DIAGNOSIS OF CARDIAC TAMPONADE

Roberts: Clinical Procedures in Emergency Medicine, 4th ed.

Patient Profile and Symptoms

Pericardial effusion is rarely diagnosed based on physical findings. In contrast, pericardial tamponade can be diagnosed based on clinical criteria, but specific clinical signs are often absent. Particularly in the setting of acute hemorrhagic tamponade, the time from the first signs of tamponade to full arrest may be brief.

Classic clinical findings have been described for tamponade. However, these findings are often obvious only when the patient is unstable due to tamponade. Ideally, tamponade is diagnosed early, when the patient suffers no more than dyspnea, weakness, or perhaps right heart failure. It is common to attribute respiratory symptoms (e.g., dyspnea on exertion) to a more common condition such as heart failure or pulmonary pathology and to overlook pericardial effusion until the classic late signs (e.g., hypotension) appear.

Acute pericardial tamponade may resemble tension pneumothorax, acute hemothorax, hypovolemia, pulmonary edema, or pulmonary embolism. Severe right ventricular contusion can mimic the findings of tamponade. The patient is often agitated or panic-stricken, confused, uncooperative, restless, cyanotic, diaphoretic, and acutely short of breath. In the late stages, the patient is moribund. Hypotension in the presence of severe cyanosis and distended neck veins is a helpful but late finding.

Physical Signs

The classic physical findings of tamponade were first characterized by Beck in 1935. He described two triads, one for acute and one for chronic compression. The chronic compression triad consists of high CVP; ascites; and a small, quiet heart. The triad in acute compression consists of high CVP, decreased arterial pressure, and muffled heart sounds. Unfortunately, in most major trauma series, only about one third of patients demonstrate the complete acute triad, although almost 90% have one or more signs. *The simultaneous occurrence of all three physical signs is a very late manifestation of tamponade and is usually seen most consistently shortly before cardiac arrest*.

Careful hemodynamic monitoring reveals earlier changes that indicate the progression of tamponade. In grade I tamponade, cardiac output and arterial pressure are normal, but CVP and heart rate are increased. In grade II tamponade, blood pressure is normal or slightly decreased, CVP is increased, and tachycardia persists. In grade III tamponade, the classic findings of Beck's acute triad occur. Although this sequence represents the natural history of acute tamponade, the time course varies. Some patients are stable at a given stage for hours; others proceed to cardiac arrest within minutes. Unfortunately, not all patients with early tamponade respond with a predictable pattern of change in vital signs. Brown and coworkers found that 6 of 18 patients with tamponade, defined through right heart catheterization, responded to tamponade with elevated systolic blood pressure. After **pericardiocentesis**, these patients had a marked reduction in systolic blood pressure accompanied by increased cardiac output. All of these patients had previously been hypertensive.

Pulsus paradoxus is defined as an exaggeration of the normal inspiratory fall in blood pressure. A paradoxical pulse (pressure) is one of the classic physical signs of tamponade, but it is not pathognomonic. It is also caused by pulmonary emphysema, asthma, labored respirations, obesity, cardiac failure, constrictive pericarditis, pulmonary embolism, and cardiogenic shock. Measuring the paradoxical pulse is difficult and time-consuming, and any frightened, hypotensive patient with labored breathing can demonstrate this finding.

If the difference between inspiratory and expiratory systolic blood pressures is greater than 12 mm Hg, the paradoxical pulse is abnormally high. Most patients with proven tamponade will demonstrate a difference of 20 to 30 mm Hg or more during the respiratory cycle. This may not be true of patients with very narrow pulse pressures (typical of grade III tamponade); they will have a "deceptively small" paradoxical pulse of 5 to 15 mm Hg. The decreased *pulsus paradoxus* with hypotension occurs because the paradoxical pulse is a function of actual pulse pressure, and the inspiratory systolic pressure may be below the level at which diastolic sounds disappear. For this reason, the ratio of the paradoxical pulse to the pulse pressure is a more reliable measure. A paradoxical pulse greater than 50% of the pulse pressure is abnormal.

Pulsus paradoxus in tamponade has been correlated with the degree of impairment of cardiac output. In atraumatic patients, a 15% *pulsus paradoxus* in the face of relative hypotension was found in 97% of patients with moderate or severe tamponade and only 6% of patients with absent or mild tamponade. A similar study of right ventricular diastolic collapse by echocardiography found that an abnormal *pulsus paradoxus* had a sensitivity of 79%, a specificity of 40%, a positive predictive value of 81%, and a negative predictive value of 40%.

Grade	Pericardial Volume (mL)	Cardiac Index	Stroke Index	Mean Arterial Pressure	CVP	Heart Rate	Beck's Triad
I	<200	Normal or ↑	Normal or ↓	Normal	↑	↑	Venous distention, hypotension, muffled heart sounds usually not present
II	≥200	↓	↓	Normal or ↓	↑ (≥12 cm H_2 O)	↑	May or may not be present
III	>200	↓↓	↓↓	↓↓	↑↑ (up to 30–40 cm H_2 O)	↓	Usually present

TABLE 16-3 -- Shoemaker System of Grading Cardiac Tamponade

From Shoemaker WC, Carey SJ, Yao ST, et al: Hemodynamic monitoring for physiologic evaluation, diagnosis, and therapy of acute hemopericardial tamponade from penetrating wounds. J Trauma 13:36, 1973.

The absence of a paradoxical pulse does not rule out tamponade. Although the mean paradoxical pulse was 49 mm Hg in one series of nonhemorrhagic tamponade, 23% of the patients had a paradoxical pulse of less than 20 mm Hg, and 1 patient had no measurable paradoxical pulse. An abnormal *pulsus paradoxus* has been reported to be absent in tamponade when there is an atrial septal defect, aortic insufficiency, localized collections of pericardial blood, or extreme tamponade with hypotension. It may also be absent when left ventricular diastolic pressure is intrinsically elevated owing to poor left ventricular compliance. This was seen in one half of uremic patients with tamponade. In traumatic tamponade, *pulsus paradoxus* is deemed unreliable. In one study, only 35% of trauma patients had an abnormal paradoxical pulse when elevated CVP and decreased heart sounds were present. In another study of 197 traumatic cases, only 8.6% of the diagnoses of tamponade were made by finding an abnormal *pulsus paradoxus*.
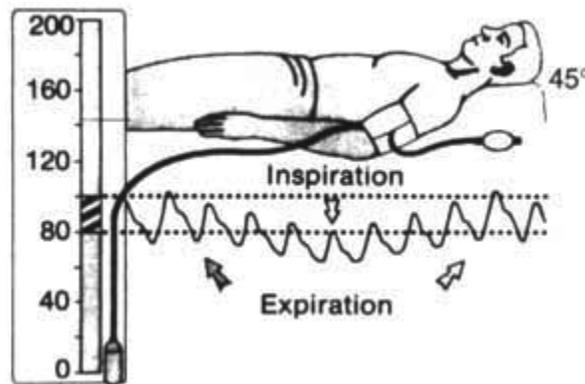

Figure 16-4 Normally systolic blood pressure drops slightly during inspiration. To measure *pulsus paradoxus*, the patient breathes normally while lying at a 45-degree angle. The blood pressure cuff is inflated well above systolic pressure and slowly deflated. When the pulse is first heard only during expiration, this is the upper value. The cuff is deflated until the pulse is heard during both inspiration and expiration, and this is the lower value. The difference in the two values is the amount of *pulsus paradoxus*. A difference of more than 12 mm Hg is abnormal.

Although the absence of a paradoxical pulse rules against severe tamponade, it does not completely rule it out. Whether time is taken to determine *pulsus paradoxus* depends on the patient's status. If the patient is moribund or rapidly deteriorating, taking time to check this parameter is obviously a poor choice of priorities.

Venous Distention

Venous distention, reflecting increased CVP, is also a late sign in cardiac tamponade. It may be masked by venoconstriction as a result of vasopressors (e.g., dopamine), intrinsic sympathetic discharge, or hypovolemia. Neck vein distention may be obvious clinically, but the measured CVP is more reliable than the presence of venous distention. The CVP reading should take into account positive-pressure ventilation and the effects of a Valsalva maneuver. Most patients with significant tamponade will have a CVP of greater than or equal to 12 to 14 cm H_2O. Hypovolemia changes the intrapericardial pressure-volume curve in tamponade and will lower the CVP reading at any given stage in the tamponade process.

Animal studies have documented that right atrial pressure can be normal in tamponade when hypovolemia is present. One case of low-pressure cardiac tamponade was reported in a patient with no jugular venous distention, no paradoxical pulse, and a right atrial pressure of 8 mm Hg. Thus, although the initial CVP reading is useful and diagnostic if grossly elevated (e.g., 20 to 30 cm H_2O), a series of CVP readings looking for an upward trend is the most sensitive diagnostic tool. A rising CVP, especially when there is persistent hypotension, is extremely suggestive of tamponade in the trauma patient. In the rare case of the hypovolemic patient in whom tamponade is suspected but who demonstrates a low CVP, a fluid challenge will help clarify the situation and will also improve cardiac output at least temporarily.

Ancillary Testing

Routine chest radiographs and electrocardiograms may be useful in increasing the level of suspicion for pericardial effusion and tamponade. Noninvasive diagnosis of effusion, however, must be made by computed tomography or, preferably, cardiac ultrasound. If available, bedside ultrasound is the fastest and most reliable for the emergency clinician to demonstrate a significant pericardial effusion, although it may not be diagnostic of tamponade.

Chest Radiographs

Chest radiographs are not useful in the diagnosis of acute traumatic tamponade, because *the cardiac size and shape do not change acutely*. However, the radiographs may reveal hemothorax, bullet location, or even pneumopericardium.

In the patient without trauma and with chronic effusion, a chest film often reveals an enlarged, sac-like "water bottle" cardiac shadow. Unfortunately, it is difficult to differentiate pericardial from myocardial enlargement, and radiographs cannot be used to distinguish between simple pericardial effusion and tamponade. One finding that is useful in identifying effusion on the plain chest film is the epicardial fat pad sign. The water density space between the radiolucent epicardial fat and the mediastinal fat represents the pericardial tissues and is normally less than 2 mm. An increase in this width suggests pericardial fluid or thickening. This sign may be seen in 41% of upright lateral and 23% of frontal chest films in proven pericardial effusion. The diagnostic value may be enhanced by using a supine rather than upright cross-table, lateral chest radiograph. Obtaining a supine lateral film increases the sensitivity of the epicardial fat pad sign from 31% to 51%.

Electrocardiograms

Electrocardiograms may suggest, but should *not* be used to diagnose, pericardial effusion or cardiac tamponade. Most electrocardiogram changes, such as PR-segment depression, low-voltage QRS complexes, and electrical alternans, have acceptable specificity but poor sensitivity for pericardial effusion or tamponade. Low voltage is defined as a QRS amplitude less than or equal to 5 mV in all limb leads (or a sum of the limb lead QRS amplitude less than or equal to 30 mV), and PR depression is defined as greater than or equal to 1 mV depression in at least 1 lead other than aVR. In a study correlating the electrocardiogram with echocardiographic evaluation, electrocardiogram signs had an overall sensitivity of only 1% to 17% and a specificity of 89% to 100% for pericardial effusion. Others have demonstrated significantly higher sensitivity, i.e., in the range of 32 to 68% for voltage criteria. PR-segment depression is the most common electrocardiogram finding in pericardial tamponade, and low voltage is most commonly associated with a moderate to large effusion. It is important to note that none of the ECG findings differentiate tamponade from effusion.

Electrical alternans is caused by pendulum motion of the heart within the pericardial sac. Alternans of the QRS complex has been seen in about 22% of medical tamponade cases[72] but in only 5% of cancer patients with tamponade. Electrical alternans of both the P wave and the QRS complex (total electrical alternans) is a rare finding,

but when seen is thought to be pathognomonic of tamponade. Like electrical alternans, low voltage may be a finding associated with tamponade, but not simple effusion.
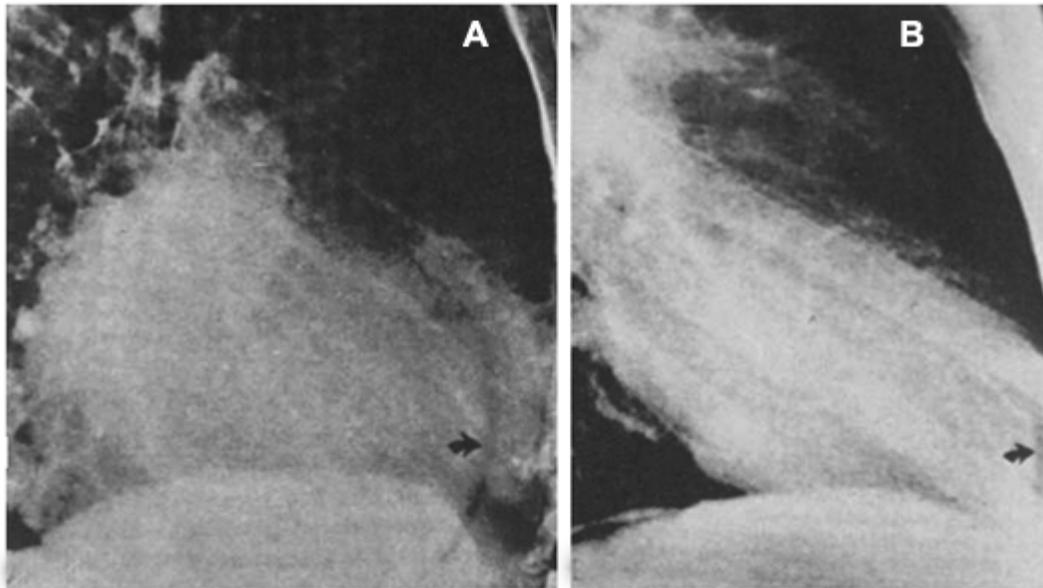

Figure 16-5 Epicardial fat pad sign. The water density space between the radiolucent epicardial fat and the mediastinal fat represents the pericardium and its contents and should be 2 mm or less. An increase suggests pericardial fluid or thickening. *A,* Left anterior-oblique chest film. *B,* Lateral chest film. In acute tamponade, the chest radiograph has very minimal diagnostic value.
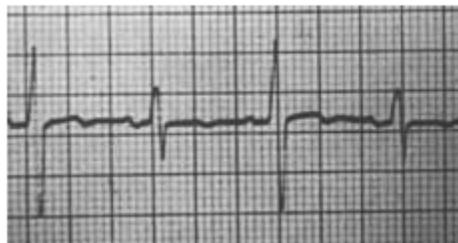

Figure 16-6 Overall, the electrocardiogram (ECG) has a low sensitivity for pericardial effusion or tamponade, but PR depression, low voltage, or electrical alternans may be seen. Lewis lead ECG showing total electrical alternans of both amplitude and configuration of P and QRS complexes. This is rarely seen but is highly suggestive of tamponade. Note that electrical alternans may not be evident in standard ECG leads. *(From Sotolongo RP, Horton JD: Total electrical alternans in pericardial tamponade. Am Heart J 101:854, 1981. Reproduced by permission.)*

Echocardiography

Echocardiography is the best available tool for diagnosing pericardial effusion and has the further advantage of being noninvasive. Echocardiography is very sensitive in the diagnosis of pericardial effusion and tamponade.

The disadvantages of echocardiography are that it requires ultrasound equipment and is dependent on a skilled operator who is specifically trained in echocardiography. Even when immediately available, echocardiography may take at least 5 minutes, which may be too much time for a patient who is deteriorating rapidly. If the patient in not in full arrest and ultrasound is available, ultrasound should always be used to diagnose effusion and tamponade and to guide the procedure. Pericardial fluid is relatively easy to demonstrate with bedside ultrasound, but since many ill patients will demonstrate some pericardial fluid, bedside ultrasound may not be able to differentiate incidental fluid from tamponade.

Computed Tomography

At some institutions, computed tomography (CT) is much more readily available than echocardiography. However, it requires that the patient be transported to the site of the CT equipment and patient stability must be considered. If clinically indicated, CT is effective in defining the presence and extent of pericardial effusion in the stable patient. In certain circumstances, CT can provide a more definitive diagnosis than echocardiography. In one series, eight equivocal echocardiograms were evaluated by CT. Two patients thought to have pericardial effusion by ultrasound were found by CT to have pleural effusions. Another patient with pericardial effusion by ultrasound was found by CT to have an epicardial lipoma. CT defined three loculated pleural effusions not seen by ultrasound. A final two patients had hemopericardium visualized by CT but not ultrasound. In circumstances where the patient is stable and ultrasound produces equivocal results or is not available, CT may provide a definitive diagnosis of pericardial effusion.

INDICATIONS FOR PERICARDIOCENTESIS

There are two indications for **pericardiocentesis**: (1) to diagnose the cause or presence of a pericardial effusion and (2) to relieve tamponade. The former is an elective procedure and ideally should be accomplished under ultrasound guidance. The latter may be semi-elective and performed with ultrasound guidance or emergent and performed blind with ECG assistance.

Diagnostic Pericardiocentesis

The use of **pericardiocentesis** for diagnosis of the etiology of nonhemorrhagic effusions is widespread, although opinions of its utility vary. Neoplastic cells, blood, bacteria, viruses, and chyle can be sought. Measurement of pericardial fluid pH can be helpful, because inflammatory fluid is significantly more acidotic than noninflammatory fluid. When a specific etiology is suspected, additional diagnostic testing may be useful (e.g., adenosine deaminase in tuberculosis, and carcinoembryonic antigen in suspected malignancy).

The diagnostic accuracy of **pericardiocentesis** varies greatly from series to series, depending on the vigor with which a definitive etiology was sought and the prevalence of certain etiologies in the patient population under consideration. In one large series, fluid was obtained in 90% of the taps, but a specific etiologic diagnosis was obtained in only 24% of the fluid specimens. Certain diagnoses are unlikely to be made from pericardial fluid. Pericardial fluid has been shown to give false-negative cytologic results in several cases of lymphoma and mesothelioma. In HIV patients, effusions caused by Kaposi sarcoma and cytomegalovirus have been diagnosed by pericardial biopsy after fluid studies were non-diagnostic.

An alternative diagnostic tool is subxiphoid pericardiotomy. This technique, performed in the operating suite, obtains both fluid and a pericardial biopsy specimen. It is more likely to provide a definite diagnosis and has been performed safely without general anesthesia. In a prospective series of 57 patients, 36% obtained a definitive diagnosis; 40%, a probable diagnosis; 16%, a possible diagnosis; and 7% remained undiagnosed with subxiphoid pericardiotomy. Although it is uncertain whether this technique is safer than ultrasound-guided **pericardiocentesis**, published reports show a low rate of complications in experienced hands.

Regardless of technique, the need to sample small effusions or obtain pericardial tissue has been questioned. A prospective series found a diagnostic rate of 6% with pericardial fluid and 5% with pericardial tissue when a small persistent effusion was sampled for the specific purpose of diagnosis. In contrast, when patients from the same population had therapeutic intervention for tamponade, the yields from fluid and tissue were 54% and 22%, respectively.

The use of **pericardiocentesis** as a diagnostic tool in traumatic tamponade is limited. When used diagnostically to determine the presence of pericardial bleeding in trauma, the procedure has a false-negative rate of between 20% and 40%. The reason for the high false-negative rate (defined as no blood aspirated) is well demonstrated by typical stab wounds of the heart. Ninety-six percent of the patients had blood in the pericardium, but it was clotted in 41% of the patients and partially clotted in another 24%. In only 19% was the blood completely fluid and thus capable of giving a true-positive result on **pericardiocentesis**.

Therapeutic Pericardiocentesis

Tamponade of Uncertain Etiology

The primary reason for performing **pericardiocentesis** in the ED is as part of the treatment for cardiac arrest or in peri-arrest situations. In particular, the presentation of PEA with elevated jugular venous pressure should cause immediate consideration of **pericardiocentesis**. In this setting, blind, ECG-guided **pericardiocentesis** can be life saving. However, the overwhelming majority of patients with PEA have neither significant effusion nor tamponade, and other etiologies for the PEA also should be sought. **Pericardiocentesis** also may be considered in other presentations of effusion with existing or incipient tamponade.
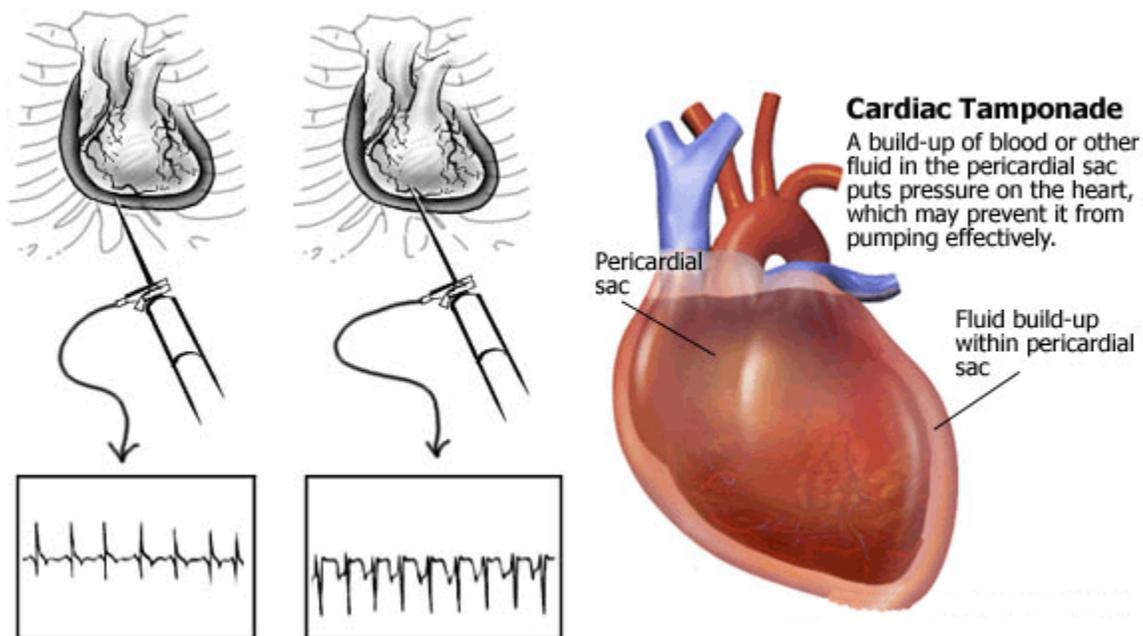
Tamponade Caused by Nonhemorrhagic Effusions

Pericardiocentesis is often, at least temporarily, therapeutic in cardiac tamponade. Most nonhemorrhagic effusions are liquid and can be drained easily through a small needle. Removal of even a small amount of fluid can immediately and dramatically improve blood pressure and cardiac output. **Pericardiocentesis** relieves tamponade due to nonhemorrhagic effusions in 60% to 90% of cases. Patients in whom it fails often have purulent pericarditis or malignant invasion of the pericardium.

Pericardiocentesis without catheter placement may be much less useful for long-term management of these patients; 26% of the patients in the study by Guberman and coworkers eventually required pericardial resection. In Krikorian's series, 24% of the patients were managed successfully with one **pericardiocentesis**, 37% needed multiple taps or an indwelling catheter, and 39% required surgical drainage. Fifty-five percent of the last group had traumatic hemopericardium.

Patients with renal failure and pericardial effusion may be better managed by dialysis than **pericardiocentesis**. In one series, 63% of these patients were successfully managed with dialysis alone, and only 6% needed surgical treatment over the long term. Tamponade is less frequent with pericarditis when it occurs within the first months of dialysis, and such patients are much more likely to be successfully managed without invasive intervention. When invasive treatment is needed for dialysis patients, **pericardiocentesis** is probably a poor choice; 9 of 10 patients who received it had complications in one series, and it was the only invasive treatment that resulted in death.

An algorithm for the urgent management of nonhemorrhagic cardiac tamponade is shown.

Cardiac Tamponade
A build-up of blood or other fluid in the pericardial sac puts pressure on the heart, which may prevent it from pumping effectively.

Pericardial sac

Fluid build-up within pericardial sac

Use in Hemorrhagic Tamponade

Pericardiocentesis is never the definitive treatment in hemorrhagic tamponade.

Although aspiration of a small quantity of fluid may cause dramatic improvement, blood usually reaccumulates. Thus, patients with pericardial hemorrhage ultimately require thoracotomy to explore and repair the cardiac injury.

One of the greatest potential drawbacks of **pericardiocentesis** in traumatic tamponade is that it may delay thoracotomy. In one study of 25 trauma patients with cardiac injury, all of those who were operated on within 2 hours of injury survived, regardless of age or type of wound. With greater delay, none survived. Sugg and colleagues, in a study of 459 similar patients, found a mortality rate of 43% when **pericardiocentesis** was the sole treatment, but only 16% when surgery was performed. Most investigators agree that with early thoracotomy and little or no reliance on **pericardiocentesis**, the number of deaths due to stab wounds has decreased. Sugg and associates reported that 10 of 18 patients with traumatic tamponade who were managed by repeated **pericardiocentesis** alone died within 1 to 2 hours. At autopsy, all patients had repairable wounds.

Nonetheless, while other temporizing treatments are instituted and arrangements for definitive surgical treatment are being made, **pericardiocentesis** may temporarily improve the patient's hemodynamic situation. Some clinical evidence supports the usefulness of **pericardiocentesis** as a temporizing measure. In a study of 174 patients with tamponade from penetrating trauma, 96 underwent operating room thoracotomy, 44 underwent ED thoracotomy, and 34 received only **pericardiocentesis** followed by observation. Of those who underwent operating room thoracotomy, 68% were hemodynamically unstable, and preoperative **pericardiocentesis** decreased the mortality rate from 25% to 11%. Ninety-one percent of those who underwent ED thoractomy were unstable, and pre-thoracotomy **pericardiocentesis** decreased the mortality rate from 94% to 63%. For the unconscious and hypotensive or agonal patient, emergency thoracotomy is the preferred treatment.

When a trauma patient's condition is relatively stable, but a high level of suspicion for a penetrating cardiac wound is present, an alternative to thoracotomy is the subxiphoid pericardial window. The procedure has been done under local anesthesia. Although it is possible to perform the procedure in the ED, most authors believe the procedure should be reserved for the operating suite.

CONTRAINDICATIONS

There is no absolute contraindication to **pericardiocentesis**. It should not be performed when better treatment modalities are immediately available (e.g., dialysis for uremic patients and immediate surgery for trauma patients). For diagnostic or non-emergent **pericardiocentesis**, echocardiographic or CT diagnosis is imperative. Ultrasound or fluoroscopic guidance should be used in all non-emergent situations.