Blunt Cardiac Injury

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Abstract

The diagnosis and management of blunt cardiac injury, formerly known as myocardial contusion, has challenged clinicians for decades. Caused primarily by motor vehicle collisions, significant blunt cardiac injury carries a high mortality rate. Yet no reliable diagnostic test exists to identify those patients at greatest risk for an adverse outcome. A literature search using the MEDLINE database was performed to compose a review of epidemiology, diagnostic intervention, and therapeutic approach.

The results of the search indicate that, along with a high index of suspicion, utilizing a combination of electrocardiogram, troponin, and echocardiography for appropriate patients may improve the diagnosis, risk stratification and disposition of patients sustaining blunt cardiac injury.

Key Words: Myocardial contusion, myocardial concussion, myocardial rupture, blunt cardiac injury, pericardial tamponade, blunt trauma, echocardiography, pericardiocentesis, cardiogenic failure.

Introduction

The term "blunt cardiac injury" (BCI) describes a spectrum of myocardial lesions acquired from non-penetrating mechanisms. Sequelae from these injuries range from the benign to catastrophic. Traumatic injuries remain the fifth leading killer of Americans (1). It has been estimated that in the prehospital setting, 20% of traumatic deaths are caused by cardiac-related injuries (2). Over the past 2 decades, the improvement of prehospital transport and early implementation of advanced life support (ALS) have enabled victims of formerly nonsurvivable cardiac injuries to arrive in advance of terminal shock. A heightened level of suspicion and early identification of the blunt cardiac injury is vital. Reliable detection, however is challenging, as there are still no diagnostic criteria for blunt cardiac injury (3).

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Accepted for publication July 2005.

Nomenclature

Historically, the term "myocardial contusion and concussion" was criticized for its lack of specificity for severity and injury pattern. "Myocardial contusion" was used as a general term encompassing the gamut of myocardial injuries. The true incidence of myocardial injury is difficult to discern, since studies differ in their diagnostic criteria. The reported incidence of myocardial contusion ranges from 17–70% in different study populations despite similar mechanisms of injury (2, 3). The interpretation of literature using these terms remains problematic and fails to assist in the identification of patients at greatest risk. In 1992, Mattox recommended the replacement of "myocardial contusion and concussion" with "blunt cardiac injury" along with specific descriptors to provide less ambiguous identification of the injury and its sequelae (Table) (3).

The following review presents an evidence-based approach to the evaluation and management of the patient who presents with blunt thoracic injury that may involve a cardiac injury.

Epidemiology

The Centers for Disease Control and Prevention (CDC) reports that approximately 30,000 pa-

TABLE
Types of Blunt Cardiac Injury (3)

Blunt cardiac injury with minor ECG or enzyme abnormality
Blunt cardiac injury with complex arrhythmia
Blunt cardiac injury with coronary artery thrombosis
Blunt cardiac injury with free wall rupture
Blunt cardiac injury with septal rupture
Blunt cardiac injury with cardiac failure

Copied with permission from Mattox KL, Flint LM, Carrico CJ, et al. Blunt cardiac injury. J Trauma 1992; 33(5):649 – 650 (3).

tients per year with a diagnosis of BCI survive to discharge (4). While a majority of cardiac injuries are sustained during motor vehicle collisions (MVC), falls, blast injuries, and other blunt mechanisms have also been described (2, 5). The contused myocardium is characterized by muscle necrosis, edema, and hemorrhagic infiltrate. Outcomes vary from asymptomatic electrocardiographic changes to cardiogenic shock and death.

The spectrum of injuries is dependent upon the mechanism and degree of force (2, 5). Parmley et al., in a 1956 autopsy study of 546 non-penetrating cardiac trauma cases, found that 64% had at least one chamber rupture, 20% had two or more, 23% had a myocardial contusion, 6% had pericardial lacerations, 4% had a papillary muscle rupture, 2% had a valvular rupture, and 1% had a coronary artery laceration (2). The authors describe 7 categories of force resulting in cardiac injury: (a) direct, (b) indirect, (c) bidirectional or compressive, (d) decelerative, (e) blast, (f) concussive, and (g) combined (2). Direct, compressive, decelerative, and concussive forces are discussed below.

Direct precordial impact and crush injuries result from compression between the sternum and spine. Injuries to the pericardium, myocardium and coronary vessels are characteristic (2, 6). The compressible thoracic and abdominal cavities are adjacent to the heart. Forceful, abrupt pressure fluctuations in these compartments can be distributed to the heart, resulting in tears to its structural walls and valves (2, 6, 7). Because the heart is a dynamic organ, movements in the chest cavity are exaggerated during deceleration, causing the heart to strike rigid structures and forcefully tear itself from its static attachments. The aortic attachments, as well as regions between the atria, pulmonary vessels and the vena cava, are especially vulnerable (2, 6).

Concussive force tends to be of low magnitude and is not associated with pathologic lesions to the myocardium or its vessels (2). Further discussion on concussive force is found in the section “Blunt Cardiac Injury with Complex Arrhythmia.” Finally, penetrating cardiac lesions, despite blunt mechanism, may result paradoxically from closed thoracic injuries secondary to fractured ribs or sternum (6, 8). Blunt cardiac injuries, by virtue of the degree of force, present with coexisting injuries. In order of frequency, the following injuries have been shown to be associated with BCI: head injury, rib fracture, extremity injury, hemothorax, sternal fracture, pulmonary contusion, aortic or great vessel injury, pneumothorax, abdominal injuries, and flail chest (9).

Approach to the Patient with Suspected Blunt Cardiac Injury

Cardiac injury can be missed due to concomitant bodily injuries. Patients with neurologic, multiple extremity or organ injuries are especially at risk, as their manifestations can mask those of the injured heart. A high index of suspicion and proper triage is essential.

History

Determine whether the presenting trauma patient has sustained a thoracic injury. MVC patients should be asked specifically whether restraints were used and if there was steering wheel impact to the chest. The most common presenting symptom is chest pain. Assess if there are any cardiac risk factors, including prior myocardial infarction (MI), recent angioplasty or stress test, angina, hypertension, hypercholesterolemia, tobacco use, cocaine use, or family history of early death secondary to an MI. Document medications, particularly beta-blockers or calcium channel blockers, which may alter findings on clinical examination; for example, by masking tachycardia.

Physical Examination

Perform a thorough physical examination with emphasis on vital signs, head and neck, lungs, and heart. Suggestive of cardiac injury are findings of hypotension, jugular venous distension (JVD), tachypnea, wheezing, rhonchi, chest abrasion or ecchymosis, imprints of a seatbelt or steering wheel, chest tenderness, palpable crepitus, flail chest, fractures of the rib(s), sternum or clavicle , distant heart sounds, S3, tachycardia, bradycardia, rub or murmurs. Pulsus paradoxus (excessive decline of systolic pressure during inspiration) is diagnostic of cardiac tamponade (10).

Initial Stabilization

Ensure that the patient has a patent secured airway, supplemental oxygen, adequate intravenous access, and continuous cardiac monitoring. A chest X-ray and ECG are compulsory for the blunt throracic patient (11). If the patient has underlying risk factors for cardiac disease or is over the age of 65 and complaining of chest pain or discomfort, cardiac troponin levels can be obtained (11). If the patient is unable to provide an adequate history or exam, presume that a cardiac injury exists. A Focused Assessment of Sonographic Trauma (FAST) exam should be performed if there is an available ultrasound, with special attention given to the evaluation of the pericardium (12).

Diagnostic Intervention

In the absence of an ideal set of diagnostic criteria, perioperative visualization and autopsy remain the sole methods of reliably diagnosing a myocardial contusion. A number of tests have been used to screen for pathology and stratify patient severity.

Chest Radiography

The standard work-up for all thoracic trauma patients should include a chest X-ray. X-rays lack sensitivity and specificity for cardiac injury. They do however identify other injuries, including aortic disruption, rib or sternal fractures, pulmonary contusions, hemothorax and pneumothorax, all of which are associated with BCI (5, 13).

Electrocardiogram

The electrocardiogram (ECG) is a rapid, inexpensive bedside study, and should be performed on all patients for whom BCI is suspected (11). Foil et al., in a retrospective evaluation of 524 blunt chest trauma patients, found that 85% of the patients with complications (mostly arrhythmias) had an abnormal admission ECG (14). In a retrospective analysis of 359 patients with blunt chest trauma, Biffl et al. identified an abnormal admission ECG (excluding sinus tachycardia) as the most significant independent predictor of a complication, defined as dysrhythmia requiring intervention, cardiogenic shock, valvular rupture, or pericardial tamponade (15). Maenza et al., in a meta-analysis of 43 studies, noted that an abnormal ECG correlated with the risk of developing a complication from BCI (16). In a multicenter review of 184 pediatric BCI patients published by the Pediatric Emergency Medicine Collaborative Research Committee, no hemodynamically stable patient with a normal ECG developed cardiac complications (17). Several studies have concluded that hemodynamically stable patients with normal ECGs require no additional studies (14, 18, 19). Feghali and Prisant, in a review of 35 BCI articles, concluded that ECG has a good negative predictive value (20).

There are no prototypical ECG findings that are specific for BCI. Fifty per cent of admission ECGs have nonspecific findings (9). However, when all ECG changes (including tachycardia and nonspecific changes) are included in the BCI work-up, the sensitivity of ECG for diagnosing BCI approaches 96%, with a specificity of 47% (21, 22). The most common finding is sinus tachycardia, followed by premature atrial or ventricular contractions (PAC, PVC). Other abnormalities in order of frequency include: T wave changes, atrial fibrillation/flutter, ST elevation or depression, conduction delays, ventricular dysrhythmias and new Q waves. Of all arrhythmias requiring an intervention, 80% are detected on emergency department (ED) admission (23).

Patients with normal admission ECGs have been reported to have delayed presentations of cardiac injury. These patients are typically elderly and have pre-existing cardiac disease, multiple severe chest wall injuries or unexplained hypotension (14, 15). Fildes et al., based on a prosective evaluation of 100 patients admitted to rule out myocardial injury, recommend that hemodynamically stable patients with no history of cardiac disease, who are less than 55 years old and do not require surgery or observation for other injuries can have a diagnosis of cardiac contusion excluded if there is a normal admission ECG. Patients with abnormal ECG should have at least 24 hours of cardiac monitoring and a follow-up ECG (24).

Cardiac Enzymes

The pathophysiology of trauma-induced myocardial necrosis is poorly understood. It is therefore difficult to predict the pattern of myocyte enzymatic release, and it remains unclear whether a role exists for its routine measurement. Historically, creatine kinase (CK) with myocardial type B (MB) fractions were utilized in order to assist in the detection of BCI or patients at risk for serious sequelae. Healey et al., in a retrospective evaluation of 342 BCI patients, found that a CK-MB level of >200 mg/dL had a 100% positive predictive value for cardiac complications (22). In contrast to the findings by Healey et al., a preponderance of the literature suggests that CK-MB is not predictive of complications and is not a warranted study (11). CK-MB has been isolated in skeletal muscle, lung, stomach, pancreas, liver, small intestine, and colon (25). The release of CK-MB from other organs during trauma can potentially confound the interpretation of the value. In a retrospective analysis of 133 patients, Illig et al. found that CK-MB analysis failed to predict complications secondary to BCI (26). In a retrospective review, Biffl et al. later confirmed this finding in a series of 359 patients. None of the BCI patients who went on to have complications had cardiac enzyme abnormalities (15).

The advent of the cardiac-specific troponin I poses an opportunity to selectively screen patients with BCI. Troponin I is found only in the cardiac myocyte and interacts with the actin-myosin complex. In a recent study, Relos et al. concluded that even moderate elevations in serum troponin I were indicative of myocardial injury and predictive of a 4-fold mortality increase in surgical patients (27). Troponin I in BCI patients has a sensitivity of 23–100% and a specificity of 85–97% (28–30). In a small, prospective pediatric series, 4 patients were diagnosed with BCI on the basis of history combined with abnormal echocardiographic findings and ECG abnormalities: 3 of these 4 patients had elevated troponin I levels of > 2.0 ng/mL (31). In a prospective review of 333 blunt thoracic patients, Velmahos et al. identified 13% with significant BCI, defined as hypotension in the absence of bleeding or neurogenic cause, cardiac arrythmias, post-traumatic echocardiographic abnormality, or cardiac index <2.5. By combining the ECG and troponin I at 8 hours, a negative predictive value of 100% was rendered, eliminating patients without significant BCI. The authors concluded that in the absence of other injuries or hemodynamic instability, patients with normal ECG and troponin I can be discharged (32).

Echocardiogram

Echocardiography plays a significant role in the work-up of the BCI patient. It is the primary exam in the structural assessment of the heart. Up to 30% of BCI patients have abnormal echocardiographic findings (33). Detection of pericardial effusion, myocardial contusion, valvular disruption and wall motion abnormalities may assist in defining injury severity and potentially alters management. Non-cardiac lesions such as pleural effusion, intracardiac thrombi, and aortic disruptions may also be identified by this means.

In the past decade, the bedside Focused Assessment with Sonography for Trauma (FAST) exam has increasingly become standard in US trauma centers (34). This noninvasive bedside sonographic exam can be performed accurately by trained emergency physicians and surgeons (12, 34). Four sonographic windows placed in the subcostal, right upper quadrant, left upper quadrant, and suprapubic areas detect the presence of significant amounts of free fluid. The speed of the exam was validated in a study by Sisley et al., who demonstrated that it could be completed in an average of 1.3 minutes (35). The pericardial window allows ample visualization of the pericardium and heart, to rule out pericardial effusion and tamponade. The use of the parasternal long axis, apical four chamber, and subcostal windows (used in the FAST) provides multidimensional views of the pericardial sac. Mandavia et al. concluded that, using these views, emergency physicians could reliably examine the pericardium with an accuracy of 97.5% (12). Rozycki et al. conducted a study of 247 penetrating chest injury patients, demonstrating a 100% sensitivity and specificity for hemopericardium with the use of FAST (36). In a separate multicenter study, Rozycki et al. utilized FAST to minimize ED to operating room (OR) times to an average of 12.1 minutes (37).

Formal echocardiography has proven to have great diagnostic utility for the unstable or complicated BCI patient (23, 38). Applications such as M-Mode and Doppler provide additional information not recognized on the limited FAST exam. Abnormalities such as wall motion defects and valvular incompetence are readily recognized. Echocardiography, however, lacks utility for the stable BCI patient. Karalis et al., in a prospective analysis of 105 patients, concluded that routine echocardiograms were of no value, for them, since there were few cardiac complications secondary to BCI (39). Nagy et al., in a prospective analysis of 315 patients, ascertained that the echocardiogram had no added diagnostic utility for patients with normal ECG and blood pressure on admission (19). In a meta-analysis of 18 studies of patients suspected of myocardial contusion, Christensen and Sutton concluded that there is no evidence to support the use of routine echocardiography. However, echocardiography maintains its value in detecting multiple structural abnormalities, e.g., apical thrombi, pericardial effusions, and pump failure in patients deemed to be at increased risk or suffering from a complicated in-patient course (23). In a prospective study of 118 blunt trauma patients, Lindstaedt et al. identified 14 patients (11.8%) with myocardial contusion diagnosed by an abnormal echocardiogram, defined as new akinetic or dyskinetic regional wall abnormalities. Other than a mural thrombus found on a 12-month follow-up, Lindstaedt found that none of these patients developed cardiac complications over the course of 1 year (40). Bromberg et al. similarly demonstrated, in a series of 8 children with BCI, that echocardiography was only valuable for patients who developed cardiac complications (41). Thus, echocardiography should be reserved as a complementary rather than primary screening tool for BCI. The Eastern Association for the Surgery of Trauma recommends selective use of echocardiography for patients who are hemodynamically unstable or suffer cardiac complications (11, 42, 43).

The use of transthoracic echocardiography (TTE), however valuable, may prove challenging to perform in the presence of severe chest wall injuries, pleural tubes, mechanical ventilation, and large body habitus. Karalis et al. found in his study of 105 blunt chest trauma patients, that TTE was useful for 80% of patients, yet 19% had suboptimal studies (39). Alternatively, the transesophageal echocardiogram (TEE) may be utilized. TEE, in contrast to TTE, is invasive and requires sedation and possible intubation. However, it has the added benefit of increased resolution and ability to detect thoracic aortic injuries. Chirillo et al. found that although 98% of blunt chest trauma patients successfully underwent TEE, TTE was feasible in only 38% (44). Moreover, both Chirillo and Weiss, in their respective series, concluded that TEE accurately identified evidence of contusion and valvular injuries that were missed by TTE (44, 45).

Pericardiocentesis

Pericardiocentesis, as a diagnostic modality, has virtually been supplanted by the use of ultrasound. However, lack of equipment or radiology personnel may render pericardiocentesis the exam of preference to rule out suspected tamponade. Blind insertion of the needle into the pericardial sac has been associated with misleading results 25% of the time. False positives result from the aspiration of blood directly from the cardiac chamber. False negatives result from the immediate clotting of aspirated blood from a rapidly bleeding laceration (46). ECG or sonographic guidance using the subxiphoid approach is recommended (47).

The patient in cardiac tamponade benefits from emergency pericardiocentesis. Inserting a drainage catheter may delay reaccumulation of blood in the pericardial sac, although it may clot. The procedure temporizes the condition until surgical intervention can be arranged (47).

Emergent Thoracotomy

Prompt emergency department thoracotomy in the cardiac-arrest blunt trauma patient has been documented to carry a 0 – 16% survival rate. Associated mitigating factors in survival include the identification of a cardiac injury or the presence of pupil reactivity, voluntary respiratory efforts, or purposeful movement upon ED admission (48, 49). Blunt trauma patients who arrest in the prehospital setting are not candidates for ED thoracotomy. In the event that pericardiocentesis is unsuccessful, the patient's vital signs deteriorate, or cardiac arrest ensues in the ED, thoracotomy and pericardiotomy are indicated (50). A left lateral incision is used to access the heart and clamp the descending aorta. If necessary, the lateral incision may be extended across the midline and sternum, creating a "clam shell" incision. This allows ample access to the right heart. Pericardiotomy should be performed with a parallel incision to the phrenic nerve. Once the clot has been removed, the site of injury should be identified. Atrial hemorrhage is occluded using digital pressure, a clamp, or an inflated foley catheter. Ventricular hemorrhage may be controlled similarly, with digital pressure or standard skin staples. Bimanual cardiac compressions should ensue immediately once the source of injury is stabilized (47).

Pulmonary Artery Catheter

The use of the pulmonary artery catheter, though invasive, has distinct value in the evaluation of the critical BCI patient. Evidence of hemodynamic instability, age greater than 60, abnormal ECG, and future surgical intervention under general anesthesia have been described as rationales for its insertion (21). Unlike the echocardiogram, the catheter provides continuous, precise information regarding preload, afterload, and ventricular function. These parameters may serve an essential role during the resuscitation of the multisystem-involved volume-depleted patient with heart failure or pulmonary hypertension (21).

Blunt Cardiac Injury with Minor ECG or Enzyme Abnormality

Patients with isolated ECG or enzyme abnormality represent the largest and most benign subset of BCI patients. However, the existence of ECG and/or cardiac enzyme abnormalities in BCI has been correlated with clinically significant outcomes. In a retrospective meta-analysis of 2,210 BCI patients, Maenza et al. found that the existence of a positive ECG or a cardiac enzyme abnormality correlated with an increased risk of an operative or cardiac intervention (16). In another study, Velmahos et al. evaluated 333 admitted blunt thoracic trauma patients with serial troponin and ECG at admission, 4 hours, and 8 hours. They reported that 34% of patients diagnosed with ECG *and* enzyme abnormalities had hypotension, cardiac arrhythmias, echocardiographic abnormalities, or low cardiac index <2.5. Moreover, no patient with a normal ECG and negative enzyme abnormality developed BCI (32).

Despite potential acute complications, abnormalities documented upon admission typically resolve within hours, and the patient rarely suffers long-term functional sequelae (51, 52). BCI with minor ECG and/or enzyme abnormality should receive telemetry monitoring and may require further diagnostic testing, including a formal echocardiogram. Intensive care unit (ICU) admission should be reserved for patients with associated hemodynamic instability (53).

Blunt Cardiac Injury with Complex Arrhythmia

Arrhythmias represent the most common complication associated with BCI (16). Up to 70% of patients with BCI will suffer from a rhythm disturbance. The most common arrhythmias are atrial, followed by ventricular, and conduction delays. Atrial or ventricular dysrhythmias requiring an intervention have been documented to occur in up to 30% of patients (9). However, Maenza et al. found, in their meta-analysis, that only 2.6% of BCI patients developed complex cardiac arrythmias requiring an intervention (16). Treatment options include antiarrhythmics and/or electrical cardioversion (1, 9).

Myocardial concussion, commotion or commotio cordis, originally described in 1879, is caused by a direct low impact blow to the chest resulting in an isolated dysrhythmia, syncope, or sudden death (54, 55) . It is characterized as a sudden disturbance of cardiac rhythm in the absence of significant structural damage to the myocardium (54). A majority of patients sustaining such injuries are children who are injured during sporting activities or “play fighting” (55). The electrophysiologic outcome of the injury is dependent upon the timing of impact during the cardiac cycle. Link et al. developed an experimental model that produced ventricular fibrillation when chest wall impacts occurred during a 15 and 30 ms interval preceding the T wave. Impacts during ventricular depolarization were associated with a brief period of heart block. Left bundle branch blocks and ST-T segment changes were also described to correlate with impact timing (56).

Ventricular fibrillation is thought to be the etiology of sudden death in myocardial concussion patients. In one retrospective analysis of 128 confirmed cases of commotio cordis, there was an 84% mortality rate. Selection bias is inherent in this study, since cases were obtained from a national registry of voluntarily submitted cases, a majority involving the death of a minor (55). The implementation of prompt CPR and pre-hospital automatic external defibrillator (AED) use has been shown to increase the survival rate (57, 58).

Blunt Cardiac Injury with Cardiac Failure

Patients with cardiac failure represent approximately 2 – 20% of patients with identified BCI (9). Contusions cause cellular necrosis and inflammatory changes, which alter ventricular compliance and decrease coronary perfusion (2). Valvular incompetence, pump failure, complex arrhythmias and coronary artery occlusion also compromise cardiac output, thereby causing hypotension (9). The clinical findings that may alert the clinician include hypotension, tachycardia, tachypnea, jugular venous distension, rales, and/or a systolic murmur. Critical care monitoring may reveal an elevated central venous pressure, an abnormal pulmonary artery wedge pressure and low cardiac index (21). Pericardial tamponade may present with similar findings and must be considered in the evaluation of these patients.

The use of echocardiography and pulmonary catheterization may prove instrumental in identifying the source of hemodynamic perturbations and the chamber involved (21).

ICU admission and symptomatic management with intubation, crystalloid, vasopressors, and inotropes are management options. Patients in acute failure associated with hypoxemia should be promptly intubated. This should be followed by crystalloid boluses of 20 cc/kg to correct blood pressure and cardiac index (CI) if the central venous pressure is less than 10. Inotropes such as dobutamine or milrinone should be administered to maintain a CI > 2.2 (59). If blood pressure remains low, vasopressors should be added to elevate to a systolic blood pressure (SBP) of 90. Avoid diuretics, since this may decrease intravascular volume needed to maintain the cardiac output.

Refractory cardiogenic failure has been successfully managed with intra-aortic balloon counter pulsation (IABP), according to case reports. The device decreases left ventricular afterload and increases coronary blood flow, thereby improving left ventricular function. This enables stunned myocardium to return to reasonable parameters in a period of several days to several weeks. IABP is contraindicated in aortic insufficiency (58, 60).

Key Concepts

- All patients with thoracic trauma must have an ECG
- Maintain a high level of suspicion of BCI for patients with chest pain, rib or sternal injuries, pulmonary lesions, or deceleration injury
- Rule out all hypotensive BCI patients for pericardial tamponade by echocardiography
- The risk of significant BCI is insignificant in stable patients with normal ECG and troponin
- Suspect BCI in the multisystem-injured patient with refractory hypotension

ECG = electrocardiogram; BCI = blunt cardiac injury.

Blunt Cardiac Injury with Wall Rupture

Abrupt compressive force to the torso can generate markedly elevated transthoracic and venous pressures. These pressures are transmitted from the neighboring thoracic and abdominal compartments to cardiac chambers and closed valves. The heart chambers are most susceptible to injury when at maximal filling. Although the right heart is most often involved, both ventricles may be ruptured. Ruptures can be either immediate or delayed (6). Paradoxical penetrating lesions with a fractured rib or sternum have been implicated in right atrial and ventricular ruptures (6, 8).

Chamber wall rupture carries the highest mortality rate of the BCI categories. In clinical trials the incidence of chamber rupture is 0–30%, compared to a 64% incidence in autopsy studies (9). More than one chamber will be involved in 20% of the cases (61). Clinical characteristics and outcome depend on the chamber involved and the integrity of the pericardium. In two series of patients with traumatic cardiac rupture, Mattox et al., and Shorr et al., reported 85% and 100% mortality rates, respectively (62, 63). Although, a majority of these patients expire in the prehospital setting, up to 7% of patients have been documented to survive longer than 30 minutes (2). This finding supports the importance of trauma systems with rapid transport capabilities. Rupture of a low-pressure chamber, delayed rupture of a high-pressure chamber, or small, contained pericardial tears may allow the patient to survive transport to the hospital (64). Survival is most common with right atrial rupture (65).

Approximately 70% of patients with cardiac wall rupture develop pericardial effusion and tamponade (46). Extracardiac hemorrhage in excess of 200 cc may render these conditions fatal. When the accumulation of blood exceeds the compliance of the pericardium, intrapericardial pressure rapidly increases, causing collapse of the ventricle, restricting its filling. Getz et al. describe signs of distended neck veins, persistent hypotension, hemothorax, or metabolic acidosis unresponsive to fluid resuscitation as commonly associated with myocardial rupture (46). However, coexistent injuries to other organ systems may mask classic clinical presentations of tamponade because of massive blood loss or neurogenic shock.

The finding of pulsus paradoxus on examination or diastolic ventricular collapse in the setting of pericardial effusion on echocardiogram is pathognomic for tamponade physiology. ECG may demonstrate electrical alternans, in which the QRS complex alternates voltage from beat to beat. Pulmonary artery catheter placement will demonstrate equalization of right and left ventricular pressures (10). Smaller volumes of pericardial fluid and chronic inflammatory effusions may be asymptomatic and initially undetectable. These can collect and may be visualized on subsequent echocardiographic studies (7).

Once the diagnosis is made, emergent pericardiocentesis and/or transport to the OR for a pericardial window must be expedited, since the natural course of this disease is rapidly progressive and deadly. In the event of cardiac arrest or hemodynamic decline refractory to pericardiocentesis, emergent thoracotomy and pericardiotomy is indicated (48, 49).

Septal ruptures may present with cardiac failure with left to right shunt (66). The cardiac valves are at greatest risk for rupture during periods of closure in the cardiac cycle. The most common valves involved are the aortic and mitral valves. The aortic valve is under greatest tension during early diastole, when the left ventricle is empty. The mitral valve is most vulnerable during early systole in isovolumetric contraction. Valvular rupture occurs in up to 6% of BCIs (67). Disruption causes regurgitation and cardiogenic failure that is evident immediately or within weeks. In contrast, insufficiency secondary to a tricuspid or pulmonary rupture is typically asymptomatic for several years (68). Coordination of trauma and cardiothoracic specialist care should not be delayed in cases of BCI with valvular, free wall, or septal rupture.

Pearls and Pitfalls in BCI

Case 1

A 68-year-old driver arrives at a community ED at 2 AM after a minor automobile accident, complaining only of chest pain. His ECG is consistent with an acute MI. Thrombolytics are administered and the patient becomes terminal within minutes.

This patient experienced a massive hemothorax. A thorough exam would have revealed chest wall contusions from the steering wheel, consistent with BCI and thoracic aortic injuries. Recent head injury and severe trauma are contraindications to thrombolytics. Remote incidents or mild musculoskeletal trauma are relative contraindications.

Case 2

A 19-year-old woman is admitted to a university hospital for a low-grade splenic laceration and severe cervical strain sustained upon being struck by a vehicle. Though hypotensive in the ED, the patient has an uneventful in-patient course and is discharged 3 days later in a Miami J collar with the following vital signs: blood pressure (BP) 100/72, heart rate (HR) 114, respiratory rate (RR) 16, and oxygen saturation (O_2 Sat) 98%. Twenty-four hours later, she presents to the ED complaining of severe mid-epigastric pain. Vital signs are BP 80/40, HR 130, RR 20, O_2 Sat 96%, and Temp 98.3. One hour later an abdominal CT is normal and the patient's vital signs remain unchanged despite 2 liters of fluid and a stable hematocrit. A subsequent bedside FAST reveals a pericardial effusion.

This patient presents with delayed hemopericardium and tamponade from a small tear in the right atrium. Initially, hypotension in the setting of a minor splenic laceration should have alerted the clinicians to a missed injury. An unexplained abnormal ECG (tachycardia) on discharge warrants an echocardiogram in this setting. Jugular vein distention, a classic finding, may be a late or nonexistent finding in the BCI patient with multi-organ or multi-extremity injuries. Beware of the concealing cervical collar.

Case 3

A 55-year-old trauma patient with multiple extremity fractures is to be discharged at change of shift. The patient had required intubation for agitation and had received 5 liters of fluid for hypotension. On reassessment, you notice that the patient's O_2 Sat is 95% on 100% fraction of inspired oxygen (FiO_2) and there are diffuse rales. An X-ray is obtained, and the radiologist confirms that the patient has pulmonary edema. Furosemide is administered. The patient becomes hypotensive and arrests.

Pulmonary edema is a common presentation in patients with papillary muscle rupture, aortic rupture, and ventricular contusion. Valvular incompetence should be evaluated emergently by a cardiothoracic surgeon for repair. Treatment for ventricular failure includes fluids, pressors and inotropes, preferably guided by the use of a pulmonary artery catheter. Consider an intra-aortic balloon pump for refractory cardiogenic failure.

Case 4

A patient presenting from a 30-foot fall loses his vital signs upon arrival at the ER. Emergent thoracotomy and pericardiotomy is indicated when the patient develops rapid cardiovascular decline and cardiac arrest in the ED.

A pericardiocentesis is performed blindly. Blood is aspirated; it clots immediately. The exam is declared negative; there is no hemopericardium. Thirty minutes later the patient arrests. Thoracotomy reveals a large pericardial effusion.

Blind, inadvertent aspiration from the intracardiac chamber may lead to a false negative diagnosis. Lacerated cardiac vessels or ruptured chambers can bleed quickly into the pericardial sac and will clot. Sonographic or electrocardiographic guidance may assist in averting technical miscalculations.

Case 5

A 14-year-old boy presents after losing consciousness at home while shadow boxing with his 6-year-old brother. He only complains that his chest "feels weird." This patient's presentation is consistent with commotio cordis. Electrocardiographic changes including ventricular fibrillation, heart block, and ST changes have been described. The patient should receive an ECG and appropriate electrical cardioversion or antiarrhythmic therapy, if indicated.

Blunt Cardiac Injury with Coronary Artery Thrombosis

Coronary artery lesions represent less than 2% of BCIs. Direct precordial impact, crushing between sternum and spine, and resultant myocardial edema are the proposed causative mechanisms. Coronary artery contusion, spasm, disruption of coronary plaque, laceration, dissection, and occlusion have all been described in the literature. Anginal-type chest pain, myocardial ischemia and infarction are common outcomes. The left anterior descending is more commonly injured than the right coronary artery (69, 70).

The ECG often demonstrates ST segment elevation or changes consistent with an infarction or ischemia (71). Such findings warrant an emergent echocardiogram and/or angiography. Prompt consultation with a cardiologist and/or cardiothoracic surgeon is also essential. Management options should not differ from those of the typical myocardial infarction patient. Conservative therapy, percutaneous coronary angioplasty, and thrombolytics are appropriate interventions. The option instituted should be weighed against concomitant injuries, hemodynamic status, and possible contraindications to anticoagulation or thrombolytic use (71). On long-term follow-up, sequelae range from complete recovery to cardiac complications of angina, congestive heart failure, and death (72).

Conclusion

Since there are no standard diagnostic criteria for BCI, the true incidence of blunt cardiac injury remains unknown. Signs and symptoms suspicious for cardiac injury warrant expeditious workup to exclude lethal injuries. ECG remains a sensitive test for BCI; however, all changes and dysrhythmias must be presumed to be suspicious in order to avoid missed injuries. The addition of troponin I is an option that may serve to identify those patients at increased risk of mortality, especially if the ECG is abnormal. Although it appears that troponin I is more predictive than CK-MB of an adverse outcome, the physiologic nature and timing of troponin release remains unclear. The utility of troponin I in predicting long-term BCI morbidity is also undetermined. Patients with ECG abnormality and troponin elevation or arrhythmias are clear candidates for telemetry monitoring. Echocardiography should be reserved for patients who have a complicated inpatient course or undifferentiated clinical scenarios that warrant investigation, i.e., persistent chest pain or shortness of breath. If the patient exhibits signs of hemodynamic instability, an echocardiogram is indicated to identify life-threatening injuries. Placement of a pulmonary catheter may further assist in the hemodynamic management of the unstable patient. The infrequent coronary artery thrombosis should be managed expeditiously and its management weighed against that of other injuries and co-morbidities. Future studies identifying the independent risk factors of critically ill BCI patients remain needed.

References

1. Vital and Health Statistics: 2001. US Department of Health and Human Services 2004.
2. Parmley LF, Manion WC, Mattingly TW. Nonpenetrating traumatic injury of the heart. Circulation 1958; 18:371 – 396.
3. Mattox KL, Flint LM, Carrico CJ, et al. Blunt cardiac injury. J Trauma 1992; 33(5):649 – 650.
4. National Hospital Discharge Survey: 2001. Annual summary with detailed diagnosis and procedure data. US Department of Health and Human Services: June 2004.
5. Tenzer ML. The spectrum of myocardial contusion—a review. J Trauma 1985; 25:620 – 627.
6. Fulda G, Brathwaite CE, Rodriguez A, et al. Blunt traumatic rupture of the heart and pericardium: a ten-year experience (1979 – 1989). J Trauma 1991: 31(2):167 – 173.
7. Pretre R, Chilcott M. Blunt trauma to the heart and great vessels. N Eng J Med 1997; 336(9):626 – 632.
8. Patetsios P, Priovolos S, Slesinger TL, et al. Lacerations of the left ventricle from rib fractures after blunt trauma. J Trauma 2000; 49(4):771 – 713.
9. Shultz JM, Trunkey DD. Blunt cardiac injury. Crit Care Clin 2004; 20(1):57 – 70.
10. Spodick DH. Acute cardiac tamponade. N Eng J Med 2003; 349:684 – 690.
11. Pasquale MD, Nagy K, Clarke J. Practice Management Guidelines for the Screening of Blunt Cardiac Injury; Eastern Association for the Surgery of Trauma: 1998. Available on URL www.east.org/tpg/chap2.pdf [Accessed 7/28/05]
12. Mandavia DP, Hoffner RJ, Mahaney K, et al. Bedside echocardiography by emergency physicians. Ann Emer Med 2001; 38(4):377 – 382.
13. Manyari DE, Milliken JA, Colwell BT, et al. Detection of pericardial effusion by chest roetgenography and electrocardiography versus echocardiography. Can Med Assoc J 1978; 119(5):445 – 450.
14. Foil MB, Mackersie RC, Furst SR, et al. The asymptomatic patient with suspected myocardial contusion. Am J Surg 1990; 160:638 – 643.
15. Biffl WL, Moore FA, Moore EE, et al. Cardiac enzymes are irrelevant in the patient with suspected myocardial contusion. Am J Surg 1994; 168:523 – 528.
16. Maenza RL, Seaberg D, D'Amico F. A meta-analysis of blunt cardiac trauma: ending myocardial confusion. Am J Emerg Med 1996; 14(3):237 – 221.
17. Dowd MD, Krug S. Pediatric blunt cardiac injury: epidemiology, clinical features and diagnosis. Pediatric Emergency Medicine Collaborative Research Committee: working group on blunt cardiac injury. J Trauma 1996; 40:61 – 67.
18. Dubrow TJ, Mihalka J, Eisenhauer DM, et al. Myocardial contusion in the stable patient: what level of care is appropriate? Surgery 1989; 106:267 – 274.
19. Nagy KK, Krosner SM, Roberts RR, et al. Determining which patients require evaluation for blunt cardiac injury following blunt chest trauma. World J Surg 2001; 25(1):108 – 111.

20. Feghali NT, Prisant LM. Blunt myocardial injury. Chest 1995; 108(6):1673 – 1677.
21. Orliaguet G, Ferjani M, Riou B. The heart in blunt trauma. Anesthesiology 2001; 95(2):544 – 548.
22. Healey MA, Brown R, Fleiszer D. Blunt cardiac injury: is this diagnosis necessary? J Trauma 1990; 30(2):137 – 146.
23. Christensen MA, Sutton KR. Myocardial contusion: new concepts in diagnosis and management. Am J Crit Care 1993; 2:28 – 34.
24. Fildes JJ, Betlej TM, Manglano R, et al. Limiting cardiac evaluation in patients with suspected myocardial contusion. Am Surg 1995; 61(9):832 – 835.
25. Tsung SH. Creatine kinase and isoenzyme patterns in human tissue obtained at surgery. Clin Chem 1976; 22:173 – 175.
26. Illig KA, Swierzewski MJ, Feliciano DV, et al. A rational screening and treatment strategy based on the electrocardiogram alone for suspected cardiac contusion. Am J Surg 1991; 162:537 – 544.
27. Relos R, Hasinoff I, Beilman G. Moderately elevated serum troponin concentrations are associated with increased morbidity and mortality rates in surgical intensive care unit patients. Crit Car Med 2003; 31(11):2598 – 2603.
28. Salim A, Velmahos GC, Jindal A, et al. Clinically significant blunt cardiac trauma: role of serum troponin levels combined with electrocardiographic findings. J Trauma 2001; 50(2):237 – 243.
29. Bertinchant JP, Polge A, Mohty D, et al. Evaluation of incidence, clinical significance, and prognostic value of circulating cardiac troponin I and T elevation in hemodynamically stable patients with suspected myocardial contusion after blunt chest trauma. J Trauma 2000; 48(5):924 – 931.
30. Adams III JE, Davila-Roman VG, Bessey PQ, et al. Improved detection of cardiac contusion with cardiac troponin I. Am Heart J 1996; 131(2):308 – 312.
31. Hirsch R, Landt Y, Porter S, et al. Cardiac troponin I in pediatrics: normal values and potential use in the assessment of cardiac injury. J Ped 1997; 6:872 – 877.
32. Velmahos G, Karaiskakis M, Salim A, et al. Normal electrocardiography and serum troponin I levels preclude the presence of clinically significant blunt cardiac injury. J Trauma 2003; 54:45 – 51.
33. Frazee RC, Mucha P Jr, Farnell MB, et al. Objective evaluation of blunt cardiac trauma. J Trauma 1986; 26:510 – 520.
34. Patel AN, Brennig C, Cotner J, et al. Successful diagnosis of penetrating cardiac injury using surgeon-performed sonography. Ann Thorac Surg 2003; 76(6):2043 – 2047.
35. Sisley AC, Rozycki GS, Ballard RB, et al. Rapid detection of traumatic effusion using surgeon performed ultrasonography. J Trauma 1998; 44(2):291 – 296.
36. Rozycki GS, Feliciano DV, Schmidt JA, et al. The role of surgeon performed ultrasound in patients with possible cardiac wounds. Ann Surg 1996; 223(6):737 – 744.
37. Rozycki GS, Feliciano DV, Oschner MG, et al. The role of ultrasound in patients with possible penetrating cardiac wounds: a prospective multicenter study. J Trauma 1999; 46(4):543 – 551.
38. Cachecho R, Grindlinger GA, Lee VW. The clinical significance of myocardial contusion. J Trauma 1992; 33:68 – 73.
39. Karalis DG, Victor MF, Davis GA, et al. The role of echocardiography in blunt chest trauma: a transthoracic and transesophageal echocardiographic study. J Trauma 1994; 36:53 – 58.
40. Lindstaedt M, Germing A, Lawo T, et al. Acute and long-term clinical significance of myocardial contusion following blunt thoracic trauma: results of a prospective study. J Trauma 2002; 52(3):479 – 485.
41. Bromberg BI, Mazziotti MV, Canter CE, et al. Recognition and management of nonpenetrating cardiac trauma in children. J Pediatr 1996; 128:536 – 541.
42. Cachecho R, Grindlinger GA, Lee VW. The clinical significance of myocardial contusion. J Trauma 1992; 33:68 – 73.
43. Malangoni MA, McHenry CR, Jacobs DG. Outcome of serious blunt cardiac injury. Surgery 1994; 116:628 – 633.
44. Chirillo F, Totis O, Cavazerani A, et al. Usefulness of transthoracic and transesophageal echocardiography in recognition and management of cardiovascular injuries after blunt chest trauma. Heart 1996; 75(3):301 – 306.
45. Weiss RL, Brier JA, O'Connor W, et al. The usefulness of transesophageal echocardiography in diagnosing cardiac contusions. Chest 1996; 109:73 – 77.
46. Getz BS, Davies E, Steinberg SM. Blunt cardiac trauma resulting in right atrial rupture. JAMA 1986; 255:761 – 763.
47. Roberts clinical procedures in emergency medicine: concepts and clinical practice. In: Roberts JR, editor. 4th ed. New York: Elsevier; 2004.
48. Mazzorana V, Smith RS, Morabito DJ, et al. Limited utility of emergency department thoracotomy. Am Surg 1994; 60:516 – 521.
49. Bodai BI, Smith JP, Blaisdell FW. The role of emergency thoracotomy in blunt trauma. J Trauma 1982; 22:487 – 491.
50. Symbas PN. Traumatic heart disease. Curr Prob Card 1991; 16:537 – 582.
51. Lindstaedt M, Germing A, Lawo T, et al. Acute and long-term clinical significance of myocardial contusion following blunt thoracic trauma: results of a prospective study. J Trauma 2002; 52(3):479 – 485.
52. Sturaitis M, McCallum D, Sutherland G, et al. Lack of significant long-term sequelae following traumatic myocardial contusion. Arch Intern Med 1986; 146(9):1765 – 1769.
53. Brunel W, Stoll J, May K, et al. Routine intensive care unit admission is not indicated for suspected myocardial contusion. J Int Care Med 1988; 3:253 – 257.
54. Nesbitt AD, Cooper PJ, Kohl P. Rediscovering commotio cordis. Lancet 2001; 357:1195 – 1197.
55. Maron HJ, Gohman TE, Kyle SB, et al. Clinical profile and spectrum of commotio cordis. JAMA 2002; 287:1142 – 1146.
56. Link MS, Wang PJ, Pandian NG, et al. An experimental model of sudden death due to low energy chest wall impact. N Eng J of Med 1998; 338:1805 – 1811.
57. Zangwill SD, Strasburger JF. Commotio cordis. Pediatr Clin North Am 2004; 51(5):1347 – 1354.
58. Penney DJ, Bannon PG, Parr MJA. Intra-aortic balloon counterpulsation for cardiogenic shock due to cardiac contusion in an elderly patient. Resuscitation 2002; 55:(3)337 – 340.
59. Keung EC, Siskind SJ, Sonneblick EH, et al. Dobutamine therapy in acute myocardial infarction. JAMA 1981; 245(2):144 – 146.
60. Snow N, Lucas AE, Richardson DJ. Intra-aortic balloon counterpulsation for cardiogenic shock from cardiac contusion. J Trauma 1982; 22(5)426 – 429.
61. Kato K, Kushimoto S, Mashiko K, et al. Blunt traumatic rupture of the heart: an experience in Tokyo. J Trauma 1994; 36(6): 859 – 864.
62. Mattox KL, Koch L, Beal AC, et al. Logistic and technical considerations in the treatment of the wounded heart. Circulation 1975; 52(2 Suppl):I210 –I214.
63. Shorr RM, Crittenden M, Indeck M, et al. Blunt thoracic trauma. Analysis of 515 patients. Ann Surg 1987; 206(2):200 – 205.
64. Pevec WC, Udekwu AO, Peitzman AB. Blunt rupture of the myocardium. Ann Thorac Surg 1989; 48:139 – 143.

65. Baumgartel ED. Cardiac rupture form blunt trauma with atrial septal defect. Arch Surg 1992; 127:347 – 348.
66. Pevec WC, el-Hillel M, McArdle DQ, et al. Rupture of the left ventricle and intraventricular septum by blunt trauma. Crit Care Med 1989; 17(8):837 – 838.
67. Pretre R, Faudutti B. Surgical management of aortic valve injury after non-penetrating trauma. Ann Thorac Surg 1993; 56:1426 – 1431.
68. Caparelli DJ, Cattaneo II SM, Brock MV, Conte Jr JV. Aortic and mitral valve disruption following nonpenetrating chest trauma. J Trauma; 2002; 52(2):377 – 379.
69. Nieman J, Hui WKK. Posteromedial papillary muscle rupture as a result of right coronary artery occlusion after blunt chest injury. Am Heart J 1992; 123:1694 – 1699.
70. Doty D, Anderson A, Rose E, et al. Cardiac trauma. Ann Surg 1974; 180:452 – 460.
71. Bokelman TA, Rahko PS, Meany BT, Fausch MD. Traumatic occlusion of the right coronary artery resulting in cardiogenic shock successfully treated with primary angioplasty. Am Heart J 1996; 131(2):411 – 413.
72. Vlay SC, Blumenthal DS, Shoback D, et al. Delayed acute myocardial infarction after blunt chest trauma in a young woman. Am Heart J 1980; 100:907 – 916.