Pericardial Effusion and Tamponade

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Current Treatment Options in Cardiovascular Medicine 1999, **1:**79-89
Current Science Inc. ISSN 1092-8464

Opinion statement

- Pericardial effusion may occur as a result of a variety of clinical conditions, including viral, bacterial, or fungal infections and inflammatory, postinflammatory, autoreactive, and neoplastic processes [1-4]. More common causes of pericardial effusion and tamponade include malignancy, renal failure, viral and bacterial infectious processes, radiation, aortic dissection, and hypothyroidism. It can also occur after trauma or acute myocardial infarction (as in postpericardiotomy syndrome following cardiac or thoracic surgery) or as an idiopathic pericardial effusion. Although pericardial effusion is common in patients with connective tissue disease, cardiac tamponade is rare [4]. Among medical patients, malignant disease is the most common cause of pericardial effusion with tamponade [1].
- Table 1 shows the causes of pericardial tamponade. The effusion fluid may be serous, suppurative, hemorrhagic, or serosanguineous. The pericardial fluid can be a transudate (typically occurring in patients with congestive heart failure) or an exudate. The latter type, which contains a high concentration of proteins and fibrin, can occur with any type of pericarditis, severe infections, or malignancy.
- Once the diagnosis of pericardial effusion has been made, it is important to determine whether the effusion is creating significant hemodynamic compromise. Asymptomatic patients without hemodynamic compromise, even with large pericardial effusions, do not need to be treated with pericardiocentesis unless there is a need for fluid analysis for diagnostic purposes (*eg*, in acute bacterial pericarditis, tuberculosis, and neoplasias).
- The diagnosis of pericardial effusion/tamponade relies on a strong clinical suspicion and is confirmed by echocardiography or other pericardial imaging modalities.
- Alternatively, when the diagnosis of cardiac tamponade is made, there is a need for emergency drainage of pericardial fluid by pericardiocentesis or surgery to relieve the hemodynamic compromise.
- Following pericardiocentesis, it is necessary to prevent recurrence of tamponade. Intrapericardial injection of sclerosing agents, surgical pericardiotomy, and percutaneous balloon pericardial window creation are techniques used to prevent reaccumulation of pericardial fluid and recurrence of cardiac tamponade.

Introduction

The clinical presentation of patients with *pericardial effusion* varies from complete lack of symptoms to pericardial tamponade and cardiovascular collapse. Symptoms are not related to the size of the effusion. Patients with large pericardial effusions, which may develop slowly, can be remarkably asymptomatic, whereas those with rapidly accumulating smaller effusions can present with cardiac tamponade. Nevertheless, large but not hemodynamically significant pericardial effusions can compress adjacent organs, resulting in dysphagia, hoarseness, nausea, cough, dyspnea, hiccups, and abdominal fullness.

Table 1. Causes of cardiac tamponade

Infections
Viral (coxsackievirus B, influenza virus, echovirus)
Bacterial (tubercular, staphylococcal)
Malignant disease
Primary (mesothelioma)
Pericardial metastasis (breast cancer, melanoma)
Local invasion (lung cancer, lymphoma)
Trauma
Penetrating chest injury
Cardiac catheterization and percutaneous interventional procedures
Temporary and permanent pacing
Cardiac injury
Foreign body
Nonpenetrating chest injury
Connective tissue and other autoimmune diseases
Systemic lupus erythematosus
Scleroderma
Dermatomyositis
Polyarteritis
Rheumatoid arthritis
Ankylosing spondylitis
Rheumatic fever
Postinfarction (Dressler's syndrome)
Uremia
Hemopericardium
Post–myocardial infarction complicated by heart rupture
Aortic dissection
Rupture of aortic aneurysm
Anticoagulant therapy in patients with myocardial infarction, uremia, malignancy, or pericarditis
Post–radiation therapy
Post–cardiac surgery
Postsurgery hemopericardium
Postpericardiotomy syndrome
Postoperative pericarditis

Pericardial tamponade is defined as a hemodynamic abnormality produced by the accumulation of pericardial fluid, which impairs diastolic filling of the ventricles. In most patients, pericardial tamponade can be diagnosed by clinical examination. The clinical presentation of patients with cardiac tamponade varies from dyspnea and edema to circulatory collapse. Patients with cardiac tamponade have elevated systemic venous pressure, tachycardia, dyspnea, and arterial pulsus paradoxus. The heart sounds are distant. The diagnosis is confirmed by echocardiography, which demonstrates a moderately large circumferential pericardial effusion, right atrial compression, and abnormal respiratory variations in right and left ventricular dimensions as well as respiratory variations in tricuspid and mitral valve flow velocities. The classic hemodynamic findings of pericardial tamponade include arterial pulsus paradoxus, elevation and diastolic equalization of right and left ventricular diastolic pressures with pericardial pressure, and depression of cardiac output (Fig. 1).

Kussmaul's sign, defined as an inspiratory increase in mean venous pressure, never occurs in patients with pure pericardial tamponade. This is because even in the presence of severe tamponade, inspiration continues to be associated with a decrease in central venous pressure [5].

Because increased diastolic filling pressures are necessary to maintain cardiac output in patients with impending tamponade, a decrease in venous pressure, such as may occur with diuresis, bleeding, or the use of preload-reducing agents, would be associated with clinical deterioration. This is the underlying mechanism in patients presenting with low-pressure tamponade. These patients are characterized hemodynamically by low cardiac output and normal or mildly elevated filling pressures. Nevertheless, there is diastolic equalization of intracardiac and intrapericardial pressures of less than 15 mm Hg.

The electrocardiogram of patients with pericardial effusion usually shows low voltage. Electrical alternans,

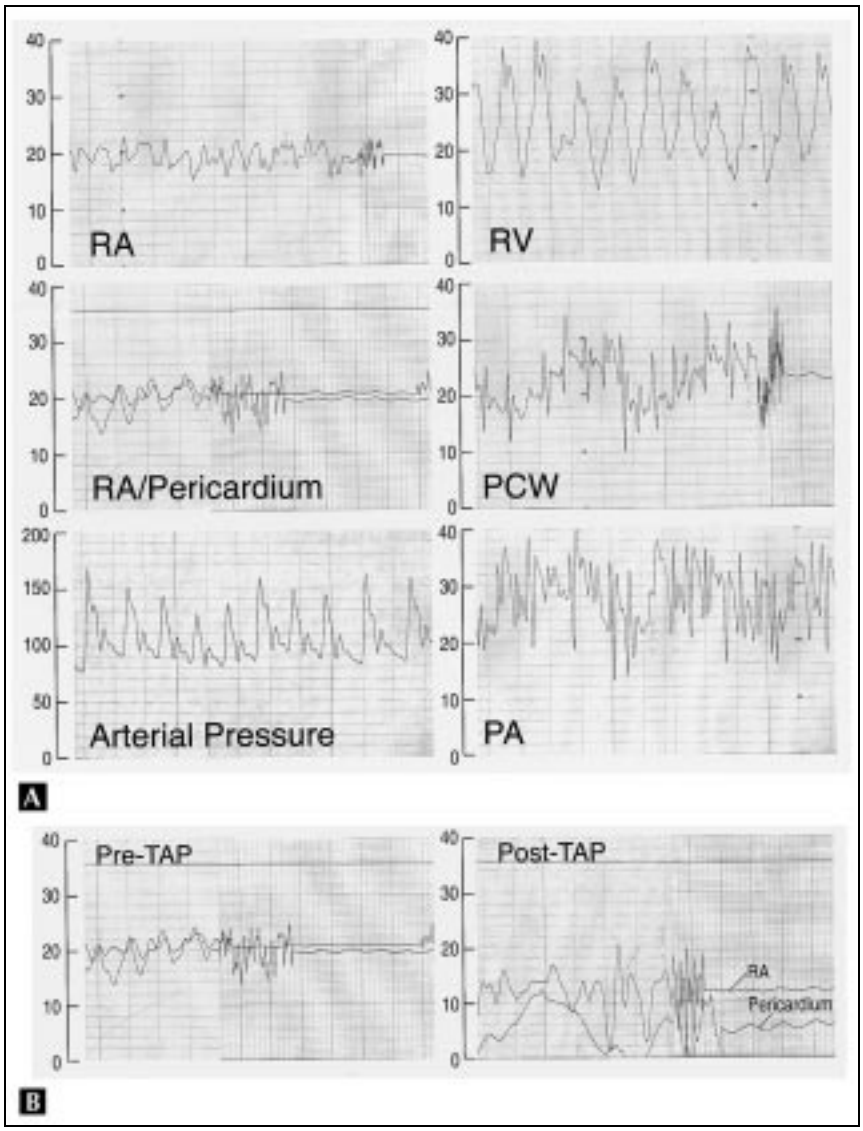

Figure 1. **A**, Hemodynamic findings in a patient with pericardial tamponade. Observe equalization of diastolic pressures in the cardiac chambers and pericardium at 20 mm Hg and the presence of arterial pulsus paradoxus.

B, Simultaneous right atrium and intrapericardial pressures before and immediately after pericardiocentesis. PA—pulmonary artery; PCW—pulmonary capillary wedge pressure; RA—right atrium; RV—right ventricle.

probably related to alterations of the anatomic position of the heart, is a more specific finding in patients with pericardial effusion.

PATHOPHYSIOLOGY

The pericardium is a thin, fibrous, and rather inelastic membranous structure that surrounds the heart and serves to limit acute cardiac distention and to modulate ventricular interdependence. The normal pericardium has a steep pressure-volume curve. It is distensible when the total intrapericardial volume is small and inextensible when the total pericardial volume is large. The normal pericardial space may contain 20 to 50 mL of fluid. Initial accumulations of 80 to 100 mL result in only mild elevation of the intrapericardial pressure over its normal subatmospheric levels. Further increases in the amount of pericardial fluid result in a sharp increase in intrapericardial pressure, however. In the presence of pericardial effusion, the intrapericardial pressure depends on the relationship between the absolute volume of the effusion, the speed of pericardial fluid accumulation, and the elasticity of the pericardium.

Because the pericardium grows to accommodate its content when subjected to chronic stretching, the hemodynamic effects of pericardial effusion are related to the speed of accumulation of the pericardial fluid. Whereas the rapid accumulation of 150 to 200 mL may result in cardiac tamponade, the slow accumulation of larger effusions (1 to 2 L) may be well tolerated without evidence of tamponade [5,6•,7••].

The normal intrapericardial pressure is negative and is similar to the intrapleural pressure (0 to -2 mm Hg). During inspiration, the intrapericardial pressure decreases abruptly to subatmospheric levels. The intrapericardial pressure is an important determinant of the transmural pressures of the cardiac chambers. Transmural pressure represents the difference between the intracardiac and the intrapericardial pressures and is the

main determinant of diastolic filling and expansion of the cardiac chambers. In cardiac tamponade, the transmural pressures are, on average, zero or below zero and are reciprocally reduced and increased during the respiratory phases in the left heart versus the right heart. For example, inspiration increases right heart filling pressure at expense of the left heart, with reversal during expiration [6•,7••]. Furthermore, ventricular interaction plays an important role in the relative filling of the right and the left ventricles and in the arterial paradoxus seen in patients with cardiac tamponade. Ventricular interaction occurs as a consequence of the competing ventricular diastolic pressures generated by the pulmonary and systemic venous beds and the associated changes in the interventricular septum. There is increased intrathoracic and, therefore, pulmonary venous pressure during expiration and concavity of the interventricular septum toward the left ventricle. Opposite changes occur during inspiration. These changes facilitate left ventricular filling during expiration and decreased pressure during inspiration. Reciprocal changes in right ventricular filling during the respiratory cycle occur [5].

The end result of the increase in intrapericardial pressure is the impediment of the diastolic filling of the ventricles, resulting in an elevation in ventricular filling pressures and a decrease in cardiac output. At a critical level of intrapericardial pressure, there is a precipitous decrease in cardiac output and arterial blood pressure and hemodynamic collapse. Because patients with critical tamponade operate on the steep portion of the pericardial pressure-volume curve, drainage of small pericardial volume results in a drastic decrease in intrapericardial pressure and rapid clinical and hemodynamic improvement. This occurs owing to shifting of the stretched pericardium back to the flat portion of the pericardial pressure-volume curve.

IMAGING OF THE PERICARDIUM

Although an unexplained increase in heart size on a routine chest x-ray remains one of the most common clues to the diagnosis of pericardial effusion, the sensitivity and specificity of this technique in the diagnosis of pericardial effusion is low. Enlargement of the cardiac silhouette on chest x-ray is not apparent until 250 mL of fluid has accumulated in the pericardial space. Small effusions may be present with a normal chest x-ray, and differentiation of large pericardial effusion from four-chamber cardiac enlargement is difficult. Nevertheless, a rapid increase in the cardiac silhouette, particularly if the lung fields are clear, favors the diagnosis of pericardial effusion and indicates the need for further testing to establish the final diagnosis. A typical chest x-ray of a patient with cardiac tamponade has a wide cardiac silhouette with little abnormality in the lungs. Typically, these patients have no evidence of pulmonary edema; however, pulmonary edema may be present when other diseases associated with left ventricular failure, such as myocardial infarction, are also present. Today, echocardiography is the procedure of choice for the diagnosis of pericardial effusion because it provides excellent sensitivity and specificity. Before the development of echocardiography, the diagnosis of pericardial effusion and other pericardial diseases was fraught by inaccuracy. In addition to echocardiography, computed tomography and magnetic resonance imaging are useful in the evaluation of patients with pericardial diseases. Their high resolution is useful in the assessment of pericardial thickness as well as in the detection of pericardial effusion, masses, and cysts.

Echocardiography and Doppler Echocardiography revolutionized the evaluation of pericardial effusion and tamponade by providing an accurate and noninvasive means of detection and follow-up of the course of pericardial effusion. Today, echocardiography is the gold standard for the diagnosis of pericardial effusion [8••,9,10]. A persistent echo-free space throughout the cardiac cycle between the parietal pericardium and the epicardium is the diagnostic characteristic demonstrated by M-mode echocardiography. Two-dimensional echocardiography allows delineation of the size and distribution of the effusion as well as detection of loculated effusions. Circumferential effusions of greater than 1 cm are considered to be large (> 500 mL); moderate effusions (100 to 500 mL) are those with a circumferential effusion of less than 1 cm; and small effusions (< 100 mL) are those that are posterior to the left ventricle and measure less than 1 cm. Swinging of the heart in the pericardial space is associated with massive pericardial effusions. The type of pericardial fluid cannot be identified by echocardiography; however, increased echogenicity raises the question of the presence of proteins or cells, or both, in the pericardial fluid. Fibrin deposits localized in the epicardial surface can be identified as echogenic masses.

Table 2. Echocardiographic findings

Abnormal inspiratory increase in right ventricular dimensions and abnormal inspiratory decrease in left ventricular dimensions
Right atrium collapse (> 30% of the cardiac cycle)
Right ventricular early diastolic collapse
Abnormal inspiratory increase in blood flow velocity through the tricuspid and pulmonic valves and abnormal inspiratory decrease in mitral and aortic valve flow velocity
Respiratory variations in pulmonary and hepatic venous flow
Dilated inferior vena cava with lack of inspiratory collapse
Swinging heart

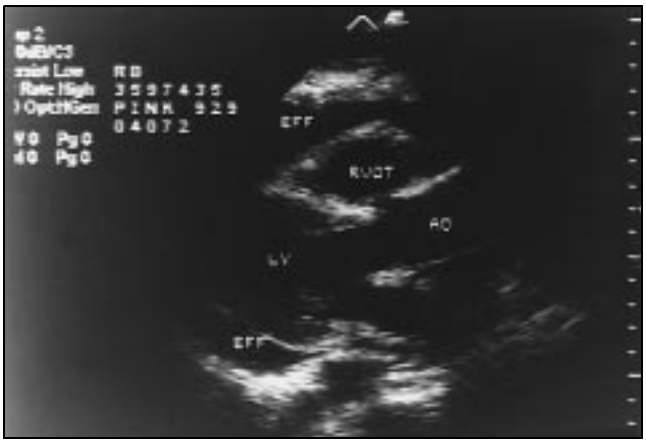

Figure 2. Two-dimensional echocardiogram of a patient with pericardial tamponade shows early diastolic collapse of the right ventricle and a large pericardial effusion. AO—aorta; EFF—pericardial effusion; LV—left ventricle; RVOT—right ventricular outflow tract.

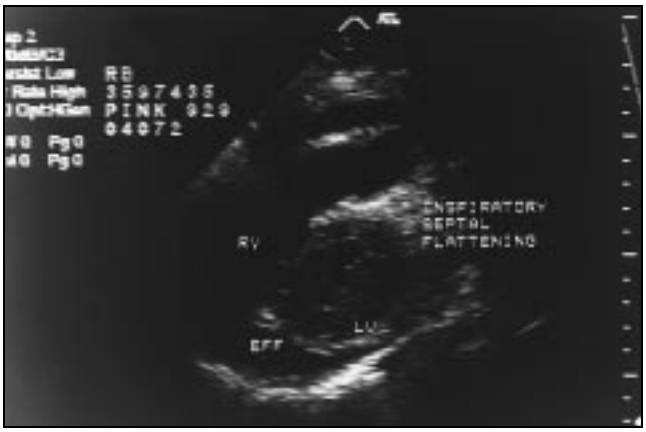

Figure 3. Two-dimensional echocardiogram of a patient with pericardial tamponade shows inspiratory septal flattening and a large pericardial effusion. EFF—pericardial effusion; LV—left ventricle; RV—right ventricle.

Two-dimensional echocardiography is a fast and noninvasive technique used to make the diagnosis of pericardial tamponade. Table 2 displays the echocardiographic findings seen in cardiac tamponade. The classic signs of cardiac tamponade are right atrial and right ventricular diastolic collapse (Figs. 2–4). Two-dimensional echocardiography allows careful examination of the respiratory changes in ventricular filling present in cardiac tamponade. A greater-than-normal increase in right ventricular dimensions and decreases in left ventricular dimensions occur during inspiration. Early diastolic collapse of the right ventricle is present when the intrapericardial pressure exceeds right ventricular pressure, which represents transient negative transmural right ventricular pressure (*see* Fig. 2). Late diastolic right atrium collapse is a very sensitive but less specific sign of pericardial tamponade (*see* Fig. 4). Dilatation of the inferior vena cava with lack of inspiratory

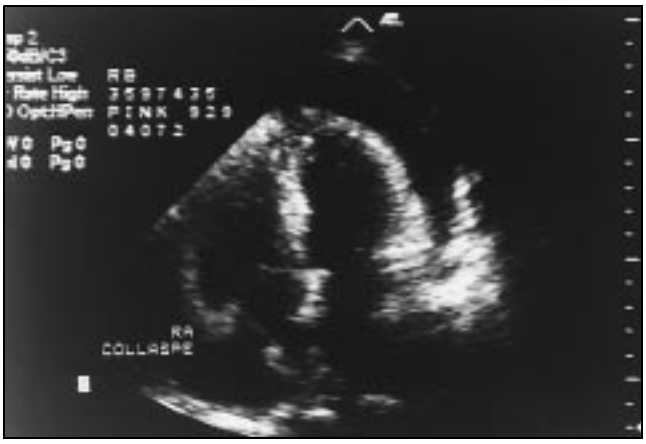

Figure 4. Two-dimensional echocardiogram of a patient with pericardial tamponade shows right atrium (RA) collapse.

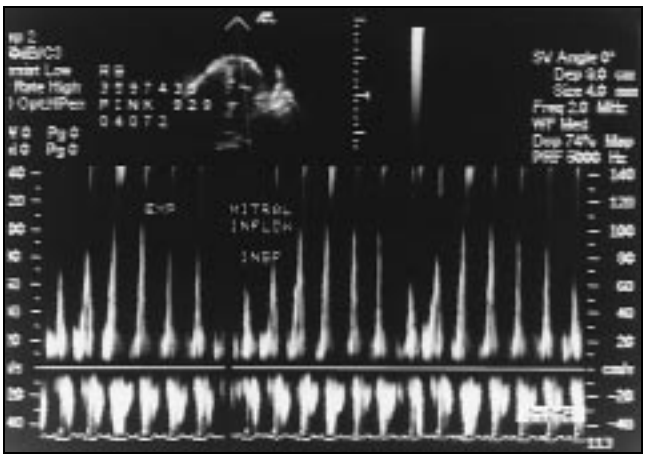

Figure 5. Doppler echocardiogram in a patient with pericardial tamponade. Note the inspiratory decrease and expiratory increase in mitral flow velocity. EXP—expiration; INSP—inspiration.

collapse and swinging of the heart are also seen in patients with pericardial tamponade.

Doppler echocardiography provides direct assessment of ventricular filling patterns characteristic of pericardial tamponade [8••,9,10]. Patients with pericardial tamponade have both a marked increase in tricuspid and pulmonic flow velocities and a marked decrease in mitral and aortic valve flow velocities during inspiration when compared both with normal and with patients with effusions but without tamponade (Fig. 5). In addition, in patients with pericardial tamponade, there are changes in the pattern of left atrium inflow and exaggerated respiratory variations in pulmonary venous flow velocity.

CARDIAC CATHETERIZATION

Cardiac catheterization has historically been the standard for making the diagnosis of cardiac tamponade. Right heart catheterization can confirm the significance

of a pericardial effusion and allows evaluation of hemodynamic changes occurring after pericardiocentesis. Table 3 shows the typical catheterization findings seen in cardiac tamponade. In patients with cardiac tamponade, there is elevation and diastolic equalization of all cardiac chamber and intrapericardial pressures (*see* Fig. 1). This means that the intrapericardial pressure, the mean right atrium pressure, the mean pulmonary capillary wedge or left atrium pressure, the diastolic pulmonary artery pressure, and the right and left ventricular end-diastolic pressures are elevated and equal. Conventionally, equalization represents diastolic pressures differing by no more than 5 mm Hg. In addition to producing elevation of the central venous pressure, cardiac tamponade produces characteristic changes in the waveform of the cardiac chambers. With increasing severity of cardiac tamponade, the Y descent and the early diastolic dip in the ventricular pressure tracings are gradually obliterated and finally abolished. This absence of the Y descent in the right atrium tracing, which occurs as a consequence of the absence of pressure changes from early to late diastole, is one of the hemodynamic hallmarks of pericardial tamponade. As pericardial fluid is removed, the intrapericardial pressure should return to intrapleural pressure values, and the right atrial waveform should normalize with reappearance of the diastolic Y descent. If after pericardiocentesis the right atrium pressure remains elevated and a prominent Y descent appears, however, the diagnosis of effusive-constrictive disease must be considered.

Pulsus paradoxus is another hallmark of pericardial tamponade. It is an exaggeration of the normal physiologic decrease in systolic arterial blood pressure during inspiration. It is defined as a greater than 10 mm Hg decrease in systolic arterial blood pressure during inspiration [5,6•,7••]. In an attempt to compensate for patients with extremely high or low blood pressure, a percentage rather than an absolute decrease in systolic arterial pressure during inspiration has been used [5]. A greater than 9% decrease in systolic arterial pressure during inspiration is diagnostic of pulsus paradoxus. Except for the extremes of arterial pressure, however, calculation of this percentage decrease in systolic blood pressure during inspiration does not provide additional advantage. Although arterial pulsus paradoxus is an important sign of pericardial tamponade, it may be absent under certain circumstances in some patients presenting with tamponade (*eg*, regional left heart compression after cardiac surgery) [11]. In these patients, severe hemodynamic compromise occurs despite normal right atrium and systemic venous pressures. Similarly, pulsus paradoxus could be absent in localized right atrial compression. In addition, pulsus paradoxus may not be present in those patients with left ventricular dysfunction, in those receiving positive-pressure ventilation, and in those with atrial septal defect, aortic regurgitation, or pulmonary artery obstruction. On the other hand, patients with severe chronic obstructive pulmonary disease may have increased venous pressure and pulsus paradoxus in the absence of cardiac tamponade.

Table 3. Cardiac catheterization findings

Elevated filling pressures
Diastolic equalization of pressures
Absent or blunted Y descent in the right atrium pressure tracing
Absent or blunted early diastolic dip in the right ventricular pressure tracing
Arterial pulsus paradoxus

Treatment

Interventional procedures

Pericardiocentesis

Pericardiocentesis is most commonly performed via a subxiphoid approach under electrocardiographic and fluoroscopic guidance. Traditionally, pericardiocentesis has been performed in the cardiac catheterization laboratory with arterial and right heart catheterization monitoring. The procedure is also now performed in noninvasive laboratories, intensive care units, or even at bedside under echocardiographic guidance. Pericardiocentesis is a safe procedure when performed by trained personnel.

Standard procedure

After administration of 1% lidocaine to the skin and the deeper tissues, the pericardial needle is connected to an electrocardiographic V lead. The needle is advanced from the left of the subxiphoid area, aiming toward the left shoulder. Sometimes a discrete pop may be felt as the needle enters the pericardial space. ST segment elevation is seen on the V lead tracing when the needle touches the epicardium. The needle should be retracted slightly until the ST segment disappears. Once the pericardial space has been entered, a guidewire is introduced into the pericardial space through the needle. The

needle is then removed, and a catheter is inserted into the pericardial sac over the guidewire. The catheter is frequently left in place to monitor pericardial fluid drainage and to act as a route for instillation of sclerosing or chemotherapeutic agents.

Complications The success rate of pericardiocentesis increases and the incidence of complications decreases with increasing size of the effusion. The potential complications of pericardiocentesis include laceration of the heart or a coronary vessel, sometimes with fatal consequences.

Special points Echocardiography could be useful during pericardiocentesis to guide one toward the most readily accessible portion of the pericardial space, particularly in those patients with loculated effusions. Samples of pericardial fluid should be sent for cell counting (complete blood count, differential, and cell count), chemistry analysis (glucose, proteins, and lactic dehydrogenase), cytologic studies, aerobic and anaerobic culturing, and special staining (Gram and acid-fast).

Cost effectiveness Not known.

Prevention of fluid reaccumulation

- Following pericardiocentesis, it is desirable to prevent recurrence of tamponade. For many patients with pericardial effusion and tamponade, standard percutaneous pericardial drainage with an indwelling pericardial catheter is sufficient to avoid recurrence of pericardial effusion and tamponade.
- Recurrences following catheter drainage have been reported in 14% to 50% of patients with pericardial effusion and tamponade [2,12–14]. Reaccumulation of pericardial fluid is common in those patients with malignant pericardial effusions. Autopsy and surgical studies have shown that myocardial or pericardial metastases are found in approximately 50% of patients who present with pericardial tamponade due to malignancy [15–18]. In patients with continued drainage of more than 100 mL/24 h 3 days after standard catheter drainage, more aggressive therapy should be considered.
- Several additional approaches are available to prevent reaccumulation of pericardial fluid; these include intrapericardial instillation of sclerosing agents, use of chemotherapy, and radiotherapy [19,20].
- The intrapericardial instillation of 500 to 1000 mg of tetracycline diluted in 20 mL of normal saline solution has been used with good initial results. A failure rate of 17% has been reported, however [19]. The intrapericardial instillation of nonabsorbable steroids has been used with good results in patients presenting with uremic pericardial effusion or tamponade.
- Reaccumulation of fluid with recurrence of cardiac tamponade has been considered as an indication for a surgical intervention [21–31]; however, morbidity and late recurrence of symptoms are not uncommon with this technique [22–24].

Percutaneous balloon pericardial window creation

Subxiphoid and transthoracic approaches to creating a surgical pericardial window have been used. Some surgeons have recommended the subxiphoid approach as primary therapy for malignant pericardial tamponade based on its high initial success rate and acceptable rate of recurrence [22–27]. Transthoracic techniques offer a lower recurrence rate but are associated with higher morbidity [23–31]. Therefore, extensive pericardial resection is usually reserved for patients for whom a longer survival can be anticipated.

Patients with advanced malignancy and pericardial tamponade are often poor candidates for surgical therapy because of their limited life expectancy. Palacios *et al.* [32•] introduced the technique of creation of a percutaneous balloon pericardial window as a less invasive alternative to creation of a surgical pericardial window. With this technique, a pericardial window and adequate drainage of pericardial effusion can be done percutaneously with the use of a balloon-dilating catheter (Fig. 6).

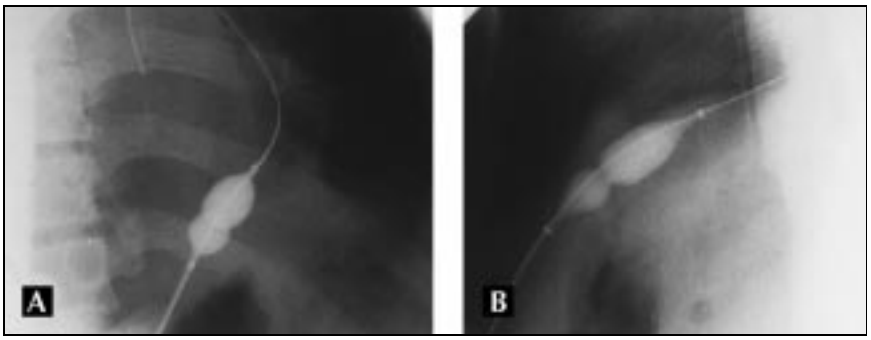

Figure 6. Anteroposterior (*A*) and lateral (*B*) fluoroscopic images of a percutaneous balloon pericardial window. As the balloon is inflated manually, a waist is seen at the pericardial margin.

After this initial favorable experience, a multicenter percutaneous balloon pericardial window registry evaluated the therapeutic efficacy and risk of this technique in a larger group of patients [33•,34•].

Standard procedure

The technique of percutaneous pericardial window creation is relatively simple and safe. It is performed in the catheterization laboratory with minimal discomfort with the patient under local anesthesia and receiving mild sedation with intravenous narcotics and a short-acting benzodiazepine. It is offered 1) as an alternative to a surgical procedure for those patients in whom there is drainage of more than 100 mL per 24 hours 3 days after pericardiocentesis or 2) as primary therapy at the time of initial pericardiocentesis.

The subxiphoid area around the indwelling pigtail pericardial catheter is infiltrated with 1% lidocaine. A few milliliters of iodine contrast injected into the pericardial space helps to outline the parietal pericardium. A 0.038-inch guidewire with a preshaped curve at the tip is advanced through the pigtail catheter into the pericardial space. The catheter is then removed, leaving the guidewire in the pericardial space. After predilation along the track of the wire with a 10 F dilator, a balloon dilating catheter that is 20 mm in diameter and 3 cm long is advanced over the guidewire and positioned to straddle the parietal pericardium. Care should be taken to advance the proximal end of the balloon beyond the skin and the subcutaneous tissue. The balloon is inflated manually until the indentation produced by the parietal pericardium on the dilating balloon disappears (*see* Fig. 6). Biplane fluoroscopy is helpful to assure the correct position of the balloon. Two to three inflations are performed to assure an adequate opening of the pericardium. The balloon-dilating catheter is then removed, leaving the 0.038-inch guidewire in the pericardial space. A new pigtail catheter is then advanced over this guidewire and placed into the pericardial space.

If balloon pericardial window creation is being performed at the time of primary pericardiocentesis, the pericardium is entered by a standard subxiphoid approach, and pericardial window creation is performed as just described after removal of the majority of the pericardial fluid.

Contraindications

Patients with marginal pulmonary mechanics, such as is found after pneumonectomy, should be evaluated with caution because the development of a left pleural effusion may compromise their remaining lung function. Finally, an increased risk of bleeding from the pericardiotomy site occurs in those patients with either platelet or coagulation abnormalities. In these patients, a surgical procedure under direct visualization may be safer.

Complications A significant concern following percutaneous balloon pericardial window creation is the development of a large pleural effusion. Most patients develop a left pleural effusion within 24 to 48 hours of the procedure (Fig. 7). In most cases, this resolves spontaneously, presumably owing to the greater reabsorption capacity of the pleural surface. Thoracocentesis or chest tube placement was required in 15% of patients with preexisting pleural effusions compared with 9% of patients without preexisting effusions. For this reason, it is desirable to remove most of the pericardial fluid before creating the balloon window to limit the potential volume of fluid that can immediately move to the pleural space. If the chest x-ray before percutaneous pericardial window creation reveals evidence of a large pleural effusion, the chance of needing thoracocentesis is higher, and percutaneous pericardial window creation should be performed only if the cardiac benefits outweigh the risks of thoracentesis or chest tube placement.

Special points After percutaneous balloon pericardial window creation, patients return to a regular medical floor. The pericardial catheter should be aspirated every 6 to 8 hours and flushed with heparinized solution (5 mL, 100 U/mL). Pericardial drainage volumes should be recorded and the catheter removed when there is less than 75 mL of drainage in 24 hours. Frequently, at the time of catheter removal, there is evidence of a new or increasing pleural effusion on chest x-ray. Follow-up two-dimensional echocardiography is performed within 24 to 48 hours after removal of the pericardial catheter. Postprocedure echocardiography can be used to monitor for the reaccumulation of pericardial fluid. Chest radiography should be performed to monitor for the development of pleural effusion caused by drainage of the pericardial fluid.

Technical variations in the subxiphoid technique of percutaneous balloon pericardial window creation include the dilation of two adjacent pericardial sites, the use of the apical approach, the use of an Inoue (Toray Medical Group, Houston, TX) balloon catheter, the use of double balloons, and the use of an 18-mm dilating balloon to facilitate the introduction of a 16 F chest tube into the pericardial space [35–38]. Other investigators have attempted laparoscopic pericardial fenestration [39,40]. Thoracoscopic techniques have been developed to create a larger pericardial window with low morbidity compared with open surgical techniques [41]. With this technique, adequate long-term drainage may be provided and specimens for pathologic analysis may be obtained.

Cost effectiveness Less expensive than surgical pericardial window.

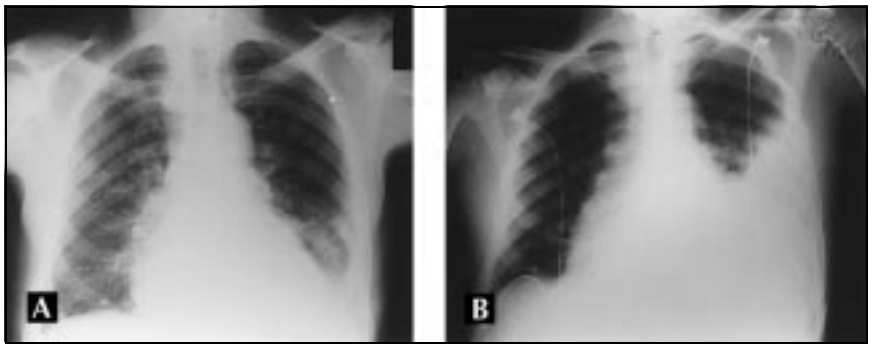

Figure 7. Posteroanterior chest x-ray before (*A*) and 48 hours after (*B*) successful percutaneous balloon pericardial window creation. On admission, an enlarged cardiac silhouette is present. Forty-eight hours following percutaneous balloon pericardial window creation, a new left pleural effusion is present.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:
- • Of special interest
- •• Of outstanding interest

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In most patients, cardiac tamponade should be diagnosed by means of a clinical examination, which shows elevated systemic venous pressure, tachycardia, dyspnea, and paradoxical arterial pulse. The diagnosis is confirmed by echocardiographic demonstration of moderately large or large circumferential pericardial effusion and, in most instances, right atrial compression as well as abnormal respiratory variation in right and left ventricular dimensions and in tricuspid and mitral valve flow velocities.

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Excellent review of the subject.

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The technique and pitfalls of recording, the criteria for the diagnosis of pericardial effusion, and the sensitivity of these criteria are discussed. Echocardiographic findings in special instances (*eg*, accumulation of pericardial effusion behind the left atrium, the swinging heart syndrome, and cardiac tamponade) are also discussed.

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