

Penetrating Cardiac Trauma

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Penetrating injuries to the heart are fatal in a significant percentage of patients so injured. In fact, many patients are found dead at the scene. However, patients who arrive at the hospital with intact vital signs have a good chance of survival if well managed. The following discussion describes the natural history, initial evaluation, and issues that will affect anesthetic management of patients experiencing penetrating cardiac injury.

Penetrating Cardiac Trauma

Despite improvements in trauma care, penetrating injuries to the heart continue as a source of significant mortality. A South African study found only 6% of patients with these injuries reached the hospital alive.¹ In Seattle, a city known for the quality of prehospital care, the overall survival in patients receiving CPR was only 17%.² With statistics so ominous, it is clear that an organized approach to the management of such patients is essential.

Most penetrating cardiac injuries are secondary to violent acts or industrial accidents. Penetration with sharp objects is associated in general with a better outcome than penetration resulting from gunshot. Penetrating injuries may also be iatrogenic, such as secondary to cardiopulmonary resuscitation (CPR), central venous catheterization, or percutaneous cardiac procedures. Fractures of the sternum or ribs may occasionally penetrate the heart.

The anatomic position of the heart within the thorax determines to some degree the injury encountered.³ The right and left ventricles each are injured about 40% of the time. The frequency of right atrial injury is 24% and that of left atrium injury, 3%. Injuries are classified as complex if they create shunts, disrupt valves or papillary muscle apparatus, or lacerate coronary arteries (~5% of injuries).

Prehospital Management

"Scoop and go" is an absolute prehospital principle of the management of patients suspected to have penetrating cardiac injuries.⁴ The physiologic status of the patient at hospital presentation, as well as the mechanism of injury (stabbing vs. gunshot) and the presence of cardiac tamponade (limiting exsanguination) have been identified as significant prognostic factors in outcome for penetrating injuries.⁵ In a retrospective review of 302 patients with penetrating cardiac injuries, Tyburski et al found that every patient with no vital signs at the scene died, even those who redeveloped some cardiac activity.⁶ Only 4% of patients arresting in the ambulance survived. All

patients having gunshot wounds and requiring emergency department (ED) thoracotomy died. But 74% of the patients who arrived in the operating room (OR) and underwent thoracotomy survived. Clearly, these injuries are ominous and require rapid transfer to a center experienced in managing such patients.

Initial Assessment

Though there may be the occasional patient who presents with normal vital signs, penetrating injuries typically present in two ways: either in cardiac tamponade or with hypovolemia (or some combination of the two).⁵ Small lacerations often tamponade, as the fibrous pericardium tends to seal, accumulating blood and clot within the pericardial space. Though small amounts of blood may defibrinate in the pericardial space, vigorous bleeding results in clot formation. Interestingly, bleeding into the pericardium may have a protective effect because it prevents exsanguination into the left hemithorax. Patients with missile injuries often exsanguinate and have a high probability of associated intrathoracic injuries. Depending on the phase of respiration and diaphragmatic position when injury occurred, intraabdominal viscera may be penetrated. Similarly, cardiac injury with abdominal penetrating injuries should be considered.

Chest radiographs are of limited value in the initial assessment of the patient with penetrating cardiac injury. The cardiac silhouette is not enlarged in about 80% of patients in acute cardiac tamponade, as the pericardium has not had sufficient time to stretch. At least 250 ml of pericardial fluid must be present to detect heart enlargement radiographically. Occasionally pneumopericardium may be detected. The electrocardiogram may suggest tamponade if QRS voltage is decreased or if the dominant QRS axis constantly changes (electrical alternans) secondary to the heart "floating" in the pericardium. ST segment elevation may be observed. A normal electrocardiogram (ECG) does not rule out a cardiac injury.

Most certainly of greater value is transthoracic echocardiography. Nagy et al retrospectively reviewed 121 clinically stable patients with penetrating wounds in proximity to the heart.⁷ Twelve penetrating cardiac injuries were detected and managed successfully and no significant injuries were missed. Two-dimensional echocardiography is a rapid, noninvasive, and accurate tool for evaluating penetrating cardiac trauma. It has greater than 90% accuracy, specificity, and sensitivity in detecting pericardial effusions, and technical performance is learned quickly. Cardiac chamber filling can be assessed as can impingement of chamber filling due to tamponade. It has largely supplanted pericardial windows as a *diagnostic* entity, though pericardial windows may continue as a diagnostic mainstay where echo capabilities do not exist. Hemothoraces may make echo evaluation more difficult; in this setting, pericardial window may be valuable. In patients intubated and receiving positive end expiratory pressure (PEEP), transesophageal echo may be more valuable than transthoracic echo.

An algorithm for the initial assessment of penetrating injuries is presented in Figure 1.

Cardiac Tamponade

A distinction should be made between the tamponade often observed in medical patients, which has developed over a protracted time and is associated with a markedly enlarged pericardial sac and perhaps a liter or two of pericardial fluid and the acute tamponade associated with trauma and a fundamentally nondistensible pericardium. Presenting signs and symptoms differ somewhat, as do compensatory mechanisms.^{8,9} This discussion focuses on acute cardiac tamponade associated with trauma.

Tamponade should be considered whenever hypotension is associated with a penetrating lesion in the chest or epigastrium.¹⁰ Acutely, approximately 100 to 200 ml of blood accumulating within the pericardium may result in physiologic trespass. Patients may present agitated or combative. Cool, vasoconstricted extremities are a common but nonspecific sign. An exaggeration of the normal 3- to 6-mmHg drop in systolic blood pressure with inspiration may be observed. This is called *pulsus paradoxus* when the inspiratory systolic decrease is greater than 10 mmHg. Initial compensatory mechanisms in tamponade include tachycardia, increased contractility, and enhanced right ventricular preload. However, as fluid increases within the pericardial space, pericardial pressure increases to the level of right ventricular end-diastolic pressure (RVEDP). Until this point, cardiac output is maintained. This is an early phase of the so-called equalization of pressures. As more fluid accumulates, pericardial pressure and RVEDP rise and equi-

brate with left ventricular end-diastolic pressure, and by this time, decreased cardiac output is apparent. Myocardial ischemia may occur as coronary perfusion pressure (MAP-LVEDP) decreases.

Though the false-positive rate may be as high as 50%, a temporizing measure often successful in the management of cardiac tamponade is pericardiocentesis. A large-gauge needle or needle-catheter combination is introduced into the pericardial sac and bloody fluid withdrawn. Leaving a catheter in the pericardium has the advantage of allowing further withdrawals of blood if the patient's hemodynamic picture again deteriorates—not an infrequent event. Pericardiocentesis may induce dysrhythmias or lacerate myocardium or coronary arteries. Clotted pericardial blood may produce false negatives.

Perioperative Management

Patients with penetrating cardiac injuries can present very quickly to the OR and can be reasonably stable or absolutely moribund, so it is difficult to make blanket recommendations. A few generalizations are useful.

A patient presenting with a penetrating cardiac injury should be assumed to be hypovolemic and in some degree of tamponade. Distended neck veins and muffled heart tones, two of the three components of the classic “Beck’s triad,” are likely to be absent or missed within the OR environment. Arterial and central venous pressure monitoring both clearly have advantages but perhaps more essential is good intravenous access.

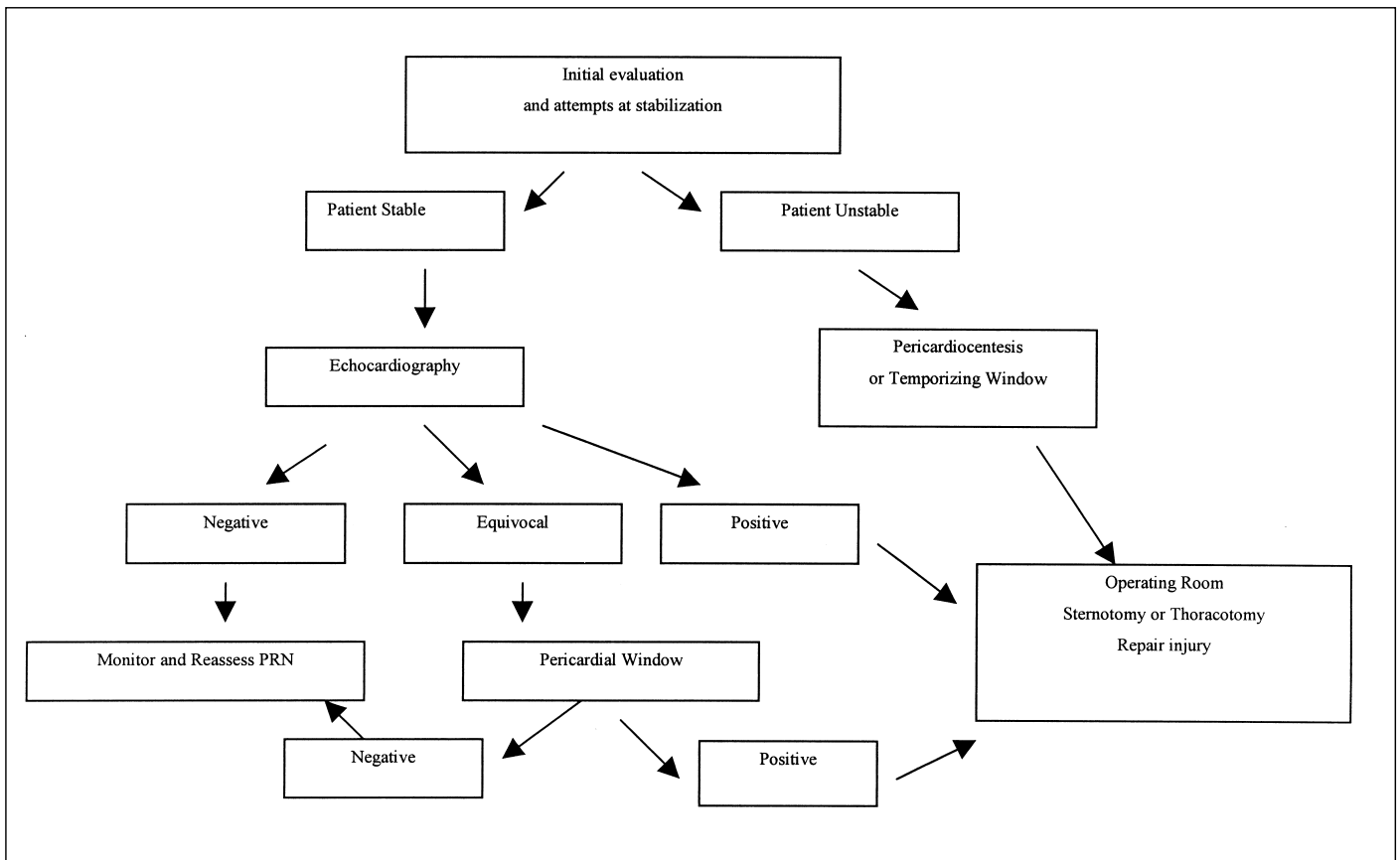


Figure 1. Initial assessment of suspected penetrating cardiac injuries. (Modified from Ivatury RR. The injured heart. In Mattox KL, Feliciano DV, Moore EE, eds. *Trauma*, 4th Ed. New York, McGraw-Hill, 2000.)

The value of fluid resuscitation should never be underestimated. Filling pressures of 25 to 30 mmHg may be necessary to oppose the resistance to heart filling presented by an expanding pericardial volume. Pressure in central veins must exceed RVEDP to ensure cardiac filling.

General anesthetic induction is potentially catastrophic until the bloody pericardial effusion is released. Though pericardiocentesis may be a successful temporizing measure, a pericardial window is more appropriate, because clots can be evacuated. This procedure can be undertaken with local anesthesia supplemented by ketamine, administered intravenously in divided doses; fentanyl administered cautiously in divided doses is also acceptable. Interestingly, while a compensatory tachycardia is expected, stretching of the pericardium may incite vagally mediated bradycardia. These patients are absolutely dependent on an increased heart rate to maintain cardiac output, and administration of atropine is indicated in this instance. A word of caution: paralysis and endotracheal intubation should be undertaken thoughtfully, as positive-pressure ventilation reduces right ventricular preload and can decrease cardiac output acutely.¹¹ PEEP is even worse, be it dialed-in PEEP or auto-PEEP. Awake intubation with topical anesthesia allows spontaneous ventilation but is a time-consuming process, requires a cooperative patient, and may delay definitive therapy. The patient is probably in an enhanced contractile state due to sympathetic stimulation, and the administration of inotropes is almost never indicated. Vasodilator therapy is likely to result in precipitous hypotension secondary to hypovolemia. Severe metabolic acidosis should be treated, as contractility may be impaired. Urine output should be followed as in all trauma patients. Air embolism may occur, especially with injuries to low-pressure chambers, and should be considered in the setting of precipitous onset of arrhythmias or cardiac arrest. Air may be observed in the coronary arteries.

Postoperative Complications

Occasionally, lesions are recognized days after the initial injury. These are often intracardiac shunts, valvular decompensation (manifesting as hypotension, pulmonary edema, and new murmurs), and ventricular aneurysms. Echocardiography and cardiac catheterization are often needed to characterize the abnormalities. The patient may also develop the postpericardiotomy syndrome, manifesting as fever, chest pain, pleural effusions, pleural rub, and an ECG consistent with pericarditis (diffuse ST segment abnormalities). These patients are often treated with nonsteroidal antiinflammatory drugs.

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