Common Cause of Mortality in Trauma but Manageable Nonetheless

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Foreword
Information about a real patient is presented in stages (boldface type) to expert clinicians (Drs Eachempati and Salemi), who respond to the information, sharing their reasoning with the reader (regular type). A discussion by the authors follows.

An obese but otherwise healthy 53-year-old man was unloading a delivery van when he was struck from behind by a slow-moving garbage truck, pinning him between the 2 vehicles. The patient was lying supine on the ground when emergency medical services arrived. He was alert and oriented with a Glasgow Coma Scale score of 15. The patient was complaining of pain in his right upper extremity and numbness in his left foot. His initial vital signs were notable for a heart rate of 100 bpm; his blood pressure was 150/90 mm Hg; and his breathing was elevated at 22 breaths per minute. The patient denied loss of consciousness and had no neck, back, or chest pain. Per emergency medical services, the patient’s midsternal appearance erythematous and his left lung field was coarse on auscultation, and he had a small abrasion on his left knee. The rest of his in-the-field examination was unremarkable. A cervical collar was placed, and the patient was placed on a back-board for transport. The patient’s vital signs remained stable en route per the emergency medical services’ report, although he became increasingly anxious and repeatedly removed both his cervical collar and a non-rebreather mask that had been placed empirically.

On arrival in our trauma bay, the patient was found to be in shock: pulse rate, 129 bpm; blood pressure, 69/51 mm Hg; respiratory rate, 20 breaths per minute; and O2 saturation, 80% on room air. He remained awake with a Glasgow Coma Scale score of 15 but now was complaining of difficulty breathing and of right-sided chest and back pain. His primary survey was notable for rhonchus, breath sounds bilaterally and right-sided chest wall crepitus; his abdomen was soft and had no peritoneal signs. The patient remained tachycardic, but his blood pressure improved to 127/113 mm Hg as 2 L crystalloid and 1 U red blood cells were given in bolus. His O2 saturation improved to 96% on a non-rebreather mask providing O2 at 15 L/min. His initial ECG demonstrated sinus tachycardia with questionable electric alternans in leads V1 and V2 (Figure 1).

The patient’s focused assessment with sonography for trauma (FAST) examination was unremarkable. A chest radiograph revealed extensive right subcutaneous emphysema, multiple right lateral rib fractures, a small right-sided apical pneumothorax, and bilateral patchy opacities consistent with pulmonary contusion or hemorrhage. His cardiomedastinal silhouette was normal (Figure 2A). A pelvic radiograph was negative for injury. A right chest tube was inserted with minimal serosanguinous output (Figure 2B), and a Foley catheter was placed.

Dr Eachempati: Care of traumatically injured patients demands the participation of various members of a large, interdisciplinart team and, depending on local practice, can involve surgeons, emergency medicine physicians, anesthesiologists, internists, and even cardiologists. Consequently, the basics of trauma management should be appreciated by anyone whose expertise might be called on during the care of an injured person. As a first rule, we all must understand that a hypotensive trauma patient such as this has bleeding until proven otherwise.

The mechanism of this man’s injury, a crushing blunt trauma to the torso, points to the chest or abdomen as a likely source of the blood loss. Our differential diagnosis at this juncture was thus quite broad and included, among other things, tension hemothorax or pneumothorax or cardiac tamponade from a blunt cardiac injury (BCI), from an aortic injury, or from flail chest; other potential diagnoses included pulmonary hemorrhage, tracheobronchial injury, sternal fracture, diaphragm or esophageal ruptures, and possible intra-abdominal and pelvic pathologies, including liver or splenic lacerations, hollow viscus injury, retroperitoneal hematoma, and pelvic fracture.

The patient’s responses to our immediate interventions, while positive, were nondiagnostic. His hemodynamics transiently improved after fluid resuscitation, and little blood drained from his chest tube. The results of our initial diagnostic studies were similarly nonspecific. The patient’s ECG showed sinus tachycardia; this increase in heart rate in response to hypovolemia and stress was of course expected. The patient’s FAST examination (which, for example, has been shown to have a sensitivity of 36% for blunt traumatic cardiac rupture1) was normal, and his chest radiograph confirmed the presence of right-sided pneumothorax.

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of a pneumothorax and thoracic injuries (ie, rib fractures, bilateral lung contusions) but suggested a normal-appearing mediastinum. It should be noted, however, that even 1 L blood can look like only a slight haze on a supine chest radiograph, and up to 300 mL can be obscured by the diaphragm on an upright film.

Knowing that patients with catastrophic aortic injuries usually die in the field,\(^2,3\) we thought it was unlikely that our patient had sustained such an injury. His abdominal examination was benign, and in conjunction with an unremarkable FAST examination and a normal pelvic radiograph, it was also less likely that our patient was bleeding out from an abdominal or pelvic source. Moreover, the patient was responsive to fluid, so we also knew that whatever was bleeding was doing so at a rate that we could keep up with, at least for the time being.

Hence, a somewhat more indolent intrathoracic process crept to the top of our differential: a BCI, especially in light of subtle clues on the man’s ECG, that is, variable QRS complex morphologies in leads V\(_1\) and V\(_5\). Did this represent electric alternans and thus a pericardial effusion? Correlating such an observation with physical examination findings is difficult in trauma situations. The very real dilemma of “Are those heart sounds actually muffled, or are they just hard to hear over the din of the trauma bay?” typifies the diagnostic complexity of BCIs and underscores that physical examination alone is insufficient for definitively diagnosing this type of trauma.

The patient needed additional diagnostic imaging as part of his trauma workup, and obtaining central venous access would prove prudent given his labile hemodynamics and because central venous catheters also have important diagnostic utility: the ability to measure central venous pressure (CVP). Rapid cross-sectional imaging and CVP determination would, we hoped, reconcile the significance of the above-mentioned ECG findings by further assessing for a BCI (an injury that had possibly caused, and might now have been manifesting as, a potentially lethal pericardial effusion).

**Patient presentation (continued):** Briefly stabilized, the patient thus underwent computed tomography (CT) of the head; cervical, thoracic, and lumbar spines; chest; abdomen; and pelvis. CT imaging of the head, spine, and pelvis was normal. However, the chest CT was notable for pericardial hemorrhage without evidence of tamponade, bilateral confluent airspace opacities consistent with pulmonary contusion and hemorrhage, a moderate-sized right pneumothorax (despite tube thoracostomy), moderate hemomediastinum/pneumomediastinum, and multiple right first through seventh rib fractures. The sternum was intact, and the aorta was normal in appearance (Figure 3). Of note, CT of the abdomen further demonstrated a 6×8-cm hepatic contusion without evidence of intraperitoneal contrast extravasation or free air. The patient sustained no other bony injuries; numerous radiographs of the extremities were all normal.

The patient’s initial laboratory tests revealed a hematocrit of 42.5%, a lactate of 3.0 mmol/L, and an ethanol level of 70 mg/dL. His aspartate and alanine aminotransferases were 843 and 886 IU/L, respectively. The remainder of his emergency room laboratory values were within normal limits; no cardiac enzymes had been drawn. The patient was then brought to the trauma/surgery intensive care unit for continued resuscitation and monitoring. Arterial and central venous lines were placed. His vital signs at this point were as follows: pulse, 121 bpm (sinus tachycardia); blood pressure, 130/86 mm Hg; respiratory rate, 24 breaths per minute; and \(O_2\) saturation, 98% on a nonrebreather mask providing 15 L \(O_2\)/min. His CVP was 22 mm Hg.

**Dr Eachempati:** Reviewing the patient’s CTs in real time, we became increasingly suspicious of an injury to the heart,  

![Initial 12-lead ECG on arrival in the trauma bay that is notable for sinus tachycardia and variable QRS complex amplitudes, most evident in leads V\(_1\) and V\(_5\).](http://circ.ahajournals.org/doi/abs/10.1161/CIRCULATIONAHA.115.018036)
a nebulous BCI. Yes, his chest CT demonstrated bilateral pulmonary contusions and his many right-sided rib fractures had likely caused his pneumothorax (which was not under tension). However, these findings could not explain his hemodynamic picture of tachycardia, hypotension (that was only transiently responsive to volume), and (most crucial) increased CVP, nor could his abdominal imaging, which showed only a liver contusion without extravasation. Then there were the worrisome cardiac findings on CT: There was blood in the pericardial space, and elsewhere in the mediastinum, we visualized several pockets of air and blood, yet there was no radiological evidence of a blunt aortic injury.

BCIs rarely occur in isolation\(^4\) and are encountered in \(\approx15\%\) of patients with blunt chest trauma.\(^6\) Thus, it was quite plausible that our patient had indeed sustained a heart injury in the setting of these concomitant findings. What is more, his elevated CVP indicated to us that the patient’s cardiac injury was likely confined to the pericardial space and hence the effusion. This intrapericardial fluid was now obstructing venous return to the right heart, which we witnessed as his high CVP. We were thus concerned that whatever the exact BCI, it might soon lead to frank tamponade. Therefore, we began alerting our cardiology and cardiac surgery colleagues.

**Patient presentation (continued):** Shortly after arriving in the trauma/surgery intensive care unit, the patient became increasingly tachypneic at 39 breaths per minute and hypoxic with an \(O_2\) saturation of 87% and a \(P_0_2\) of 55 mm Hg. He therefore was emergently intubated. Despite ongoing fluid resuscitation, the patient once again became hypotensive, necessitating 16 \(\mu\)g/min norepinephrine to maintain his mean arterial pressure at 65 mm Hg. His urine and chest tube outputs were minimal, and his lactate had increased to 5.04 mmol/L. Urgent bedside trans-thoracic echocardiography performed by cardiology staff showed a hyperdynamic left ventricle (70% ejection fraction) and a large pericardial effusion (Figure 4). Early diastolic invagination of the right ventricular free wall and marked respirophasic variability of transmitral and tricuspid diastolic velocities were noted (Figure 4).

*Dr Salemi:* As cardiac surgeons, we are always surveilling our postoperative patients for cardiac tamponade, in which blood accumulating at the operative site (resulting from failed hemostasis or bypass-induced coagulopathy) can overwhelm the heart in short order. It is rare, however, to be consulted for tamponade after chest trauma because most of those patients die before we ever see them.\(^6\) Clearly, this patient had a hemorrhagic pericardial effusion that was now verging on tamponade, that is, the state of circulatory decompensation that results from cardiac compression caused by increased intrapericardial pressure.

As blood enters the pericardial space, the noncompliant parietal pericardium prevents its expansion, and intrapericardial pressure therefore rises. This mounting pressure in turn reduces the diastolic compliance of all 4 cardiac chambers equally, which means that higher pressures are required to fill the heart. In the face of an early pericardial effusion, our patient was initially able to achieve these higher filling pressures through parallel increases in his systemic and pulmonary venous pressures. Coupled with his tachycardia and aggressive volume expansion by the trauma team, the patient temporarily staved off overt tamponade and circulatory collapse. However, these compensatory efforts were now becoming futile, as evidenced by his high filling pressures (ie, grossly elevated CVP) and ultrasound findings that were consistent with early tamponade physiology.

Confident of our diagnosis of a BCI, we knew that, as long as he remained reasonably stable with fluid and pressors, we could forgo a heroic bedside procedure and quickly bring the patient to the relative safety of the operating room, where we are best equipped to provide definitive treatments. As outlined in the discussion that follows, BCI is an umbrella term that encapsulates many uncommon injuries, not all of which require surgical repair. This patient’s problem, however, was not a nonoperative one.

**Patient presentation (continued):** The patient was brought emergently to the operating room. He became

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*Figure 2. A.* Initial supine chest radiograph demonstrating multiple thoracic injuries, including a right apical pneumothorax (green arrows), right subcutaneous emphysema, multiple right lateral rib fractures, and bilateral patchy opacities consistent with pulmonary contusion or hemorrhage. The cardi mediastinal silhouette appears normal. *B.* Follow-up supine chest radiograph after right-sided chest tube placement (blue arrow) showing residual right apical pneumothorax (green arrow).
profoundly hypotensive and tachycardiac while being prepped for a subxiphoid pericardial window. Therefore, a crash median sternotomy was performed, and the pericardium was opened, exposing copious blood and clot. Evacuation of this hematoma led to even more bleeding, the origin of which was obscured by upwelling. The patient was

Figure 3. Representative axial images from chest computed tomography. A, and B, Lung-window views demonstrating bilateral confluent airspace opacities (purple arrows) consistent with pulmonary contusion and hemorrhage and a residual, moderate-sized right pneumothorax (green arrows) despite the presence of a chest tube (blue arrow). C and D, Soft-tissue-window views showing pericardial hemorrhage (red arrows) without evidence of tamponade.

Figure 4. Representative images from emergency bedside transthoracic echocardiogram demonstrating a large pericardial effusion (red arrows). A, Apical 4-chamber view. B, Apical 2-chamber view. C, Parasternal short-axis view. D, Parasternal long-axis view showing early diastolic invagination of the right ventricular free wall (green arrow).
Immediately heparinized and placed on cardiopulmonary bypass, maintaining flows of 2.4 L·min⁻¹·m⁻² and a mean arterial pressure of 60 mm Hg. Under the relative protection of cardiopulmonary bypass, a prompt but thorough examination of the mediastinum soon revealed an ≈1-cm linear tear in the right inferior pulmonary vein near its entry point into the left atrium (Figure 5). The injury was repaired with 2 layers of 4-0 polypropylene suture. The patient was then warmed to 36°C and further resuscitated with a total of 1.5 L lactated Ringer’s solution, 2 U red blood cells, 1 U platelets, 10 U cryoprecipitate, and 250 IU of prothrombin complex concentrate. He was weaned from cardiopulmonary bypass after a total of 29 minutes on pump, at which point transesophageal echocardiography demonstrated normal ventricular function and no residual intrapericardial fluid. Mediastinal and bilateral chest tubes were placed; the chest was closed; and the patient was transferred critically ill, but stable, to the cardiothoracic surgery intensive care unit.

**Dr Salemi:** We planned on first relieving the patient’s intrapericardial pressure via a pericardial window, which can be performed quite expeditiously. Knowing we would likely still have to open the man’s chest to definitively address his underlying injury, we hoped that a window would rapidly improve his forward flow in the interim. Given his sudden decompensation, however, we jumped directly to sternotomy; and the patient was transferred critically ill, but stable, to the cardiothoracic surgery intensive care unit.

Table. Autopsy Findings in 303 Fatalities From BCI*  

<table>
<thead>
<tr>
<th>Finding</th>
<th>n (%)</th>
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<tbody>
<tr>
<td>Right ventricular injuries</td>
<td>121 (40)</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>Pericardial tears</td>
<td>108 (36)</td>
</tr>
<tr>
<td>Right atrial injuries</td>
<td>101 (33)</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/VC tear with epicardial hematoma</td>
<td>18 (6)</td>
</tr>
<tr>
<td>Left ventricular injuries</td>
<td>96 (32)</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>
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<tr>
<td>Left atrial injuries</td>
<td>47 (16)</td>
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<tr>
<td>Transmural left atrial rupture</td>
<td>39 (13)</td>
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<tr>
<td>Left atrial hematoma</td>
<td>5 (2)</td>
</tr>
<tr>
<td>Left atrial/ PV tear with epicardial hematoma</td>
<td>3 (1)</td>
</tr>
<tr>
<td>Other</td>
<td>53 (17)</td>
</tr>
<tr>
<td>Complete heart avulsion</td>
<td>13 (4)</td>
</tr>
<tr>
<td>Ventricular septal tear</td>
<td>12 (4)</td>
</tr>
<tr>
<td>Aortic valve injury</td>
<td>8 (3)</td>
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<tr>
<td>Tricuspid valve injury</td>
<td>6 (2)</td>
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<tr>
<td>Mitral valve injury</td>
<td>5 (2)</td>
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<tr>
<td>Coronary artery tear</td>
<td>5 (2)</td>
</tr>
<tr>
<td>Coronary artery dissection</td>
<td>3 (1)</td>
</tr>
<tr>
<td>Pulmonary valve injury</td>
<td>1 (&lt;1)</td>
</tr>
</tbody>
</table>

BCI indicates blunt cardiac injury; IVC, inferior vena cava; and PV, pulmonary vein. *Many patients had >1 finding. Data are presented as number of patients with a given finding; percentages are expressed in terms of the 303 total patient fatalities. Adapted from Yousef and Carr with permission from the publisher. Copyright © 2014 Elsevier. Authorization for this adaptation has been obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.
Conversely, BCIs that bleed into a pleural cavity, which would require a communication via a concomitant pericardial tear, might be more rapidly fatal, given that the resultant unchecked exsanguination into the chest would briskly drain a substantial portion of the circulating blood volume. Such a scenario possibly explains the high prevalence of pericardial tears seen in BCI fatalities (Table). In retrospect, it should have been more reassuring to us that little blood had emptied from the patient’s chest tube.

Intraoperatively, pulmonary vein bleeding can be controlled momentarily by digital compression or by hilar clamping while definitive repair is undertaken. Cardiopulmonary bypass can be a very useful adjunct in that it provides life-sustaining circulatory support and makes for a drier operative field. If primary suture repair is not feasible, lobectomy or pneumonectomy may be required because pulmonary vein ligation causes venous gangrene, given the segmental nature of the pulmonary circulation.

Patient presentation (continued): The patient’s postoperative course was not without complications. He required reintubation on postoperative day 4 secondary to concomitant blossoming pulmonary contusions and Moraxella catarrhalis and methicillin-susceptible Staphylococcus aureus pneumonias (after having been extubated the previous day without incident). On postoperative day 12, he developed atrial fibrillation with rapid ventricular response, necessitating β-blockade and amiodarone for control. The patient was found to have a left popliteal vein thrombus on postoperative day 19, for which he was started on anticoagulation. He was ultimately discharged to rehabilitation on postoperative day 20. Of note, the patient’s liver contusion had been managed nonoperatively and resolved without incident. Two months after his injuries, the patient was without complaints and was walking unlimited distances and climbing stairs without difficulty.

Dr Salemi: All told, this patient has made a remarkable recovery, and he embodies what we in surgery call “a huge save.” His positive outcome depended on equal parts superb trauma care; a keen, swift diagnosis; readily available operative staff; and luck. In hindsight, his case may now appear somewhat straightforward, but patients with BCIs serve physicians with complex diagnostic and management questions, which we address next.

Discussion
Cardiac injuries are identified in roughly 12% of all blunt-trauma fatalities and, when found, are the causative factor in nearly half of those deaths. About 50% of BCIs occur after motor vehicle crashes; 35% are the result of pedestrians being struck; 9% are secondary to motorcycle crashes; and the balance are mostly attributable to falls from significant heights. Although less frequent than blunt aortic injuries, nonpenetrating injuries to the heart (ie, BCIs) are often lethal if the injuries include structural damage to a cardiac chamber. However, as our case demonstrates, a BCI must not always come as a death knell. Understanding this entity is therefore requisite if similar saves are to be made.

Patients with BCIs who survive to emergency room admission often pose medical dilemmas. Precise diagnostic definitions of and management algorithms for BCI are lacking, especially for patients whose injuries manifest only as ECG or cardiac enzyme disturbances. Moreover, the use of vague descriptors like myocardial contusion and myocardial concussion in characterizing these types of BCIs can further cloud the subsequent management of such injuries, often leading to unnecessary monitoring and wasteful use of resources. This jargon should be abandoned in favor of terms that specifically describe the pathology that is present. Under this more precise framework, BCIs can thereby be categorized as involving structural damage to the heart (ventricular rupture, coronary artery dissection, etc), electric or conduction abnormalities, or some combination of these manifestations. We briefly discuss common diagnoses within these categories and highlight key points in their treatment with an emphasis on specific BCIs that are both manageable and survivable. We close by reviewing a screening algorithm whose simplicity demystifies diagnosing BCIs in patients.
whose injuries are more subtle than those sustained by our patient.

**Structural Injuries**

Structural damage to the heart can occur after blunt chest trauma when compressive, deceleration, or shearing forces are transmitted through the sternum, chest wall, or spinal column. The ensuing loads can crush or tear underlying cardiac tissues, most commonly, the right ventricle because of its location just beneath the sternum.4,12 However, many of these injuries are devastating and rapidly fatal (Table) and of those patients surviving to the hospital, most will have sustained relatively less severe injuries including intramural hematomas (IMHs), coronary artery dissections, and/or valvular injuries.4

As is the case for BCIs as a whole, IMHs have nonspecific presentations. Patients may complain of angina-like chest pain or dyspnea. Physical examination may reveal a precordial thrill or murmur, but characteristic signs of a heart injury are often lacking. IMHs are best diagnosed with echocardiography and typically are found in the right ventricle.8,9 IMHs can cause premature ventricular contractions and transient bundle-branch blocks. Fortunately, their clinical course tends to be benign.9 Most IMHs resolve spontaneously after 4 to 12 weeks of conservative management.13–15 These injuries should therefore be followed up with serial echocardiography to ensure their complete regression.

BCI can also present as a coronary artery dissection whereby a direct impact over an already-diseased portion of an artery leads to an intimal tear, which serves as the nidus for a dissection when blood then enters the false lumen created by this tear.16 Myocardial infarction can result if the torn portion of the intima acts as a flap that obscures blood flow within the true lumen of the coronary artery or if propagation of the dissection otherwise obliterates distal perfusion.17,18 The ensuing ischemia will be evidenced on ECG and by elevations in cardiac troponins and should prompt immediate coronary angiography with percutaneous or surgical revascularization as appropriate.16,19

Valve injuries, particularly papillary muscle rupture leading to acute valvular regurgitation, are another type of structural BCI seen in survivors.20 As for IMHs, echocardiography is the method of choice for diagnosing valvular BCIs. Although new-onset tricuspid regurgitation after injury to a papillary muscle, the chordae, or the valve itself may be well tolerated, analogous injuries in the mitral position usually necessitate valve replacement.21

Saving patients with profound structural blunt trauma to the heart is very rare (compared with people having one of the above-listed “minor” injuries). As illustrated by our patient presentation, interventions in the severe cases (see the Table for the most common of these) must be made swiftly and are tailored to the specific injuries that are encountered. In terms of blunt pulmonary vein injuries like our patient sustained, only a handful of similar instances of traumatic vein rupture are described in the world literature, which dates back >40 years, and of those, only 10 other survivors have been identified.7,22–29

**Electric/Conduction Abnormalities**

Blunt injury to the heart can cause electric disturbances such as atrial and ventricular dysrhythmias and conduction abnormalities. These can either arise in isolation or be secondary to structural damage, as mentioned above. The most damning of these electroconductive BCIs is commotio cordis, in which sudden death occurs in someone (who does not have structural cardiovascular disease) after a blunt impact to the torso.30 Usually associated with blows to the chest in younger people playing competitive sports, commotio cordis is thought to befall when an impact transmitted to the myocardium strikes during an electrically vulnerable phase of ventricular excitability (ie, during repolarization), leading to intractable

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**Figure 7.** Blunt cardiac injury (BCI) screening algorithm. AMI indicates acute myocardial infarction; CT, computed tomography; MRI, magnetic resonance imaging; TEE, transesophageal echocardiography; and TTE, transthoracic echocardiography.
ventricular fibrillation. Unlike cardiac arrest resulting from hypoxic or ischemic causes, this injury tends to be refractory to cardiopulmonary resuscitation and defibrillation.

Atrial arrhythmias are common among all types of trauma, so attributing a specific atrial arrhythmia (e.g., sinus tachycardia or atrial fibrillation) to a direct cardiac injury is difficult. Their treatment thus should follow the normal management protocols for these abnormalities. With respect to pure conduction defects, it has historically been held that BCI can induce protocols for these abnormalities. With respect to pure conduction defects, it has historically been held that BCI can induce.

Many structural BCIs also have secondary electric or conduction manifestations. For example, pericardial effusions and cardiac tamponade can be evidenced on ECG as electric alternans, wherein the amplitude and vector of successive P waves, QRS complexes, and T waves alternate as the heart swings back and forth in the fluid-filled pericardium (Figure 6). In addition to the aforementioned bundle-branch blocks that can develop after IMHs and to the ST-segment changes that can result after coronary injury, it is worth noting that an association between traumatic tricuspid injury and heart block (ranging from first to third degree) has also been reported. Here, too, therapy is dictated by the accepted standards of care for the given block and should occur in conjunction with any possible tricuspid valve procedure.

BCI Screening Algorithm
Not all BCI patients arrive in extremis. Often, the only indication that a patient may have sustained a BCI is the reported mechanism of the injury, and that alone is sometimes sufficient for the cardiologist to service to be consulted. Although those with clear hemodynamic compromise should undergo standard assessment by a trauma team with the immediate goal of stabilizing the patient with injury-specific interventions, in the algorithm shown in Figure 7, we instead focus on those patients with less obvious presentations. These recommendations, which are based largely on a recent practice management guideline published by the Eastern Association for the Surgery of Trauma, should steer providers (whether emergency room doctors or cardiologists) through the workup of a suspected BCI in an otherwise stable patient.

Because there are still no accepted criteria, it has been suggested that all stable patients with significant blunt trauma to the chest be screened for BCI, beginning with an ECG and troponin I measurement. The combination of a normal ECG and a normal troponin I level has a negative predictive value that nears 100%. Thus, these results establish that BCI has been ruled out. Abnormalities in either of these tests should trigger admission to a monitored unit where both the ECG and troponin I can be followed serially, although the optimal timing and duration of this monitoring have not yet been established. Admitted patients with persistent arrhythmias, troponin I elevation, or hemodynamic instability should subsequently undergo either transthoracic or transesophageal echocardiography as a diagnostic test (rather than as an initial screening modality) to further characterize their injuries. Additionally, because similar derangements may be seen in both BCIs and acute myocardial infarctions and because treatments for these diagnoses are often divergent (e.g., anticoagulation is beneficial in acute myocardial infarction but could be harmful in a BCI with concomitant hemorrhage), cardiac CT or magnetic resonance imaging should be used to help differentiate between BCI and acute myocardial infarction in patients with abnormal ECGs, troponin I concentrations, or echocardiograms.

Conclusions
This case exemplifies the complexity of BCIs and demonstrates how the care of patients with blunt injuries to the heart requires the coordination of physicians representing numerous specialties, from the trauma surgeon inserting a chest tube to the cardiologist performing bedside transthoracic echocardiography, to the cardiac surgeon repairing a pulmonary vein laceration. The scope of possible injuries is broad, and although many carry high mortalities, with a thorough understanding of this type of trauma, and a little luck, even some of the most severe BCIs can be managed.

Disclosures
None.

References


**Key Words:** heart arrest ■ resuscitation ■ shock ■ surgery ■ wounds and injuries
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