Acute Pericardial Tamponade


**Epidemiology**

The reported incidence of acute pericardial tamponade is approximately 2% in patients with penetrating trauma to the chest and upper abdomen; it is rarely seen after blunt chest trauma. It occurs more commonly with SWs than GSWs, and 60% to 80% of patients with SWs involving the heart develop tamponade. Patients with acute pericardial tamponade can deteriorate in minutes, but many can be saved if proper steps are taken.

**Anatomy/Pathophysiology**

The primary feature of a pericardial tamponade is an increase in intrapericardial pressure and volume. As the volume of the pericardial fluid encroaches on the capacity of the atria and ventricles to fill adequately, ventricular filling is mechanically limited and thus the stroke volume is reduced. This results in decreased cardiac output and ultimately diminished arterial systolic blood pressure and decreased pulse pressure. Concomitantly CVP rises because of the mechanical backup of blood into the vena cava.

Several compensatory mechanisms then occur. The heart rate and total peripheral resistance rise in an attempt to maintain adequate cardiac output and blood pressure. A less effective compensatory response, resulting in a greater rise of CVP, is an increase in venomotor tone caused by contractions of the smooth muscles within the wall of the vena cava.

In a normotensive patient, the earliest response to pericardial tamponade is a progressive rise in CVP to a level greater than 15 cm H₂O. A rising CVP in a hypotensive patient indicates that the normal compensatory responses are unable to maintain an adequate cardiac output. A simultaneous fall in both the CVP and blood pressure, which can occur precipitously and without warning, signals decompensation and imminent cardiac arrest. In animal studies, electromechanical dissociation and sudden bradycardia have been shown to precede the terminal event.

The diagnosis of pericardial tamponade should be suspected in any patient who has sustained a penetrating wound or blunt trauma to the thorax or upper abdomen. One is never certain of the trajectory of the bullet or the length, force, and direction of a knife thrust. Obviously wounds directly over the precordium and epigastrium are more likely to produce a cardiac injury resulting in tamponade than those in the posterior or lateral thorax. Nevertheless, it must be assumed that a penetrating wound, particularly a GSW, anywhere in the thorax or upper abdomen may have injured the heart.

**Clinical Features**

Patients with cardiac tamponade may initially appear deceptively stable if the rate of bleeding into the pericardial space is slow or if the pericardial wound allows intermittent decompression. Other patients may complain primarily of difficulty breathing, which suggests pulmonary rather than cardiac pathology.

The physical findings of pericardial tamponade are hypotension, distended neck veins, and, rarely, distant or muffled heart tones. This so called *Beck’s triad* is sometimes difficult to demonstrate clinically, especially in the midst of a major resuscitation with concomitant hypovolemia. The most reliable signs of pericardial tamponade are an elevated CVP (>15 cm H₂O) in association with hypotension and tachycardia. When this triad is present either before or after adequate volume replacement, the diagnosis of acute pericardial tamponade, as well as tension pneumothorax, should be considered.

Acute pericardial tamponade may be seen with three distinct clinical pictures. If the hemorrhage is confined to the pericardial space, the patient is initially normotensive but will have a tachycardia and elevated CVP. If untreated, most of these patients go on to develop hypotension.

If significant hemorrhage has occurred outside the pericardial sac, either through a rent in the pericardium or from associated trauma, the clinical picture is that of hypovolemic shock with hypotension, tachycardia, and a low CVP. If the CVP rises to a level of 15 to 20 cm H₂O with volume replacement and hypotension and tachycardia persist, the
presence of a pericardial tamponade must be considered. One must also consider other causes such as a tension pneumothorax, Valsalva’s maneuver, or pulmonary edema secondary to fluid overload.

The third clinical picture is that of an intermittently decompressing tamponade. In this case, intermittent hemorrhage from the intrapericardial space occurs, decompressing and partially relieving the tamponade. The clinical picture may wax and wane depending on the intrapericardial pressure and volume and total blood loss. In general, this condition is compatible with a longer survival than are the first two clinical presentations.

*Pulsus paradoxus* is defined as an excessive drop in systolic blood pressure during the inspiratory phase of the normal respiratory cycle. This sign may be an additional clue to the presence of pericardial tamponade, but is often difficult to measure during an intensive resuscitation.

**Diagnostic Strategies**

**Ultrasound**

Ultrasound (US), which is becoming more widely available in EDs around the world, enables rapid, accurate, and noninvasive diagnosis of pericardial tamponade. This study can be performed at the bedside in the ED during the initial resuscitation of the patient. Although the sonographic definition of tamponade is the simultaneous presence of pericardial fluid and diastolic collapse of the right ventricle or atrium (Figure 38–11), the presence of pericardial fluid in a patient with chest trauma is highly suggestive of a patient with pericardial hemorrhage (Figure 38–12). An indirect sonographic sign of tamponade is the demonstration of a dilated inferior vena cava in a hypotensive patient. EDs performing cardiac ultrasonography using subcostal and long parasternal views have reported a sensitivity of 98.1% and a specificity of 99.9% for the detection of pericardial effusion. Because US is noninvasive and extremely accurate, its immediate availability in the initial phase of a major trauma resuscitation can be very helpful, as it can detect pericardial fluid before the patient deteriorates hemodynamically.

**Figure 38–11** Lewis lead ECG revealing total electrical alternans of the QRS complexes. *From Sotolongo RP, Horton DJ: Am Heart J 101:853, 1981.*

**Electrocardiography**

Many ECG changes of pericardial tamponade have been described in the literature, but few are diagnostic, and each is more likely to be seen with chronic rather than acute tamponade. Electrical alternans has been reported to be a highly specific marker of pericardial tamponade. Electrical alternans is an ECG change in which the morphology and amplitude of the P, QRS, and ST-T wave in any single lead alternates in every other beat (Figure 38–13). The postulated cause is the mechanical oscillation of the heart in the pericardial fluid, which is called the swinging heart phenomenon. In uncomplicated pericardial effusion, the heart swings back and forth but returns to approximately the same position before the next systole. Electrical alternans does not occur in this situation.

Echocardiographic studies have revealed that when fluid accumulates to a critical extent and cardiac tamponade ensues, the frequency of cardiac oscillation may abruptly decrease to half the heart rate. The cardiac position will alternate, with the heart returning to its original position with every other beat, and electrical alternans may be seen. Electrical alternans, when present, is pathognomonic for tamponade. However, it is much more common in chronic pericardial effusions that evolve into a tamponade, and it is rarely seen in acute pericardial tamponade.

**Radiography**
The radiographic evaluation of the cardiac silhouette in acute pericardial tamponade generally is not helpful, unless a traumatic pneumopericardium is present. Because small volumes of hemopericardium lead to tamponade in the acute setting, the heart will typically appear normal. This is in contrast to the “water-bottle” appearance of the heart with chronic pericardial effusion. This latter condition is tolerated for a long period (Figures 38–14 and 38–15).

Figure 38-12 Nondescript cardiac silhouette in a patient with acute pericardial tamponade.

Figure 38-13 Air fluid level in pericardium of same patient as in Figure 38–12 immediately after pericardiocentesis.

Figure 38-14 Cardiac ultrasound demonstrating pericardial effusion and right ventricular collapse. PE, Pericardial effusion; RV, right ventricle; LV, left ventricle.
Prehospital Care

Field treatment for cases of pericardial tamponade is essentially the same as outlined for any victim of major trauma. The diagnosis of tamponade should be suspected by the location of penetrating wounds or by the patient's poor response to vigorous volume resuscitation.

Tension pneumothorax, which is much more common, may mimic acute pericardial tamponade. If the patient presents in extremis or the clinical condition rapidly deteriorates, consideration should be given to performing a needle thoracostomy, which, if not therapeutic, suggests pericardial tamponade under the appropriate clinical presentation by virtue of “diagnosis of exclusion.” Expedient transport to the nearest trauma center should be of paramount concern.

Emergency Department

When the patient arrives in the ED, volume expansion with crystalloid solution via two or three large-bore (14-or 16-gauge) catheters should be established immediately. A CVP catheter is helpful for monitoring CVP. It is preferable to place the CVP line on the same side as the trauma to avoid iatrogenic pulmonary or vascular injury to the nontraumatized side. However, if periclavicular trauma has occurred, the catheter should be inserted contralaterally because the integrity of the venous system may be altered on the traumatized side. The presence of a pneumothorax or hemothorax, which is often associated with penetrating cardiac trauma, must be treated expeditiously with tube thoracostomy. If the diagnosis of pericardial tamponade is strongly suspected on clinical grounds or is diagnosed by bedside US, urgent surgical repair is required.

There is increasing controversy with regard to the role of pericardiocentesis. Earlier literature recommended it should be performed for both diagnostic and therapeutic reasons. Aspiration of as little as 5 to 10 ml of blood may result in dramatic clinical improvement. Reducing the total intrapericardial volume to just below the critical level allows compensatory mechanisms to maintain adequate hemodynamics.

However, it should be emphasized that pericardiocentesis is not a benign or invariably successful procedure. Blood in the pericardial space tends to be clotted and aspiration may not be possible. The possible complications include the
production of pericardial tamponade, the laceration of coronary artery or lung, and induction of cardiac dysrhythmias. Whenever possible, pericardiocentesis should be performed under sonographic guidance because this approach will increase success rate and decrease the incidence of complications. A catheter may be introduced into the pericardial space for repeated aspirations while preparations are under way to quickly transport the patient to the operating room for definitive therapy. If pericardiocentesis is unsuccessful or the clinical status deteriorates and if acute pericardial tamponade remains important in the differential diagnosis, thoracotomy should be performed as quickly as possible. Patients with penetrating cardiac injury invariably require surgical repair. The location (operating room versus ED) and timing (immediate versus urgent) depend on the patient’s clinical status.

**Emergency department thoracotomy**

Emergency department thoracotomy (EDT) is a drastic, dramatic, and potentially life-saving procedure in which emergency physicians should be proficient. Although the procedure will not be described in detail here, a few technical points merit discussion. A left lateral incision is preferred because it is rapidly accomplished; allows the best exposure of the heart, aorta, and left hilum; and facilitates open cardiac massage and internal defibrillation. With right-sided or multiple injuries, it may be necessary to extend the incision across the sternum and right chest wall creating a "clam shell" incision. The internal mammary arteries need to be ligated if this maneuver restores effective perfusion. After the heart is sufficiently exposed, the pericardium is incised anterior to the phrenic nerve. Release of a tamponade may rapidly restore cardiac output. The heart is then delivered through the pericardium and penetrating wounds identified.

There are several alternatives for repairing cardiac wounds. Small wounds can be compressed by digital pressure to control bleeding en route to the operating room, or a Foley catheter inserted into the wound with the balloon inflated with saline can temporarily seal the wound with slight traction. The Foley catheter is sutured into position with a purse-string suture. Neither technique is effective for large wounds, however, and more formal repair is required in these cases. Suture of cardiac wounds over pledgets is the time-honored and effective technique but is technically more difficult. Stapling cardiac wounds with standard skin staplers has been shown to be much quicker and equally effective in closing these wounds. Care must be taken to avoid ligating coronary arteries during the repair. Direct insertion of a large-bore catheter (e.g., a 5 Fr catheter) into the left atrial appendage provides a route for rapid infusion of fluids. If the heart is empty or the patient fails to respond to rapid fluid administration, the aorta is cross-clamped to divert cardiac output to the brain and heart. Prolonged ischemia and severe acidosis often result in postresuscitation myocardial depression with ineffective contraction and diminished cardiac output.

**Indications for emergency department thoracotomy**

Although it is often tempting to perform EDT on all traumatic arrest victims presenting to the ED, there are clearly cases in which it has virtually no chance of salvaging the patient. In addition, EDT is costly, requires the undivided attention of all personnel in the ED, diverting care from other more salvageable critical patients, and poses a risk to ED personnel for injury from needle sticks and other blood-contaminated exposures. Consequently, guidelines have been established for performing EDT to restrict the procedure to patients with some chance of achieving a neurologically functional outcome (see Box 38–4).

The absence of field vital signs in patients with blunt trauma appears to be incompatible with survival. Patients with blunt trauma who lose vital signs in the ED are candidates for EDT, although the outcomes remain poor, with less than 2% survival. Patients with penetrating trauma with signs of life in the field, even if only electrical activity on cardiac monitor or agonal respirations, are also candidates for EDT if transport times are less than 10 minutes. Additional contraindications have been suggested including severe acidosis (pH < 6.8) or closed chest CPR for more than 10

<table>
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<tr>
<th>Condition</th>
<th>Survival (%)</th>
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<tbody>
<tr>
<td>Cardiac arrest in field</td>
<td>0</td>
</tr>
<tr>
<td>Cardiac arrest in ED</td>
<td>30</td>
</tr>
<tr>
<td>Agonal in ED</td>
<td>40</td>
</tr>
<tr>
<td>Unresponsive shock in ED</td>
<td>50</td>
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</tbody>
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minutes in an intubated patient or 5 minutes in a nonintubated patient, because no neurologically functional survivors were found beyond these limits.

**Prognosis**

After penetrating wounds to the heart, several factors adversely affect survival. These include GSW mechanisms and wounds that involve the left ventricle, multiple cardiac chambers, intrapericardial great vessels, or one or more coronary arteries. The factors favorable to survival include stab wound mechanisms with minor perforations, isolated right ventricular wounds, a systolic blood pressure greater than 50 mm Hg on arrival at the ED, and the presence of **cardiac tamponade**. For patients who have a systolic blood pressure greater than 50 mm Hg on arrival to the ED, an aggressive approach has resulted in survival rates as high as 95%. The overall survival from EDT ranges from 4% to 16%. The survival of patients with SWs to the heart who reach the operating room is 70% to 80%, and with GSWs, 20% to 40% (Table 38–1).