Blunt Cardiac Trauma: A Review of the Current Knowledge and Management

Raid Yousef, MD, and John Alfred Carr, MD
Department of Trauma Surgery, Hurley Medical and Level 1 Trauma Center, Flint, Michigan

Blunt cardiac injuries are highly lethal. A review of the world’s English literature on the topic reveals a lack of Level 1 Evidence and few cohesive guidelines for the management of these patients. An online database query was performed using the PubMed medical database.

In 2011 the National Trauma Data Bank reported that 773,292 people in the United States required admission to a hospital for traumatic injuries [1]. This information was collected from their 744 participating hospitals. The data show that cardiac trauma (blunt and penetrating) is identified in less than 10% of all trauma admissions, and yet cardiac trauma is associated with a much higher mortality than other organ system injuries. Considering the lethality of this type of injury, better guidelines should exist to direct management.

This review will only focus on blunt cardiac injury (BCI) and will not address penetrating injuries or blunt trauma to the aorta. The world’s English literature on the topic of BCI demonstrates a significant lack of Level 1 Evidence and few cohesive guidelines in the management. Therefore, this article will provide an exhaustive assessment of the data on the topic of BCI and provide conclusions based on the known Levels of Evidence.

Pathophysiology

Most BCIs occur due to motor vehicle crashes (approximately 50%), followed by pedestrians being struck by motor vehicles (35%), motorcycle crashes (9%), and the remainder are mostly secondary to falls from a significant height [2, 3]. One autopsy study of more than 1,597 fatalities from blunt trauma identified cardiac injuries in 190 individuals (11.9%) [3]. Approximately 70% to 80% of those patients who sustained significant BCI had multiple other injuries: brain (42% to 54%), thoracic aorta (47% to 49%), lung (44% to 46%), hemothorax (37% to 89%), rib or sternal fractures (26% to 97%), and spinal injuries (37%) [2-7]. Patients with severe BCI who have other associated organ injuries often die at the scene of the accident [2-7]. Cardiac injuries are the only cause of death or contribute to the fatal outcome in 45% to 76% of the individuals who die [3, 6].

The most common injury is transmural rupture of a cardiac chamber, seen in 39% to 64%, or multiple chamber ruptures in 26% [3, 7].

In contrast to motor vehicle crashes, up to 54% of people who fall from a height exceeding 6 meters (20 feet) have some type of BCI, from small endocardial tears to transmural rupture [8]. The chance of cardiac injury increases as the height increases, such that the surgeon should be concerned about some type of possible endocardial tear or intramural hematoma in any patient who falls more than 6 meters (20 feet) [8]. For those with documented cardiac injury by autopsy after death from a fall, sternal fractures were present in 76% and multiple sternal fractures in 16% [8]. Thus, the combination of a fall greater than 20 feet with a sternal fracture should prompt a thorough cardiac evaluation.

Death at the Scene

When death from blunt trauma is proven by autopsy to be from cardiac injury, the most common lethal cardiac injuries were transmural rupture of 1 or more cardiac chambers (64%), tears occurring at the venous-atrial confluence (33%), or a blunt coronary artery dissection or tear [2, 3, 5-8]. Owing to its anterior location, the right ventricle is the most commonly injured chamber in 40% of the victims [3, 6-8] (Table 1). The right atrium and left ventricle follow closely second and third, with injuries in 30% to 33% [7-9].

Tears occurring at the venous-atrial confluence are believed to be due to rapid deceleration that results in the freely mobile ventricles (which also have most of the mass of the heart) continuing to move forward or laterally while the tethered posterior veins do not move, resulting in a distraction-avulsion injury at the venous-atrial...
Table 1. Autopsy Findings in 303 Blunt Cardiac Injuries

<table>
<thead>
<tr>
<th>Finding</th>
<th>No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pericardial tears</td>
<td>108 (36)</td>
</tr>
<tr>
<td>Transmural right atrial rupture</td>
<td>64 (21)</td>
</tr>
<tr>
<td>Right atrial hematoma</td>
<td>19 (6)</td>
</tr>
<tr>
<td>Right atrial/IVC tear with epicardial hematoma</td>
<td>18 (6)</td>
</tr>
<tr>
<td>All right atrial injuries</td>
<td>101 (33)</td>
</tr>
<tr>
<td>Transmural left atrial rupture</td>
<td>39 (13)</td>
</tr>
<tr>
<td>Left atrial hematoma</td>
<td>5 (2)</td>
</tr>
<tr>
<td>Left atrial/pulmonary vein tear with</td>
<td>3 (1)</td>
</tr>
<tr>
<td>epicardial hematoma</td>
<td></td>
</tr>
<tr>
<td>All left atrial injuries</td>
<td>47 (16)</td>
</tr>
<tr>
<td>Transmural right ventricular rupture</td>
<td>83 (27)</td>
</tr>
<tr>
<td>Right ventricular intramural hematoma</td>
<td>38 (13)</td>
</tr>
<tr>
<td>All right ventricular injuries</td>
<td>121 (40)</td>
</tr>
<tr>
<td>Transmural left ventricular rupture</td>
<td>61 (20)</td>
</tr>
<tr>
<td>Left ventricular intramural hematoma</td>
<td>35 (12)</td>
</tr>
<tr>
<td>All left ventricular injuries</td>
<td>96 (32)</td>
</tr>
<tr>
<td>Ventricular septal tear</td>
<td>12 (4)</td>
</tr>
<tr>
<td>Tricuspid valve injury</td>
<td>6 (2)</td>
</tr>
<tr>
<td>Mitral valve injury</td>
<td>5 (2)</td>
</tr>
<tr>
<td>Aortic valve injury</td>
<td>8 (3)</td>
</tr>
<tr>
<td>Pulmonary valve injury</td>
<td>1 (&lt;1)</td>
</tr>
<tr>
<td>Coronary artery dissection</td>
<td>3 (1)</td>
</tr>
<tr>
<td>Coronary artery torn</td>
<td>5 (2)</td>
</tr>
<tr>
<td>Heart completely avulsed</td>
<td>13 (4)</td>
</tr>
</tbody>
</table>

* Data compiled from references 3, 6, and 8. Many patients had more than 1 type of injury.

IVC = inferior vena cava.

Confluence. This type of injury can occur in two places: at the junction of the inferior vena cava and the right atrium and at the junction of the pulmonary veins and the left atrium [3, 8, 10, 11]. One autopsy study of 190 deaths due to BCI found almost all of the right atrial tears occurred near the area where the inferior vena cava enters the right atrium, and left atrial ruptures were located predominantly in the areas where the great veins lead into the atria [3]. Unlike chamber rupture, small tears at the confluence can be contained, and the patient may initially be stable. Diagnosis by echocardiography (ECHO) and repair with and without cardiopulmonary bypass has been reported [10, 12]. No reports have documented an isolated tear occurring at the confluence of the superior vena cava and right atrium.

Commotio Cordis

Commotio cordis is sudden death from cardiac arrest in a young person, often occurring during sports, after a blunt blow to the chest in the absence of structural cardiovascular disease [13]. Unlike motor vehicle accidents or falls, this injury shows no anatomic structural damage to the heart but is a pure conduction abnormality induced by the trauma. Commotio cordis is also a completely different phenomenon from sudden sports-related cardiac death from underlying congenital heart disease or hypertrophic cardiomyopathy. This traumatic injury occurs in the athlete with a normal heart.

The United States Commotio Cordis Registry (224 cases as of 2011) documented that 50% of the cases causing death from blunt blows to the chest occurred in competitive sports. This most often occurs in baseball and is usually triggered when players are struck in the chest by balls that have been pitched, batted, or thrown in a variety of scenarios. Other than baseball, commotio cordis is seen mainly in softball, ice hockey, football, or lacrosse [14]. Another 25% of commotio cordis events occur in recreational sports played at home, on the playground, or at family gatherings. The remaining 25% of commotio cordis happens in events unrelated to sports, such as kicks in the chest by animals or other odd events [13, 14]. Among patients in the United States registry, 65% of athletes with commotio cordis were aged between 10 and 25 years, 26% were younger than 10 years, and only 9% were older than 25 years. Interestingly, chest protection, in the form of a polymer foam pad or hard shell, appears not to be protective, because athletes wearing these still died [15, 16]. Two separate studies found that 28% and 40% of the deaths from commotio cordis occurred in athletes wearing commercially available chest protectors [13, 15].

The underlying pathophysiology of the cardiac arrest in these individuals has always been assumed to be caused by the impact transmitted to the myocardium inducing ventricular fibrillation during an electrically vulnerable phase of ventricular excitability, more specifically, cardiac repolarization [13, 16]. In a swine model with a ball propelled at 30 to 40 miles per hour striking the chest, ventricular fibrillation could only be induced 28% of the time [17]. These chest wall strikes were timed to occur during the narrow window of cardiac repolarization. Multivariate analysis revealed that the highest chance of inducing fibrillation occurred when higher left ventricular pressures were generated by the impact, with longer QRS duration, and QTc variability [17].

Unlike hypoxic or ischemic cardiac arrest, however, most of the time this population tends to be refractory to cardiopulmonary resuscitation and defibrillation, even when it is begun immediately after arrest [18, 19]. This raises the possibility that commotio cordis may occur due to a combination of ventricular fibrillation and coronary vasospasm or segmental changes in myocardial contractility [18]. Although a review of the literature from the 1990s will identify a few rare case reports of survivors, most victims died immediately at the scene [13]. For example, a published report in 1995 that documented the chronology in 25 patients showed that without cardiopulmonary resuscitation and defibrillation within 3 minutes, all died (n = 6). With cardiopulmonary resuscitation and defibrillation within 3 minutes (n = 19), only 2 were restored to a normal cardiac rhythm, but both later died from severe hypoxic brain injury [13].
The latest commotio cordis registry data show that survival has increased over time and that the number of successful resuscitations between 2006 and 2009 exceeded the number of deaths by 20%. This improvement is probably the result of increased public awareness, the increased availability of automatic external defibrillators, and earlier activation of the chain of survival, including initiation of cardiopulmonary resuscitation, defibrillation, and advanced life support measures [14].

More effort should be made to prevent these largely avoidable deaths, and this can be accomplished by providing more education, especially to coaches, better-designed athletic equipment (eg, effective chest-wall protectors), and wide access to automatic external defibrillators at organized athletic events [14, 19, 20].

Survivors to the Hospital

Most of the blunt trauma victims with BCI who do survive to the hospital tend to have injuries that are less severe. Other than cardiac tamponade from bleeding, the other potentially treatable and reversible BCIIs can be divided into two large groups: (1) structural cardiac injuries, which include intramural hematoma (IMH), valvular injury, ventricular septal rupture, coronary artery injury, myocardial infarction, and (2) electrical disturbances, such as atrial and ventricular dysrhythmias, and conduction disturbances.

Intramural Hematoma

IMH most commonly occurs in the right ventricle (13% of all patients with BCI), followed closely by the left ventricle (12%) [3, 6, 8]. Although diagnosis of IMH can be made by computed tomography or magnetic resonance imaging, the method of choice for noninvasive diagnosis is by ECHO [21–24]. IMH can cause premature ventricular contractions and transient bundle branch block (BBB), but the clinical course tends to be benign. Knowledge of the resolution and natural history of IMH does not come from the trauma literature but from complications that have occurred after surgical and percutaneous coronary interventions resulting in an IMH [25–29]. With conservative management, all IMHs tend to resolve spontaneously after 4 to 12 weeks, as documented by follow-up ECHO [25–29]. A large IMH can cause a BBB, bifascicular block, or complete heart block if it is within the interatrial septum near the atrioventricular node [30, 31]. This requires close observation, but the treatment is supportive because the IMH will resolve with time.

Valvular Injury

Another common injury pattern from blunt cardiac trauma is papillary muscle rupture leading to acute valvular regurgitation. This can occur with the tricuspid and mitral valves. Acute aortic valve injury has also been described; however, only one case of BCI causing acute pulmonary valve disruption has been documented [3].

Injury to the papillary muscle, the chordae, or the mitral valve is well documented [32, 33]. A review of the world’s literature on the subject from 1964 to 2010 identified 82 reported patients, and 57% required a valve replacement [34]. The papillary muscles were most commonly injured, followed by the chordae, and then the mitral leaflets [34].

The same injury pattern can occur with the tricuspid valve, although the presentation may be subtler because wide-open tricuspid regurgitation can be well tolerated. In fact, patients may be asymptomatic after the accident and only be diagnosed with tricuspid regurgitation months or years later [35]. However, this is not true in all cases, and acute right ventricular failure has been described in patients with complete papillary disruption [36]. An association of traumatic tricuspid injury and heart block has been documented, ranging from first degree to complete heart block [37]. Thus, close observation is necessary and may require transvenous temporary pacing, followed by permanent pacemaker insertion if necessary. Diagnosis of all valvular injuries is made by ECHO, and the operation required is usually a valve replacement, although recently more of these injuries are being treated by repair or annuloplasty with valve salvage [38–40].

Postinjury findings of a new holosystolic murmur cannot always be attributed to valve pathology. A rare, but well-described, lesion occurring after cardiac trauma is an acute ventricular septal defect (VSD), which usually occurs within the membranous portion [41–43]. The defect may be single or multiple [44]. Depending on the size of the defect, there may be little hemodynamic change or complete hemodynamic instability [45, 46]. Diagnosis can be made promptly using ECHO.

Coronary Artery Injury

Acute coronary artery dissection can also occur from blunt cardiac trauma. The pathophysiology in these cases tends to be direct impact over the left anterior descending coronary artery or the left main coronary artery [47–49]. The area of the dissection usually originates in a previously diseased portion of the left anterior descending coronary artery and very rarely occurs in an elastic, normal vessel. The dissection is likely related to plaque rupture, which initiates the dissection as the intima over the plaque tears, and the blood flow then propagates the injury [50, 51]. The torn intima then creates a flap that obstructs the blood flow and produces infarction. The infarction will usually involve the apex, septum, or both, as the septal branches and distal left anterior descending artery occlude and thrombose. Acute coronary occlusion and dissection from blunt trauma has been successfully managed by percutaneous techniques [48, 51–53]. An elevated level of cardiac troponin I (cTnl) with ST segment elevation represents coronary artery occlusion or dissection and should prompt immediate coronary angiography with revascularization as appropriate [54, 55].

Traumatic VSD also requires surgery and patch closure in most cases with a large defect or the presence of a significant shunt. The first successful transcatheter closure of a traumatic VSD was described in 2004 [56] and
has been successfully performed many times since [57]. However, it is important in these cases for the clinician to realize that unlike a congenital VSD with a fibrous ring of tissue around the defect, a traumatic VSD will be surrounded by necrotic and friable muscle that is not resilient and may not hold or support an occluder device [58].

Serum Troponin Levels After Cardiac Trauma

The literature on detecting a BCI by serum troponin measurement is somewhat controversial. There are three types of Tn: TnI, which binds to actin to hold the troponin-tropomyosin complex to the myofilaments; TnT, which binds to tropomyosin to form the troponintropomyosin complex; and TnC, which binds to calcium ions to cause a conformational change in TnI. All three types of Tn are present in cardiac and skeletal muscle. There is no Tn in smooth muscle.

TnI is present in skeletal muscle and cardiac muscle. The three isoforms of Tn I are: cTnI, a slow skeletal muscle TnI isoform (ssTnI), and a fast skeletal muscle TnI isoform (fsTnI). They are encoded by different genes and are specifically expressed in cardiac, slow skeletal muscle, and fast skeletal muscle, respectively. Commercially available testing kits purport to detect one isoform and not the other, such that no cross-detection occurs between the cardiac and skeletal isoforms. The cardiac isoform (cTnI) differs from the skeletal muscle isoform (ssTnI) by only an extended N-terminal sequence of 32 amino acid residues [59]. If there is significant cross-reactivity, then an elevated TnI after blunt chest trauma may represent myocardial damage (mostly cTnI) or may represent skeletal muscle and chest wall damage (mostly ssTnI or fsTnI).

When the initial monoclonal antibody enzyme immunoassays were first developed, separate studies found cross-reactivity with both skeletal and cardiac TnI in up to 30% of the antibodies [60, 61]. In addition, intraassay and interassay variances within different commercially available TnI assay kits range from 4% to 14% [62, 63]. Even recently, the “high-sensitivity” or “fourth generation” cTnT (but not the cTnI) assays have shown cross-reactivity in patients with diseased skeletal muscle [64].

Both cTnI and cTnT have shown utility in detecting early myocardial infarction [65, 66]. In addition, sTnI has also become a useful marker of skeletal muscle damage in orthopedic and soft tissue injuries [67]. In research comparing levels of sTnI and cTnI in patients with skeletal muscle damage secondary to trauma or intense exercise, but no known cardiac injury, the results consistently show elevated sTnI (8 to 17 ng/mL) with normal cTnI (<1 ng/mL) [67-70]. Thus, both assays are useful and specific for the detection of cardiac or skeletal injury. However, it is an important distinction if the elevated TnI is skeletal or cardiac in origin, because a patient with blunt chest trauma, combined cardiac and skeletal injury, or a multitrauma patient with many injuries, may have elevations of both.

The research on this is imperfect due to poor confirmation of BCI in patients with an elevated cTnI level. Some research will consider a patient to have a BCI based on electrocardiographic (ECG) changes alone, with or without elevated cTnI. Some research will only classify a patient as having a cardiac injury if abnormalities are seen on ECHO. And finally, most reports have not clarified the level of cTnI considered as a positive result (>0.4 ng/mL or ≥1 ng/mL). Looking at the evidence from 6 research reports that addressed this topic, where cardiac injury was confirmed by ECHO, the results were consistently the same. Approximately half of the patients with a positive cTnI actually did have a cardiac injury by ECHO [71-76]. Pooling the data, 213 patients had significant blunt chest trauma and positive serum cTnI, and only 105 had a documented cardiac injury by ECHO (49%) [71-76].

Further research will need to determine what level of cTnI should be considered positive. Only 2 reports have provided that data. Using a cutoff level of 1 ng/mL, 60% to 70% of patients with a serum cTnI level exceeding 1 ng/mL after blunt chest trauma actually had a documented cardiac injury [74, 75]. Thus, based on the data that have been published, a cTnI level exceeding 1 ng/mL is a “significant” finding and the individual should be further evaluated with ECHO and close monitoring [74, 75]. Levels between 0.4 and 1 ng/mL are likely indicative of minor myocardial injury that will not be appreciated by ECHO in half of the patients and have little clinical sequelae [71-75].

These studies also provided the answer to the interesting question of what percentage of patients with a completely normal cTnI actually did have a visible IMH or cardiac injury by ECHO? The answer is 2 of 296, or 0.6% [71-76]. Thus, patients with a suspicion of BCI with a normal cTnI (<0.4 ng/mL) 4 to 6 hours after injury can be safely considered to not have BCI [77].

Electrical Disturbances

Atrial and Conduction Dysrhythmias

Sinus tachycardia is the most common ECG abnormality among trauma victims. Results from large case series suggest that ECG abnormalities, other than sinus tachycardia, are present in 1% to 6% of patients after chest trauma, and atrial fibrillation (AF) is the most common [78, 79]. One series reported that 9 of 240 patients (4%) with chest trauma showed AF on their initial ECG [80]. However, it is important to realize that the AF may not be due necessarily to direct cardiac injury, because the incidence of AF after chest trauma is the same as for head and abdominal trauma [79]. Patients with rapid AF and hemodynamic compromise should be cardioverted. Asymptomatic hemodynamically stable patients may benefit from a rate-control strategy using β-blockers or calcium channel blockers with consideration of delayed elective electrical cardioversion [81].

Paroxysmal supraventricular tachycardia after BCI is rare but has been reported [9]. Because paroxysmal
supraventricular tachycardia can lead to rapid hemodynamic compromise, especially after trauma, it is important to rapidly terminate this dysrhythmia by administering adenosine, β-blockers, or electrical cardioversion [81].

More common and more difficult to interpret are transient premature ventricular contractions and BBBs that appear after traumatic blunt injury. A classic medical teaching has been that BCI produces a right BBB. This certainly does occur and has been reported [55, 80, 82]; however, a right BBB, even when persistent, ultimately has almost no long-term sequelae and does not affect a patient’s functional outcome [81]. Thus, most ECG abnormalities after BCI tend to be transient, intermittent, evolving, and clinically irrelevant [71–77].

Ventricular Dysrhythmias
Ventricular dysrhythmias after chest trauma are less common than atrial and conduction disturbances but are much more lethal and were likely present in most of the individuals who died at the scene. Because these trauma victims never arrive in the trauma bay, the true incidence of ventricular dysrhythmias is impossible to determine. These dysrhythmias can lead to rapid deterioration, so early and complete evaluation with ECHO is warranted [81].

Initial ECG and Clinical Significance
When the Eastern Association for the Surgery of Trauma (EAST) published its practice guidelines for the management of BCIs in 2012, the only recommendation that was supported by Level 1 Evidence was to obtain an admission ECG on all patients in whom BCI was suspected [83]. This is mostly because Level 1 Evidence is hard to find on this topic. They also recommended (Level 2 Evidence) that if the admission ECG does show a new abnormality, that the patient should be admitted for continuous monitoring [83]. These recommendations were based on 5 published studies [71, 72, 84–86]; however, many more publications have addressed the issue of ECG in diagnosing BCI [71–77, 84–87]. One report suggested that ECG alone can be used to rule out a BCI [85]. Most studies, however, do not conclude that ECG by itself can make that determination. [71–76, 84, 86, 87] because a significant number of patients who have a normal admission ECG will be found to later (within 24 hours) have a clinically significant cardiac injury [71, 72, 84]. Depending upon the severity of injury, this number may be as high as 41%, although these data were determined by using serum cTnI and not cTnT [83, 84]. Regardless, based on most of the information, most experts agree that ECG alone is not sufficient to exclude a BCI. However, the combination of a normal ECG and a normal cTnI (<0.4 ng/mL) almost completely excludes a clinically significant BCI, with negative predictive values ranging from 98% to 100% [71–77]. Thus, patients with a suspicion of cardiac injury with a normal (<0.4 ng/mL) serum cTnI and a normal ECG may be safely discharged home from the emergency department.

Late Complications After Cardiac Trauma
Most patients recover from blunt cardiac trauma without any long-term sequelae [74]. However, a few patients with rare and bizarre late complications have been reported, including the late onset of complete atioventricular block, delayed cardiac rupture, heart failure, coronary-to-pulmonary artery fistula, pericardial effusion, coronary artery occlusion, and constrictive pericarditis [88–94]. One study that reevaluated patients with BCI 12 months after injury with 24-hour Holter monitoring and nuclear flow studies found that 33% had cardiac abnormalities that had persisted since the injury [95]. For this reason, it is recommended that any patient who sustains documented cardiac trauma be reevaluated within 3 to 6 months of injury, particularly if there are any symptoms of heart failure [32, 94, 95].

Conclusions
The following Levels of Evidence are based on the University of Oxford Centre for Evidence Based Medicine published guidelines [96].

1. Obtain an initial ECG on all patients in whom BCI is suspected. Level of Evidence: 1b.
2. If the admission ECG shows a new abnormality, the patient should be admitted for continuous monitoring. Level of Evidence: 2b.
3. The combination of a fall greater than 20 feet and a sternal fracture should prompt a thorough cardiac evaluation because the incidence of cardiac injury in these patients is very high. Level of Evidence: 2b.
4. Even a large IMH should be treated conservatively because most will resolve over time, and usually within 3 months (see number 7). Level of Evidence: 2b.
5. After blunt chest trauma, a serum cTnI level exceeding 1 ng/mL is abnormal and is associated with a true cardiac injury in 60% to 70% of the patients. These patients will require a formal ECHO and close monitoring. Level of Evidence: 2b.
6. Patients with a suspicion of cardiac injury with a normal (<0.4 ng/mL) serum cTnI and a normal ECG may be safely discharged home from the emergency department. Level of Evidence: 2b.
7. An interatrial septal hematoma and/or a traumatic tricuspid valve injury can both produce complete heart block, and these patients require close monitoring and insertion of a transvenous pacemaker if heart block develops. Level of Evidence: 4.
8. Small tears at the venous-atrial confluence may be contained and the patient initially stable. Diagnosis by ECHO should prompt rapid surgical consultation because continued or late bleeding is likely. Level of Evidence: 4.
Selected References*


*The reference section of the print version of this article contains 80 selected references, the numbers of which correspond to their text citation numbers. The complete list of all 96 references is viewable via the full Reference Section, which is online only at http://www. annualsforthoracicsurgery.org.


