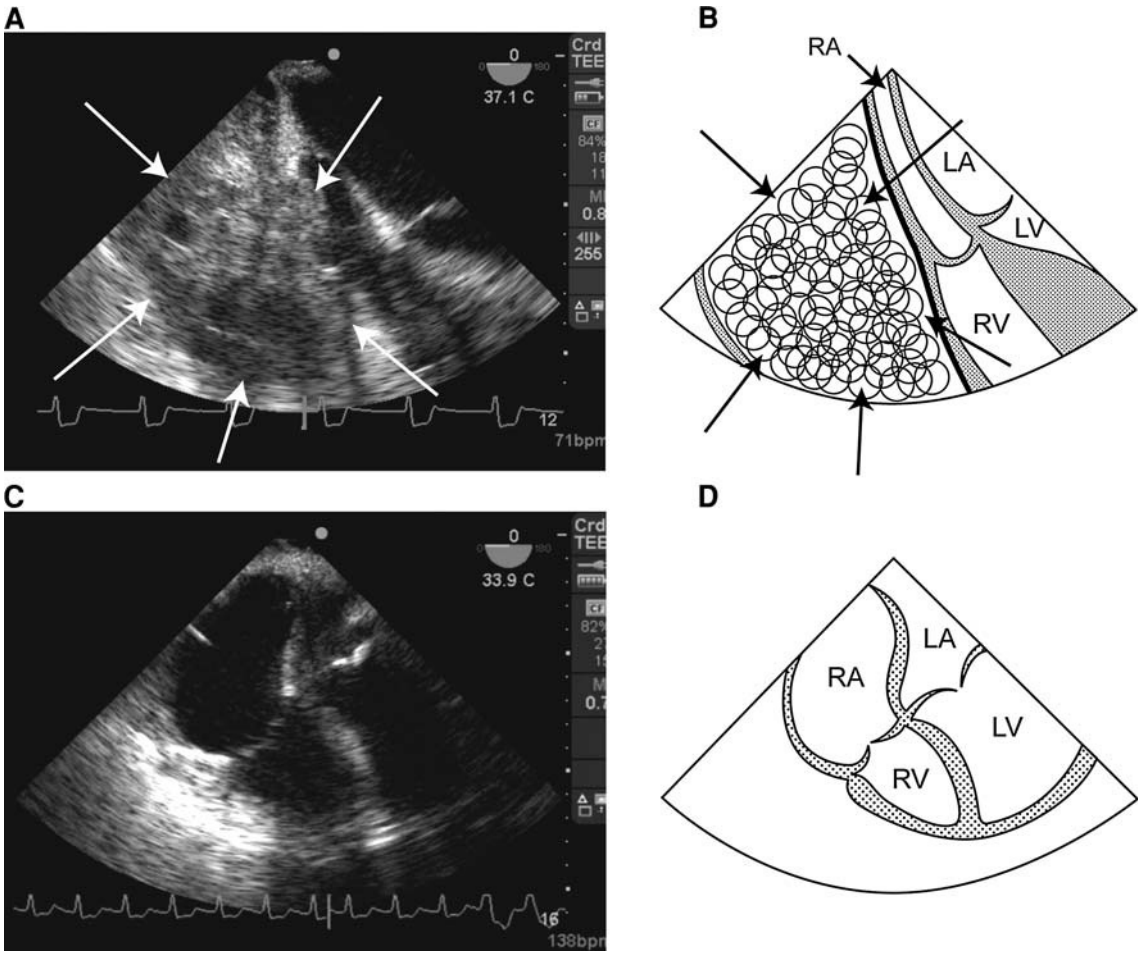

Fig. 7 Transesophageal echocardiography from a mid-esophageal view confirming the clot (*arrows*) compressing the right atrium (*RA*) and the right ventricle (*RV*) before (**a**, **b**) and after removal (**c**, **d**) (*LA* left atrium, *LV* left ventricle) (see Videos 3 and 4, available as Electronic supplementary material)

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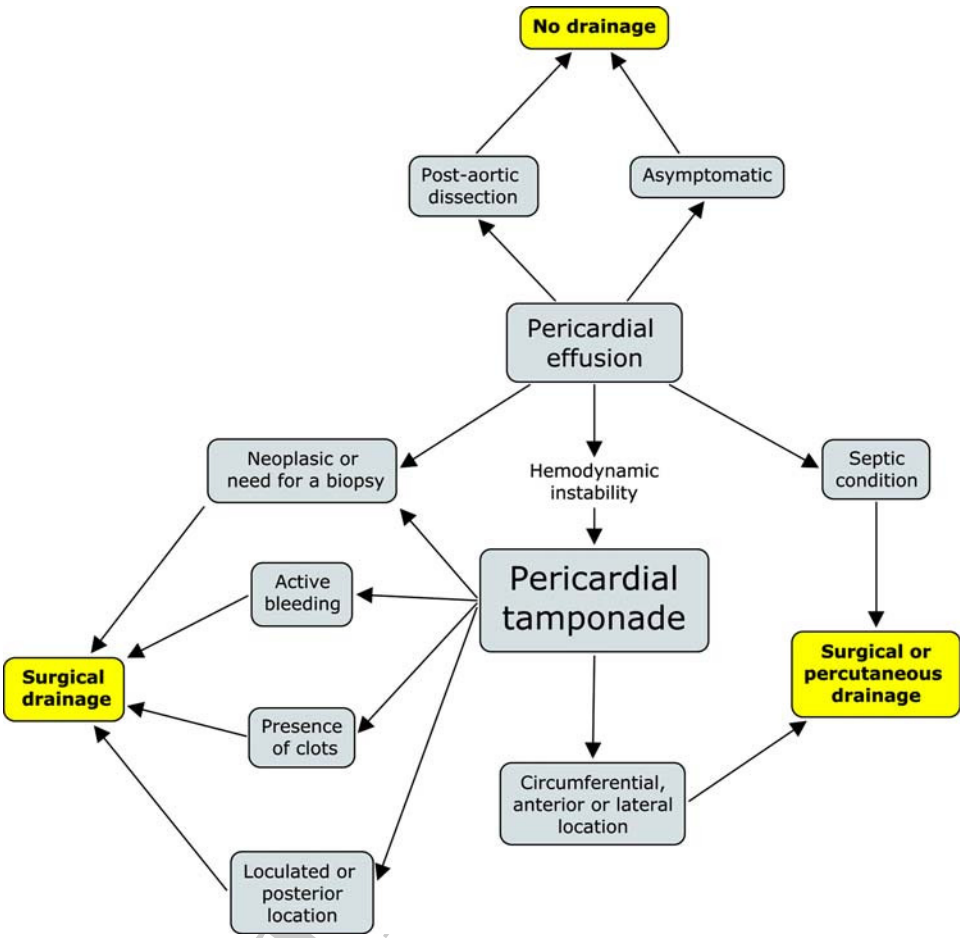

Fig. 8 A proposed approach to patient management in the presence of pericardial effusion

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Conflicts of interest None declared.

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