Clinically Significant Blunt Cardiac Trauma: Role of Serum Troponin Levels Combined with Electrocardiographic Findings

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Background: **The true importance of blunt cardiac trauma (BCT) is related to the cardiac complications arising from it. Diagnostic tests that can predict accurately if such complications will develop or not may allow early and aggressive monitoring or early discharge. We investigated the role of two simple and convenient tests, serum cardiac troponin I (cTnI) and electrocardiogram (ECG), when used to identify patients at risk of cardiac complications after BCT.**

Methods: **Over a 10-month period, 115 patients with evidence of significant blunt thoracic trauma were prospectively followed to identify the presence of clinically significant BCT (Sig-BCT), defined as cardiogenic shock, arrhythmias requiring treatment, or structural cardiac abnormalities directly related to the cardiac trauma. An ECG was obtained at admission and at 8 hours. Cardiac troponin I was measured at admission, at 4 hours, and at 8 hours. Transthoracic echocardiography was performed when clinically indicated. The sensitivity, specificity, and positive and negative predictive values of ECG and cTnI to identify Sig-BCT were calculated. Clinical risk factors for Sig-BCT were examined by univariate and multivariate analysis.**

Results: **Nineteen patients (16.5%) were diagnosed with Sig-BCT and, in 18 of them, symptoms presented within 24 hours of admission. Abnormal electrocardiographic findings were detected in 58 patients (50%) and elevated cTnI levels in 27 (23.5%). Electrocardiography and cTnI had positive predictive values of 28% and 48% and negative predictive values of 95% and 93%, respectively. However, when both tests were abnormal (positive) or normal (negative), the positive and negative predictive values increased to 62% and 100%, respectively. Other independent risk factors for Sig-BCT were head injury, spinal injury, history of preexisting cardiac disease, and a chest Abbreviated Injury Score greater than 2.**

Conclusion: **The combination of ECG and cTnI identifies reliably the presence or absence of Sig-BCT. Patients with an abnormal ECG and cTnI need close monitoring for at least 24 hours. Patients with a normal admission ECG and cTnI can be safely discharged in the absence of other injuries.**

Key Words: **Blunt cardiac trauma, Blunt myocardial injury, Troponin, Electrocardiography, Echocardiography, Cardiac complications.**

J Trauma. 2001;50:237–243.

Blunt cardiac trauma (BCT) refers to a spectrum of injuries ranging from simple electrocardiographic changes to free wall rupture.[1] Since it was first described in 1676 by Borch,[2] BCT has been the subject of much controversy,[1–6] predominantly because of the imprecise methods of diagnosis. The lack of a well-accepted "gold standard" does not allow the evaluation of the sensitivity and specificity of different diagnostic tests in detecting BCT. The electrocardiogram (ECG) and the MB fraction of creatine phosphokinase (CPK-MB) are easy and convenient tests but are affected by a wide variety of diseases.[7] Echocardiography detects cardiac motion abnormalities but cannot be used as a screening test because it is not immediately available in many institutions.[4] Recently, cardiac troponin serum levels have been used, but with conflicting results.[8–14]

Regardless of the definition of BCT, this entity becomes important only when it is associated with significant symptoms, such as arrhythmias or hypotension, or causes anatomic defects, such as valvular, septal, or free wall rupture. Patients at risk of developing these complications should be recognized early after injury, monitored closely, and treated promptly. On the other hand, patients without such risk should be managed cost-effectively by avoiding unnecessary monitoring and allowing early discharge. The diagnostic tests used should be easy to perform and repeat, inexpensive, and risk-free. In this way, appropriate patients could be screened liberally to identify those at risk of cardiac complications, that is, those who have suffered clinically significant BCT (Sig-BCT).

In this study, we evaluate the role of cardiac troponin I (cTnI) and electrocardiography in identifying Sig-BCT. We also analyze risk factors of Sig-BCT that are related to the

Submitted for publication August 3, 2000.
Accepted for publication October 28, 2000.

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Presented at the 60th Annual Meeting of the American Association for the Surgery of Trauma, October 11–15, 2000, San Antonio, Texas.
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type and severity of injury and the physiologic status of the patient.

MATERIALS AND METHODS

This study was approved by the institutional review board. Between September 1999 and June 2000, all patients admitted to our Level I academic trauma center who sustained blunt thoracic trauma and possible BCT were followed prospectively. BCT was considered in such patients when one or more of the following existed: rib fractures, sternal fracture, thoracic seat-belt sign, pneumothorax, hemothorax, or pulmonary contusion. All trauma patients were managed by dedicated trauma surgeons who offered in-house coverage around the clock.

By protocol, a chest radiograph, 12-lead ECG, and cTnI serum level were ordered at admission. The ECG was repeated at 8 hours and cTnI was measured at 4 and then 8 hours after admission. Additional ECG and cTnI, and two-dimensional echocardiography, were performed when clinically indicated. The ECG was considered abnormal if a conduction abnormality, ST-segment elevation or depression, arrhythmia, or T-wave inversion were present. Sinus tachycardia and bradycardia or nonspecific ST-segment and T-wave changes were recorded but not considered significant findings.

Cardiac troponin I was measured using a one-step immunometric assay (Heterogenous Immunoassay Module, Dimension RxL, Dade Behring, Newark, DE).[15] The assay has a reference range of 0.04 to 50.00 ng/mL. Cardiac troponin I was considered abnormal if values were greater than 1.5 ng/mL. This assay has no cross-reactivity with skeletal muscle troponin I.[16,17]

Patients were considered to have Sig-BCT when any of the following cardiac complications were observed: arrhythmia requiring treatment, pericardial effusion requiring treatment, unexplained hypotension (systolic blood pressure <90 mm Hg) requiring vasopressors, or cardiogenic shock requiring inotropes.

Statistical Analysis

We calculated the sensitivity, specificity, and positive and negative predictive values of ECG, cTnI, and the combination of both in predicting Sig-BCT. A simple κ coefficient was derived to test the agreement between ECG and cTnI; κ values of less than 0.40 reflect poor agreement, values between 0.40 and 0.75 reflect fair to good agreement, and values above 0.75 indicate strong agreement.[18] We also compared demographic, physiologic, and injury severity parameters between patients with and without Sig-BCT. Comparisons in the univariate analysis were done by the *t* test for continuous variables and χ^2 and Fisher's exact tests for categorical variables. Variables that were different at $p < 0.2$ were selected for stepwise logistic regression to identify independent risk factors of Sig-BCT. For the multivariate analysis, continuous variables were converted to dichotomous variables using clinically significant cut-off points (i.e., age >55 or ≤ 55 years, SBP >100 or ≤ 100 mm Hg, Glasgow Coma Scale score >12 or ≤ 12, Injury Severity Score >25 or ≤ 25, and the six Abbreviated Injury Scores (AISs) >2 or ≤ 2. A level of statistical significance at $p < 0.05$ was maintained for all comparisons.

RESULTS

Patient Characteristics

During the 10-month study period, 115 patients (79 men, 36 women) satisfied the inclusion criteria and were enrolled in the study. Of them, 69 (60%) were admitted after a motor vehicle crash, 26 (23%) as pedestrians hit by an automobile, 9 (7%) after a fall from a height, and 11 (10%) after crush injuries[4] or assaults.[7] Fifty-eight patients had rib fractures and 67 suffered lung injuries. Of 69 patients with extrathoracic injuries, 19 had intra-abdominal injuries, 32 had head injuries, and 51 had long bone or pelvic fractures. Of the 69 patients involved in a motor vehicle crash, 35 had a thoracic seat-belt sign.

Sig-BCT

Nineteen patients (16.5%) suffered Sig-BCT (Table 1). Of these 19, 15 underwent transthoracic echocardiography, which documented abnormalities in 7. Of the remaining eight, echocardiography was inconclusive in two because of underlying chest injuries, and normal in six patients. An additional nine patients without Sig-BCT had an echocardiogram that revealed no abnormalities. Of the seven patients who had unexplained hypotension, all required dopamine greater than 5 μg/kg/min, and two required the addition of epinephrine. Five of the seven patients had an echocardiogram performed, of which two were abnormal. One revealed tricuspid regurgitation, and the other, left ventricular dysfunction with apical hypokinesis. The manifestation of Sig-BCT occurred within 24 hours of admission in all but one patient, who was found to have hemopericardium 6 days after admission. At that time, this patient was in the intensive care unit (ICU) for severe associated injuries. Seven Sig-BCT patients died, but the death was directly related to the cardiac injury in only three (43%). An additional seven patients without Sig-BCT died because of severe associated injuries.

Table 1 Cardiac Complications Identified in 19 Patients with Clinically Significant Blunt Cardiac Trauma

Complication	No. of Patients (n = 19)
Arrhythmias requiring treatment	
Atrial fibrillation	4
Supraventricular tachycardia	3
Unexplained hypotension requiring vasopressors	7
Cardiogenic shock requiring inotropes	4
Hemopericardium requiring pericardiocentesis	1

ECG and cTnI

Abnormal electrocardiographic findings were detected in 58 (50%) patients and elevated cTnI levels in 27 (23.5%). All 58 abnormal ECGs and 22 of the 27 abnormal cTnI measurements were detected immediately after admission. In five patients, cTnI levels were initially normal but became abnormal within 4 hours of admission. All five of these patients had an abnormal initial ECG. The level of cTnI did not correlate with the severity of the cardiac dysfunction.

Figure 1 shows the relationship of electrocardiographic and cTnI findings with the development of Sig-BCT, indicating that when both tests were negative, no cases of Sig-BCT were detected. ECG and cTnI were in significant disagreement, as shown by a very low value of 0.065 (95% confidence interval [CI]: −0.086, 0.217). Therefore, ECG or cTnI alone had poor to moderate sensitivity, specificity, and positive predictive value. The negative predictive values of each test individually were good, but still not 100%. The combination of both tests predicted more reliably the presence or absence of Sig-BCT by increasing the positive predictive values to 62% when both tests were positive and the negative predictive value to 100% when both tests were negative (Table 2).

Risk Factors of Sig-BCT

Patients with Sig-BCT were older (53 ± 22 vs. 44 ± 16 years, $p = 0.076$), had a higher incidence of shock on arrival (36% vs. 7%, $p = 0.018$) and Glasgow Coma Scale score <12 (58% vs. 18%, $p = 0.050$), and had a higher Injury Severity Score (29.5 ± 10 vs. 14 ± 12, $p < 0.001$) compared with patients without Sig-BCT. They also stayed longer in the ICU (19 ± 23 vs. 6 ± 12 days, $p = 0.024$) and the hospital (30 ± 23 vs. 14 ± 20.5 days, $p = 0.003$), and had a higher mortality rate (7 of 19, [37%] vs. 7 of 96 [7%], $p = 0.002$). Table 3 shows a list of all risk factors examined. All patients with Sig-BCT were admitted to the ICU for other associated injuries before developing cardiac complications. No patient was admitted to the ICU with isolated chest trauma to rule out cardiac injury.

Six risk factors were found to be independently associated with Sig-BCT: abnormal cTnI (adjusted relative risk [ARR], 5.83; 95% CI, 1.55–12.46; $p = 0.020$), abnormal ECG (ARR, 13.95; 95% CI, 4.75–18.62; $p = 0.002$), spinal injuries (ARR, 8.76; 95% CI, 2.57–15.01; $p = 0.007$), history of preexisting cardiac disease (ARR, 5.96; 95% CI, 2.37–7.36; $p = 0.006$), chest AIS >2 (ARR, 7.05; 95% CI: 1.49, 17.28; $p = 0.028$), and head injuries (ARR, 4.45; 95% CI, 1.28−8.92; $p = 0.035$). The combination of all independent risk factors showed concordance of 95% with an R^2 of 0.67.

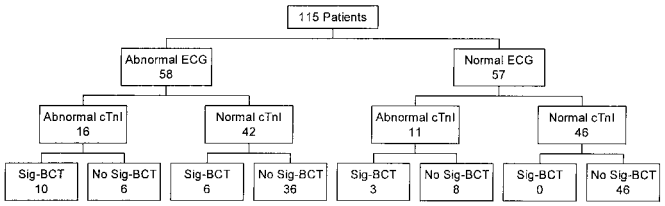

Fig. 1. *The relationship of electrocardiogram (ECG) findings and cardiac troponin I (cTnI) serum levels with the presence of clinically significant blunt cardiac trauma (Sig-BCI) among 115 patients with blunt thoracic trauma.*

Table 2 Diagnostic Value of Electrocardiogram (ECG), Cardiac Troponin I (cTnI) Serum Levels, and their Combination in Detecting Clinically Significant Blunt Cardiac Trauma

	ECG (%)	cTnI (%)	ECG + cTnI (%)
Sensitivity	84	68	100
Specificity	56	85	88
Positive predictive value	28	48	62
Negative predictive value	95	93	100

DISCUSSION

The major questions that arise regarding blunt cardiac trauma are, Which patients are at risk for developing cardiac complications? Are there any simple and reliable tests to identify these patients? How long should patients at risk be monitored and when could patients at no risk be safely discharged? The findings of our study support the use of two simple tests, electrocardiography and cTnI level, to diagnose Sig-BCT. When these tests are used in combination, the

Table 3 Selected Risk Factors of Clinically Significant Blunt Cardiac Trauma

Risk Factor	Relative Risk	95% CI	*p* Value
Age >50 y	2.22	0.968–5.086	0.071
Male gender	0.63	0.276–1.424	0.123
MVC	0.48	0.211–1.113	0.123
Thoracic seat-belt sign	0.43	0.133–1.377	0.175
Frontal impact	3.14	0.769–12.798	0.094
Lung injury	1.74	0.740–4.112	0.219
Rib fractures	2.75	1.061–7.140	0.043
Abdominal injury	2.95	1.335–6.505	0.016
Spinal injury	4.71	1.664–13.311	<0.002
Head injury	3.57	1.580–8.051	<0.004
SBP <100 mm Hg	3.38	1.502–7.592	0.018
GCS <12	2.47	1.099–5.532	0.051
Hx of cardiac disease	3.75	1.704–8.251	0.011
Abnormal CXR	1.54	0.580–4.077	0.478
Abnormal ECG	5.24	1.614–17.018	<0.002
Abnormal cTnI	7.42	3.129–17.580	<0.001
Abnormal ECG + cTnI	7.41	3.610–15.198	<0.001
ISS >25	4.86	2.109–11.184	<0.001
AIS chest >2	6.02	1.856–19.557	<0.001
AIS abdomen >2	3.45	1.595–7.480	<0.005

MVC, motor vehicle crash; SBP, systolic blood pressure; GCS, Glascow Coma Scale score; Hx, history; CXR, chest radiograph; ECG, electrocardiogram; cTnI, cardiac troponin I; ISS, Injury Severity Score; AIS, Abbreviated Injury Score; CI, confidence interval.

positive and negative predictive values improve significantly over those of each test used alone. If both tests are abnormal, then the probability that a patient with blunt thoracic trauma will develop symptoms of Sig-BCT is 62%. For this reason, such patients should be placed in a highly monitored environment. Even more importantly, when both tests are normal, the probability of BCT is 0%, and therefore, such patients can be safely discharged from the hospital in the absence of other injuries.

Electrocardiography alone is unreliable in detecting the presence of Sig-BCT. There are no specific electrocardiographic abnormalities that characterize this type of injury. Patients with blunt thoracic trauma frequently show abnormal ECG tracings associated with temporary conduction defects that rapidly return to normal, or even purely electrical phenomena that are unrelated to cardiac injury but caused by anatomic problems associated with posttraumatic subcutaneous emphysema, hemothorax, or tissue edema. Therefore, the presence of electrocardiographic abnormalities does not equal the presence of Sig-BCT. This is evident by the positive predictive value of 28% found in our study, which is similar to other reports.[5,6,19] However, the ability of the ECG to rule out patients for Sig-BCT is much better, as shown by a negative predictive value of 95%. Other studies have also documented that patients with normal ECGs are unlikely to develop cardiac complications after blunt thoracic trauma.[20–25]

Along the same lines, cTnI has a poor positive predictive value (62%) but much higher negative predictive value (93%) for Sig-BCT. Recently, cardiac troponin has replaced CPK-MB as the laboratory test of choice for the diagnosis of myocardial infarction because of its higher sensitivity and specificity.[12] This experience has been extrapolated in trauma, and many institutions use cardiac troponin as the diagnostic test of choice for blunt cardiac trauma. However, the literature evidence is still sparse and contradictory. Ferjani et al.[13] have reported that, although troponin had a greater diagnostic value than CPK-MB, it was still unreliable in diagnosing myocardial contusion. On the other hand, Fulda et al.[26] have considered troponin as an excellent predictor of the development of significant electrocardiographic abnormalities, consistent with BCT. However, the sensitivity and specificity of troponin were far from ideal, 27% and 91%, respectively. In another study, Helm et al.[27] concluded that abnormal troponin levels were a highly sensitive and specific marker of myocardial cell injury in a cohort of 125 trauma patients who had transthoracic echocardiography. However, the sensitivity of troponin to detect BCT considering echocardiography as the gold standard was 22%, and the specificity was 86%.

Much of the inconsistencies, as described above, relate to the absence of a standard definition of BCT. In 1992, Mattox et al.[1] recommended that such confusing terms as cardiac contusion or cardiac concussion be abolished and that traumatic cardiac diagnoses describing the specific anatomic or electrocardiographic abnormalities be used instead. After this recommendation, we selected the term *clinically significant blunt cardiac trauma*, which refers to the presence of anatomic defects or electrocardiographic abnormalities causing clinical symptoms and requiring close monitoring and/or treatment. Regardless of which is really the entire spectrum of the disease, these are the significant events that the clinician should take into consideration when managing patients with blunt thoracic trauma. Therefore, in the absence of any gold standard to define BCT, we used a clinical definition, *Sig-BCT*, as the gold standard against which we evaluated the diagnostic tests, ECG and cTnI.

Other reports have used echocardiography in the diagnosis of BCT.[19,28–32] In our study, only 25 patients had echocardiography because we did not use it as the gold standard. Of them, 15 had Sig-BCT, but the echocardiogram was positive in only 7. Although echocardiography is very reliable in diagnosing structural abnormalities under ideal circumstances, it can hardly be used as an emergent diagnostic tool for trauma patients. Immediate availability is the exception rather than the rule in most institutions across the nation. The usefulness of echocardiography is limited if the test is used to confirm the presence rather than predict the development of cardiac complications. Anterior thoracic trauma with associated thoracic cage fractures or subcutaneous emphysema decreases the resolution of transthoracic echocardiography. Furthermore, it does not add to the detection of conduction defects and arrhythmias, which are the most common complication of BCT. Transesophageal echocardiography, although more accurate than transthoracic echocardiograpy, presents even more problems regarding availability and performance on nonintubated patients. A more appropriate role for echocardiography would probably be in patients with abnormal findings on both ECG and cTnI to detect motion or structural defects.

Additional risk factors were associated with Sig-BCT in our analysis. Not surprisingly, almost all variables related to severity of injury or physiologic compromise were more prevalent in the Sig-BCT group compared with the group without Sig-BCT. The multivariate analysis identified, besides ECG and cTnI, that spinal and head injuries and a history of preexisting cardiac illness were independent risk factors for Sig-BCT. Although a chest AIS >2 was also isolated as an independent risk factor, it is possible that the chest AIS was higher in patients with Sig-BCT exactly because the cardiac injuries were incorporated in the calculation of the AIS.

The appropriate length of time required for monitoring blunt thoracic trauma patients at risk for Sig-BCT is still being explored. Godbe et al.[33] reported that cardiac complications may occur up to 72 hours after trauma. Most other authors agree that the interval between admission and development of signs and symptoms of BCT is shorter than this and varies from 6 to 24 hours.[1,4,6,23,34] In our study, no patient with normal initial ECG and cTnI developed a cardiac complication. Therefore, further in-house monitoring of such patients for Sig-BCT is unnecessary. All patients with Sig-

BCT developed cardiac complications within 24 hours of admission, with the exception of one patient who was diagnosed with hemopericardium on the sixth day of his ICU stay. These patients had abnormal findings on one or both tests (ECG, cTnI) at admission. These findings suggest that the vast majority of patients who have abnormal findings on one or both tests do not need to be monitored longer than 24 hours for Sig-BCT.

Of the 19 patients, only 1 patient with supraventricular tachycardia manifested the signs of Sig-BCT at arrival. All other patients were diagnosed with Sig-BCT after admission to the ICU. By that time, cTnI levels were already determined. Although it is possible that some patients had Sig-BCT at arrival, we could not diagnose it before ICU admission in the absence of a pulmonary artery catheter and continuous electrocardiographic monitoring. In that regard, the test was more useful as a predictive rather than a diagnostic tool.

Our study is limited—as is every other study in the literature—by the lack of a gold standard for the definition of BCT against which reliable predictive values of other tests can be derived. We explained already the reasons for not selecting echocardiography as the gold standard but instead relying on a clinical definition that makes sense with regard to the clinical management of such patients. Autopsy may be an ideal gold standard but is applicable only to a few patients. Additionally, detailed autopsies are not standard in all institutions. For example, at the Los Angeles County institutions, detailed autopsies are performed only in certain groups of patients or at the request of a physician. Many trauma patients have "external" autopsies, because of the load of work borne by the coroner's office.

The only modest increase of the individual negative predictive values of ECG and cTnI from 95% and 93%, respectively, to 100% when the two tests are combined could be also viewed as a limitation of our conclusions about the need for both tests. One could argue that the expense of an additional test is not justified for such a small increase. However, this increase becomes important if patients are to be discharged on the basis of these tests. The negative predictive value of 100%, when these tests are combined, allows for such decision making to be made comfortably by the treating physician. All 19 patients with Sig-BCT required ICU admission for associated injuries. None of them was admitted to the ICU solely for the diagnosis of Sig-BCT. However, of patients who had no Sig-BCT according to negative initial cTnI and ECG, 21 had no associated injuries and remained in the hospital only for follow-up for possible blunt cardiac trauma. These are the patients who we recommend to discharge safely. Negative cTnI and ECG can change the management of such patients by allowing early discharge. In that regard, an initially negative cTnI and ECG can change the management of patients who do not have associated injuries and would have remained in the hospital only for additional evaluation for possible blunt cardiac trauma.

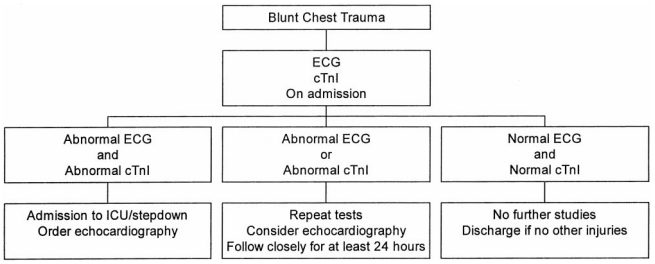

Fig. 2. *Treatment algorithm for patients with blunt chest trauma.*

In summary, our study reveals that the diagnostic value of the combined use of ECG and cTnI is highly accurate. In the presence of abnormal findings in both tests, patients should be monitored closely for at least 24 hours because almost two thirds of them will develop myocardial dysfunction. The absence of abnormal findings on the initial ECG and first cTnI equals absence of Sig-BCT, and such patients can be safely discharged in the absence of other injuries. On the basis of the above findings, we summarize in Figure 2 our recommendations for managing patients with blunt thoracic trauma at risk for Sig-BCT.

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DISCUSSION

Dr. Peter Mucha, Jr. (Morgantown, West Virginia): I would like to thank Dr. Salim and his colleagues from the University of Southern California for having forwarded the manuscript well in advance of this morning's program. I would also like to compliment them on the prospective nature of their study design, as well as the rather sophisticated statistical analysis that they employed.

They attempted to address a rather straightforward clinical question, and that is, whether cardiac myocyte-specific troponin I serum enzyme levels help in the screening of blunt thoracic trauma victims, for which they have coined another phrase, "significant blunt cardiac injury."

Is what we have deciphered from their data convincing enough for all of us to go back to our respective institutions with the attitude that henceforth all we are going to need is an EKG and a troponin I assay level at admission to safely discard a possible diagnosis of blunt cardiac injury?

First, there is no question that the subject of blunt cardiac injury, or pardon the expression, "myocardial contusion," remains as controversial and confusing as ever. In requesting a 5-year Medline search of the recent literature in preparation for this discussion, I found 192 citations identified under the heading of blunt cardiac injury and another 41 articles ascribed to myocardial contusion. I would be the first to concur with the author's statement that, to a large extent, much of the modern day misunderstanding of blunt cardiac injury is associated to our imprecise methods of diagnosis.

Just as important, however, has also been our continued inability to precisely define what we are talking about when it comes to the cardiac injury. Any variety of arrhythmias, conduction delays, and other electrophysiologic derangements are one thing, but when you are talking about objective structural wall motion abnormalities, septal defects, valvular disruptions, and hemopericardium, that is something different. They are not the same.

Again, can the authors further clarify what they actually mean by significant blunt cardiac injury? Did any of their patients with atrial fibrillation or supraventricular tachyarrhythmias have underlying preexisting or underlying cardiac disease? What do they mean by "unexplained hypertension requiring vasopressors," and how did this necessarily equate to the clinical scenario with blunt cardiac injury?

Finally, how was the diagnosis of cardiogenic shock made in four patients, and exactly what did they feel was the underlying pathophysiology involved?

Overall, I was impressed with the quality of the study and especially enjoyed Dr. Salim's presentation. The results of this study, however, should be taken for what they are worth. The unresolved problem is still how do we ensure appropriate knowledge and skills to recognize the possibility of blunt cardiac injury in all patients and, in turn, have the knowledge and ability to treat clinically significant issues? On the basis of my personal experience, more often than not,

this is still going to entail a bit more than simply measuring serum troponin I levels.

Dr. Christopher C. Baker (Chapel Hill, North Carolina): This is an interesting study. I have an observation and a question. If you have upgraded the data, it looks as if you have 11 patients per author in the series and 1.9 patients who had blunt cardiac injury, so it seems like making that diagnosis is a relatively low yield.

I wonder if you could also comment on two things. First, whether or not it made a difference to delay an operation in a patient you felt needed an operation. Second, can you tell us what the troponin cost in these patients?

Dr. Gerard J. Fulda (Newark, Delaware): The results are fairly similar to those we presented several years ago in that there were a group of patients with normal admission EKGs who subsequently developed significant cardiac rhythm disturbances.

My question is if you perform the EKG and troponin assay and either is positive, what is the time frame in which you are going to monitor these patients? How far out after admission was the last patient who had a significant event? When did that last significant event occur?

Dr. Ali Salim (Los Angeles, California): Thank you, Dr. Mucha. First, in terms of significant blunt cardiac trauma, we did use arbitrary definitions. We defined it as having any arrhythmias requiring treatment, so just PVCs didn't count as having significant blunt cardiac trauma, unexplained hypotension, cardiogenic shock or pericardial effusion that actually required treatment. The unexplained hypotension was decided by consensus on rounds. We had everyone blinded and, on the basis of the available data, we made the distinction whether the hypotension was attributed to a cardiac injury or to some other cause. So, it was only considered a significant injury if all other injuries or all other causes for hypotension were ruled out. We considered cardiogenic shock to be a cardiac index of less than two requiring inotropic support.

We had a total of seven arrhythmias. Of those seven, three patients had a cardiac history. There were still four patients who developed an arrhythmia who did not have a cardiac history.

In response to the pathophysiology of the cardiogenic shock, we are not really sure, but we do not think that it is some sort of destruction between the actin-myosin complex that reduces the contractility of the heart, depressing the cardiac output and leading to cardiogenic shock.

Regarding Dr. Baker's first question of whether it made a difference, all of these patients who were found to have significant injury were already in the ICU because of associated injuries. The diagnosis of blunt cardiac trauma never prevented them from having an operation. It really didn't change the management, per se. It just made us aware that the patient had an impaired heart.

In terms of the cost of troponin, I am sorry, I do not have that information.

Regarding Dr. Fulda's question, our response to an abnormal EKG or troponin is to observe the patient for another 24 hours, either on the floor or a monitored area, but not in the ICU. Most patients developed a cardiac complication within 24 hours, except for one patient who developed a pericardial effusion at 6 days.