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INSIDE

NEXUS vs. the Canadian C-spine Rule: Let the Battle Begin

ABSTRACT & COMMENTARY

Source: The Canadian c-spine rule versus the NEXUS low-risk criteria in patients with trauma. *N Engl J Med* 2003;349:2510-2518.

In 2000, Hoffman et al published the NEXUS study delineating five criteria to exclude the need for cervical spine (c-spine) radiographs in a "low-risk" emergency department (ED) patient population.[1] A year later, Stiell et al presented a study from Canada that identified different criteria for excluding c-spine injury (and hence the need for radiography) in a low-risk ED patient population.[2] These differing criteria are presented in the Table.

Stiell's purpose for this study, carried out at nine Canadian tertiary care hospitals, was to prospectively compare the NEXUS low-risk criteria (NLC) to the Canadian c-spine rules (CCR) for accuracy, reliability, clinical acceptability, and potential outcomes in patient care and radiography utilization.

In the study, 8283 patients prospectively were evaluated with both the NEXUS criteria and the CCR prior to radiography. There were 169 (2%) clinically significant injuries. Radiography was at the discretion of the examining physician, and approximately 30% of study patients had no radiographs. These patients were accounted for by telephone follow-up at 14 days. Notably, in 845 patients enrolled (10.2%), examining physicians neglected to determine ability to rotate the neck to 45°. These were termed "indeterminate" cases. When the two criteria were compared and these indeterminate cases were excluded, the CCR was more sensitive than the NLC (99.4% vs 90.7%), and more specific (45.1% vs 36.8%) and would have resulted in lower radiography rates (55.9% vs 66.6%). Stiell performs a secondary analysis in which all indeterminate cases are assumed to be positive, resulting in a sensitivity and specificity of 99.4% and 40.4%, respectively. Finally, he also calculates the same values if all indeterminate cases are assumed to be negative, resulting in a sensitivity and specificity of 95.3% and 50.7%, respectively. The study concludes that the CCR would have missed one clinically significant fracture, and the NLC would have missed 16 clinically significant

Table. NEXUS Low-risk Criteria and Canadian C-spine Rules: Deciding Who Doesn't Need an X-ray

NEXUS LOW-RISK CRITERIA	CANADIAN C-SPINE RULES		
1. No posterior mid-line cervical tenderness 2. No intoxication 3. Normal alertness 4. No focal neurologic deficits 5. No painful, distracting injuries	1. **NO** high-risk criteria: a. Age > 65 yrs, or b. Dangerous mechanism*, or c. Paresthesias in extremities	2. **AND** Presence of any low-risk criteria that allows safe assessment of range of motion: a. Simple rear-end collision, or b. Sitting position in the ED, or c. Ambulatory at any time, or d. Delayed (not immediate) onset of neck pain, or e. Absence of midline cervical tenderness	3. **AND** able to rotate neck to 45° left and right actively.

*Dangerous mechanism = Fall > 3 feet or 5 stairs; axial load to head (e.g., diving); motor vehicle crash at > 100 km/hr or with rollover or ejection; collision involving a motorized recreational vehicle; bicycle collision.

fractures; hence, the CCR is superior to the NLC for excluding c-spine injury in low-risk patients.

COMMENTARY BY ANDREW D. PERRON, MD, FACEP, FACSM

Decreasing the number of c-spine radiographs obtained on low-risk patients is important for any number of good reasons. Unnecessary irradiation, cost, length of time spent on a board in a collar, and prolonged ED stays are the reasons usually cited (appropriately) for this area of clinical research. As with so many resources in the ED (e.g., plain x-rays of the ankle and knee, computed tomography of the head), we have validated clinical decision rules to help guide us through the diagnostic algorithm.

So, which way should the busy clinician be looking for answers to help guide them through this evaluation: west, to Hoffman's NLC, or north, to Stiell's CCR? We know from Hoffman's study on 34,000 patients that when properly applied and prospectively evaluated, the NLC carry a 99.6% sensitivity. We know from this study that when properly applied, the CCR carry at worst a 95%, and more likely closer to 99% sensitivity. Both criteria markedly reduce unnecessary radiographs, and both result in very few missed injuries.

My conclusion? Pick one, learn it, and stick with it. I think either one gives you the ability to separate the wheat from the chaff with great diagnostic certainty. In my mind, the five NEXUS criteria are easier to remember than the 3 + 5 + 1 criteria for the Canadian rules (one does wonder how more than 10% of the patients had the Canadian rule misapplied [no neck rotation], when that was an integral part of the rule being studied). In the end, memorizing one algorithm over another is a small point, and in this age of the ubiquitous personal digital assistant (PDA), either set of criteria is only as far away as my coat pocket if I can't remember them. ❖

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Is Response to Nitroglycerin Predictive of Active CAD?

ABSTRACT & COMMENTARY

Source: Henrickson CA, et al. Chest pain relief by nitroglycerin does not predict active coronary artery disease. *Ann Intern Med* 2003;139:979-986.

To clarify nitroglycerin's (NTG) ability to distinguish ischemic chest pain, Henrickson and colleagues studied symptomatic patients in an academic emergency department (ED), evaluating NTG responsiveness as a predictor of coronary artery disease (CAD). Four hundred fifty-nine consecutive patients seen in Baltimore's Johns Hopkins Hospital comprised the study population. All reported chest pain, received NTG in the ED, and were hospitalized to rule out myocardial infarction. Average age was 59 years; 54% were female, 81% were Caucasian, and 18% African-American. Prior CAD was present in 31%, diabetes in 24%, hypercholesterolemia in 41%, tobacco abuse in 43%, and family history of CAD in 35%. Electrocardiograms (ECGs) obtained during pain were abnormal in 13%; 5% had ST segment depression, 5% had ST segment elevation, and 3% had left bundle-branch block. Patients whose chest pain resolved prior to ED arrival and those who could not rate pain on a 1-10 scale were excluded.

Each patient received NTG, either 0.4 mg sublingual or 0.4 mg oral spray. Ancillary treatments included oxygen, aspirin, morphine, and beta-adrenergic antagonist in 60%, 33%, 4%, and 3% of patients, respectively. NTG-responsive chest pain was defined as 50% or greater reduction in chest pain within five minutes, as assessed on a 1-10 point severity scale. Subsequent documentation of active CAD was defined as an elevated troponin T, angiography showing greater than 70% coronary artery stenosis, or positive exercise test with confirmatory imaging. Phone interviews were conducted four months after admission to assess interval cardiac events or testing results.

A total of 181 cases (39%) had greater than 50% reduction in chest pain and were termed NTG-responders (NTG-R). In contrast, 278 cases (61%) had insignificant or no response to NTG (NTG-NR). Subsequent cardiac evaluation, as described above, was suggestive of active CAD in 141 (31%) of the 459 patients.

Of those 141 with evidence of CAD and chest pain, 49 (35%) were in the NTG-R group. By comparison, of those in whom active CAD was excluded by testing, 113 of 278 (41%) likewise were in the NTG-R group ($p > 0.2$). Statistical analysis of this data generated a sensitivity of 34%, and a specificity of 58%, for NTG-R as a predictor of active CAD. All 95% confidence intervals included 1.0 for positive and negative likelihood ratios for NTG relief of chest pain, indicating no significant difference in predicting active CAD. At the four-month follow-up interview, there was no significant difference in NTG-R and NTG-NR groups in terms of myocardial infarction, revascularization, or death. The authors conclude that relief of chest pain with NTG does not correlate with active CAD in the ED setting.

COMMENTARY BY MICHAEL W. FELZ, MD

Sensitivity and specificity were quite low in predicting CAD, and likelihood ratios indicated that NTG-R (and NTG-NR) are not useful diagnostic signs for confirmation (or exclusion) of myocardial ischemia or infarction. Furthermore, NTG-R did not accurately predict subsequent cardiac events over a four-month follow-up period.

I am uncertain about the applicability of this data. The findings challenge commonly held practice standards for patients whose chest pain resolves with NTG in the ED, office, or hospital. The NTG-R sequence indicates to me either ischemic heart disease or, perhaps, esophageal spasm. Lack of response to NTG, however, speaks against CAD, at least in patients whose ECGs do not suggest acute infarction. In such patients, I search hard for chest wall tenderness reproducing symptomatology, or treat presumptively for GERD. Almost undoubtedly, the presence of ST depression plus chest pain, with reversal of both by NTG administration, reliably still predicts the presence of CAD; in this population, only 13% had abnormal ECGs during pain. ❖

What's the Critical Threshold in Time for Administering Antibiotics in CAP?

ABSTRACT & COMMENTARY

Source: Silber SH, et al. Early administration of antibiotics does not shorten time to clinical stability in patients with moderate to severe community-acquired pneumonia. *Chest* 2003;24:1798-1804.

The objective of this prospective observational study was to determine if there was a significant difference in time to clinical stability (TCS) between patients with moderate-to-severe community-acquired pneumonia (CAP) who received their antibiotics within four hours and those who received antibiotics after four hours. Study patients were adults admitted to the hospital from the emergency department (ED) with moderate-to-severe CAP as defined by a previously described classification system using a pneumonia severity index.[1] TCS was defined as the first day that six parameters were met, including data about vital signs, oxygen saturation, and ability to eat.[2] Patients who did not receive antibiotics consistent with standard guidelines were excluded.

Study groups were defined as those patients who received their first dose of antibiotics within four hours (group 1); within 4-8 hours (group 2); and more than eight hours (group 3) from triage. Of the 409 patients who achieved clinical stability during their hospital stay, the mean time to receiving antibiotics was 2.19 hours in group 1 (222 patients), 5.59 hours in group 2 (136 patients), and 13.07 hours in group 3 (51 patients). The mean TCS was 3.19 (SD 4.27) days in group 1, 3.16 (SD 4.48) days in group 2, and 3.29 (SD 4.31) days in group 3, with no statistically significant differences between the study groups. Secondary outcomes compared study groups for differences in mean length of stay, mean pneumonia severity index, and mortality — and no significant differences were found. The authors concluded that antibiotics administered within four hours do not reduce TCS in adult patients with moderate to severe CAP.

COMMENTARY BY STEPHANIE B. ABBUHL, MD, FACEP

These findings are not surprising, given that the study design was observational and there was no control of other factors that undoubtedly are important in determining TCS, such as length of illness before presentation to the ED, comorbid conditions, immunocompromise, nutritional status, use of oral antibiotics before presentation, and others.

These results are somewhat conflicting with those from other studies that have shown a correlation between outcome measures, such as reducing mortality and length of stay, and timeliness of antibiotics in CAP.[3,4] In one study, antibiotics administered within eight hours were shown to reduce 30-day mortality in patients at least 65 years old. It is difficult to sort out just how critical the time to antibiotics is in CAP and what standard we should be held to. No one would dispute that sooner is better. However, picking a number of hours from triage-to-antibiotic to use as a quality indicator has significant implications and must be evidence-based and of proven benefit. The Center for Medicare and Medicaid Services now specifies a four-hour goal as a quality measure, revised from their previous target of eight hours. Given the conflicting data on this complex issue, and the fact that some of the studies that were the basis of the recommendation were significantly flawed, it seems that a four-hour standard for all patients should be critically reviewed. Further studies are needed to explore the possibility that only certain subgroups of patients may benefit from a more aggressive triage-to-antibiotic effort. Then we could justify focusing our limited resources on this goal while we divert our resources from other goals that may be equally as important. ❖

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Special Feature

Electrocardiographic ST Segment Depression

By William J. Brady, MD, FACEP, FAAEM

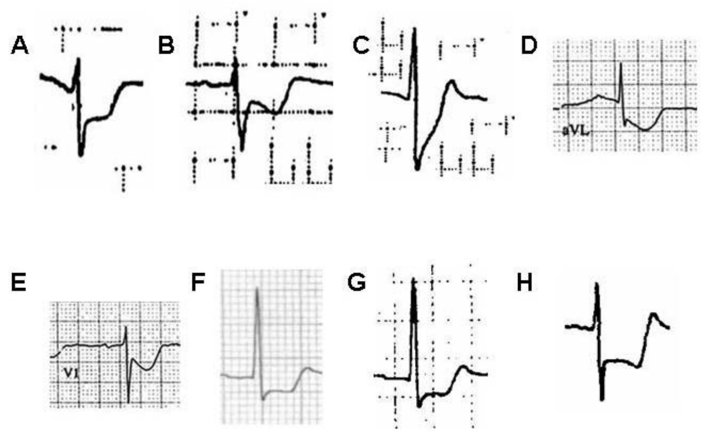
Introduction

ST SEGMENT DEPRESSION (STD) IS AN IMPORTANT FINDing on the electrocardiogram (ECG) of the chest pain patient; in fact, this electrocardiographic finding is seen in approximately 30% of ED chest pain patients.[1] While ST segment changes (both elevation and depression) are associated with an acute coronary syndrome (ACS), numerous other clinical entities manifest ST segment depression. For example, in an ED population, the following electrocardiographic diagnoses were associated with STD: ACS, 26%; left ventricular hypertrophy (LVH), 43%; bundle branch block (BBB), 21%; ventricular paced rhythm (VPR), 5%; and other patterns, 5%.[1] Appropriate management partially is dependent upon differentiating these various causes of STD on the ECG.

Acute Coronary Syndrome

In the ACS patient, STD may be a presenting abnormality on the ECG resulting from one of several ischemic syndromes, including myocardial ischemia

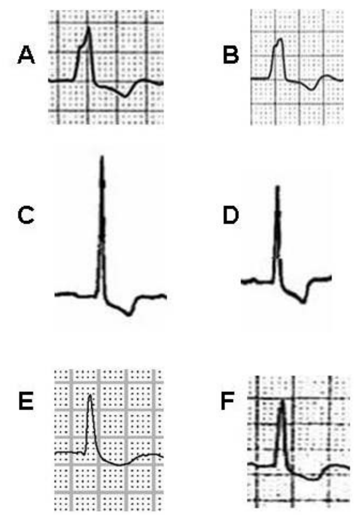
Figure 1. ACS-related STD



A. Non-AMI myocardial ischemia—horizontal. **B.** Non-AMI myocardial ischemia—down-sloping. **C.** Non-AMI myocardial ischemia—up-sloping. **D**. STD in lead aVL as an early indication of inferior wall AMI. **E.** STD in lead V_1 due to posterior wall AMI. **F.** STD in lead V_1 due to posterior wall AMI. **G.** Non-STD AMI. **H.** Reciprocal STD.

Key: AMI—acute myocardial infarction; ACS—acute coronary syndrome; STD—ST segment depression.

Figure 2. Non-ACS Causes of STD



A. Left bundle-branch block; **B.** Left bundle-branch block; **C.** Left ventricular hypertrophy; **D.** Left ventricular hypertrophy; **E.** Digoxin effect; **F.** Digoxin effect.

(without infarction), posterior wall AMI, non-ST segment elevation AMI, or reciprocal ST segment change in the setting of AMI. Considering non-AMI ACS-related STD (i.e., myocardial ischemia), a combination of two electrocardiographic diagnostic criteria traditionally has been required to diagnose myocardial ischemia—at least 1 mm depression at the J point, with horizontal or down-sloping ST segment morphology.[2] Such STD frequently is widespread, located simultaneously in both an inferior and anterior distribution; it does not necessarily localize to a particular anatomic segment. The morphology of the depressed ST segment may be of value in assessing the probability of a non-AMI ACS event. Extensive experience from cardiac stress testing shows that the actual configuration of the STD influences the specificity of this finding, with a down-sloping segment more specific for the diagnosis of ischemia than horizontal depression.[3] In a review of ED chest pain patients, the presence of STD of any morphology was not predictive of ACS. If the clinician applies several simple rules to this electrocardiographic analysis, STD morphology is much more helpful. Certain electrocardiographic patterns with abnormal intraventricular conduction have STD as a "normal" finding, including LVH, BBB, and VPR; if these patterns are removed from this analysis, then the presence of STD is highly associated with ACS. Furthermore, the morphology of the depression is also helpful with horizontal morphology highly suggestive of ACS.[1]

Posterior wall AMI, from the perspective of the standard 12-lead ECG, will manifest STD in the right to mid-precordial leads. The majority of such acute infarcts will present with co-existing acute inferior wall infarction; isolated posterior wall AMIs, however, do occur.[4] Electrocardiographic abnormalities suggestive of a posterior wall AMI include the following (in leads V_1, V_2, or V_3): horizontal STD with tall, upright T waves; a tall, wide R wave; and an R/S wave ratio greater than 1.0 in lead V_2.[5,6] As such, STD in this distribution can represent either non-AMI ischemic change or posterior AMI. Boden et al noted that in patients presenting with STD in leads V_1-V_3, approximately half were found to have had a posterior wall infarction.[7] The use of posterior leads will help distinguish between these two clinical presentations—the presence of ST segment elevation greater than 1 mm in the posterior leads V_8 and V_9 confirms the presence of posterior wall AMI.

Patients with non-ST segment elevation infarction may have transient and nonspecific findings, such as STD or T wave abnormalities in any of the leads of the 12-lead ECG. STD and inverted or biphasic T waves characteristically are seen. Differentiating non-ST segment elevation myocardial infarction from non-AMI ACS using only the ECG is difficult, if not impossible; serum marker analysis is required to rule in myocardial infarction in this particular presentation.

Reciprocal ST segment depression, also referred to as reciprocal change, is defined as STD in leads that are separate from leads demonstrating ST segment elevation. It must be stressed that reciprocal STD can only exist when co-existing ST segment elevation is noted. Further, STD seen in patients with abnormal intraventricular conduction is not included in the definition of reciprocal change. Reciprocal change can be identified in approximately one-third of patients with anterior wall AMI and three-fourths of individuals with inferior AMI. The presence of reciprocal change increases the positive predictive value for a diagnosis of AMI to greater than 90%.[8,9] In the setting of an inferior AMI with early, less pronounced ST segment elevation, the presence of STD in lead aVL alone may suggest early ACS.[10]

Left Bundle-Branch Block

Intraventricular conduction delays, such as with left BBB, will manifest significant ST segment/T wave abnormalities as a "normal" electrocardiographic finding; these changes mimic both acute and chronic ischemic changes. The left BBB pattern is the second most frequently encountered electrocardiographic pattern responsible for non-ischemic ST segment changes in the ED population, accounting for 15% of cases.[11,12] Left BBB may present with significant STD. The rule of "appropriate discordance" states that in left BBB, ST segment/T wave configurations are directed opposite from the major, terminal portion of the QRS complex. As such, leads with predominantly positive QRS complexes (the monophasic R wave) will demonstrate ST segment depression with T wave inversion. In this electrocardiographic presentation, the ST segment usually is down-sloping in form. In fact, Sgarbossa and colleagues reported that in the setting of left BBB, STD—limited to leads V_1, V_2, and/or V_3—is associated with AMI in the appropriate clinical presentation.[13]

Similar electrocardiographic issues are encountered in patients with right BBB and ventricular paced rhythms.

Left Ventricular Hypertrophy

Pressure and volume overload on the left ventricle leads to a sustained repolarization, which produces alterations in the morphology of the ST segment and T wave. In leads with either a leftward or posterior orientation, the sustained repolarization phase results in negativity of both the ST segment and the T wave (seen as STD with T wave inversion).[2] These LVH-related repolarization abnormalities are referred to as a "strain pattern," encountered in approximately 70% of LVH cases, and are characterized by down-sloping STD with inverted T waves in leads with prominent R waves (leads I, aVL, V_5 and V_6).[14] This T wave inversion is asymmetrical, with gradual down-sloping and a rapid return to baseline, often with the terminal portion of the T wave becoming positive (so-called "overshoot").

Digitalis Effect

At therapeutic levels, digitalis produces characteristic electrocardiographic changes, including STD. These changes are referred to as the digitalis effect, which must be distinguished from digitalis toxicity (which manifests primarily as cardiac dysrhythmia). The electrocardiographic manifestations of digoxin effect are as follows: "Scooped" ST segment depression, most prominent in the inferior and lateral precordial leads. Often, it may be impossible to differentiate the STD created by digitalis from ACS. In general, however, digitalis will create a "sagging" ST segment, while ischemia creates the typical horizontal or down-sloping depression. ❖

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Physician CME

20. Based on the study that looked at time-to-antibiotics in patients with moderate to severe community-acquired pneumonia, which of the following statements is *false*?

a. There was no statistically significant difference in the time to clinical stability in the patients who received antibiotics within four hours, between four and eight hours, and those who received antibiotics after eight hours.
b. The mean pneumonia severity index for the three time-to-antibiotic groups was not statistically different.
c. Other factors that may be involved in determining time to clinical stability are nutritional status and ongoing oral antibiotic therapy.
d. The secondary outcomes of mean length of stay and mortality were found to be significantly different in the three time-to-antibiotic groups.

21. New-onset ST segment depression on the ECG is consistent with all of the following *except*:

a. Post-AMI left ventricular aneurysm
b. Posterior wall acute myocardial infarction
c. Myocardial ischemia
d. The reciprocal change seen with ST segment elevation AMI

22. The ST segment depression seen with left ventricular hypertrophy is:

a. usually seen in the right precordial leads.
b. followed by an asymmetrically inverted T wave.
c. uncommon.
d. characteristically up-sloping.

23. In the recent study conducted by Johns Hopkins on response to nitroglycerin as a predictor of coronary artery disease as the cause of chest pain:

a. response to nitroglycerin was a sensitive test for underlying coronary disease.
b. lack of response to nitroglycerin was a specific finding for excluding underlying coronary disease.
c. response to nitroglycerin seemed to neither predict nor exclude underlying coronary disease.
d. most patients received aspirin in the ED.

24. With regard to the NEXUS and Canadian rules for cervical spine imaging after trauma:

a. NEXUS rules include an age threshold.
b. NEXUS rules include ability to rotate the neck.
c. Canadian rules include only men.
d. Canadian rules would x-ray all patients older than age 65 years.

25. The time-to-antibiotics study by Silber et al. did not control for:

a. duration of illness.
b. immunocompromise.
c. co-morbid conditions.
d. All of the above

Answer Key:

20. d	**23. c**
21. a	**24. d**
22. b	**25. d**

CME Objectives

To help physicians:

- Summarize the most recent significant emergency medicine-related studies;
- Discuss up-to-date information on all aspects of emergency medicine, including new drugs, techniques, equipment, trials, studies, books, teaching aids, and other information pertinent to emergency department care; and
- Evaluate the credibility of published data and recommendations.

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Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, *you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

ECG Review

What's Going On?

By Ken Grauer, MD

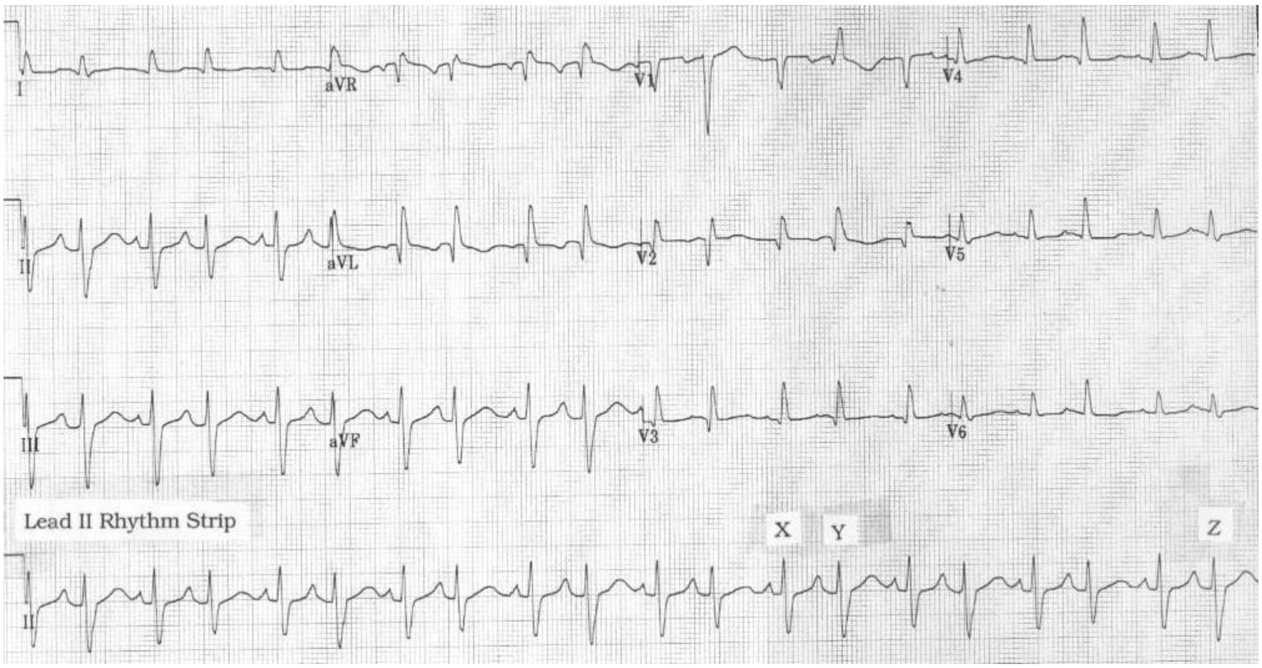

Figure. 12-lead ECG and lead II rhythm strip obtained from an 84-year-old man with acute dyspnea.

Clinical Scenario: The 12-lead ECG and accompanying rhythm strip in the Figure were obtained from an 84-year-old man who presented to the emergency department with acute dyspnea from pneumonia and heart failure. What's going on? Is RBBB (right bundle-branch block) among the findings?

Interpretation: Attention to the simultaneously recorded lead II rhythm strip is essential for understanding the subtleties that are seen in this tracing. The first finding noted is that a bigeminal rhythm is present, characterized by the repetitive "short-long" pattern in the rhythm strip. Of interest is the fact that each of the relatively longer R-R intervals is preceded by a pointed, upright P wave that confirms the underlying sinus rhythm. The beat labeled "X" is one of these sinus conducted impulses. Every other beat (the early occurring impulses) is preceded by an especially peaked T wave compared to the T wave that follows these beats. Each of these early occurring beats is a PAC (premature atrial contraction), with its premature P wave combining with the preceding T wave to produce this peaking. The rhythm is, therefore, atrial bigeminy. The most interesting feature of the rhythm is the subtle variation in QRS morphology among the early occurring complexes. This is the result of varying degrees of aberrant conduction among the PACs. A look at the simultaneously occurring QRS complex in V_1 (above beat Y) and V_6 (above beat Z), in conjunction with the deeper and wider S wave in many of the lead II PACs is consistent with a pattern of RBBB/LAHB (left anterior hemiblock) aberration. Analysis of the sinus conducted beats on this 12-lead ECG is consistent with prior anterolateral infarction (note the QR pattern in leads aVL, and V_2 through V_4), but without acute ST-T wave changes. ❖

In Future Issues:

Vasopressin vs. Epinephrine in Cardiac Arrest

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Evaluation and Management of Blunt and Penetrating Thoracic Trauma

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Trauma to the thoracic cavity is responsible for approximately 10-25% of all trauma-related deaths,[1,2] with the majority of these deaths occurring after arrival at the emergency department (ED). The mortality for isolated chest injury is relatively low (less than 5%); however, with multiple organ system involvement, the mortality approaches 30%.[1]

This article dissects the critical aspects of thoracic trauma and highlights acute care management strategies.

— The Editor

Introduction

Thoracic trauma may be either blunt or penetrating. Compression, deceleration forces, and direct impact are mechanisms that result in the injury patterns seen in thoracic trauma. Unlike blunt trauma, penetrating trauma results from extrinsic violation of the integrity of the thoracic cavity. These injury mechanisms may result in pneumothorax, hemothorax, pulmonary contusion, or injuries to the mediastinal structures. With the proliferation of firearms, penetrating chest injuries are becoming more prevalent, and nearly all result in development of a pneumothorax, with hemothorax occurring in almost 75% of the cases.[1] The use of scoring scales such as injury severity scale, abbreviated injury severity scale, or the thoracic trauma severity score may assist the ED physician and trauma team in algorithmic decision-making. Although controversies abound regarding management of traumatic arrest patients, the decision to perform thoracotomy should be individualized. Moreover, fewer than 10% of blunt chest injuries and 15-30% of penetrating injuries require thoracotomy.[2]

Morbidity and mortality from chest injuries results from the injury pattern itself and physiologic derangements such as hypoxia, hypercarbia, and acidosis. The pathophysiology of these clinical entities arises from inadequate tissue perfusion from hypovolemia (secondary to blood loss), ventilation and perfusion mismatch from pulmonary contusion, or change in intrathoracic pressure from either tension or open pneumothorax.[2]

The presence of severe respiratory distress is associated with a high mortality, with 10% of these patients requiring intubation at the scene or immediately on arrival in the ED.[1] The most common associated risk factors for respiratory distress include multiple rib fractures, shock, pneumothorax, hemopneumothorax, and coma.[1]

Thoracic Injuries

Thoracic cage injuries result from the direct effect of trauma to the chest wall or the thoracic cage. They include flail chest and rib, sternal, thoracic spine, and scapular fractures.

Rib Fractures and Flail Chest. The ribs are the most commonly injured structures in the thorax.[2] This injury should be suspected in patients with localized chest wall pain or contusion over one or more rib segments after trauma. The upper ribs, first through third, are well protected by the bony framework of the upper limb; protective structures include the scapula, humerus, and clavicle, along with their muscular attachments.[2] Fracture of the first, second, or third rib requires a significant amount of force and should lead to high index of suspicion for injuries involving the head, neck, spinal cord, tracheobronchial tree, lungs, or the great vessels.[1,2] The mortality associated with these fractures is approximately 15-30%.[1,2]

The middle ribs, four through nine, are most commonly affected by blunt trauma.[2] A direct force striking anterior-to-posterior on the thoracic cage fractures these ribs along their shafts and tends to drive the ends of the bones into the thoracic cavity. This subsequently leads to intrathoracic injuries such as pneumothorax or lung contusion.[2] Furthermore, fracture of the lower ribs (ribs 10-12) should heighten suspicion for intra-abdominal injuries involving the liver and spleen.

A flail chest results when fractures occur in two or more ribs, or a fracture involves multiple segments of a rib. This results in a lack of continuity between the fractured segments and the rest of the thoracic cage, resulting in the disruption of normal chest wall movement. The morbidity from flail chest is not from the paradoxical chest wall movement produced, but rather from the underlying injury to the lung parenchyma (pulmonary contusion), and persistent splinting that results from severe pain. The pulmonary contusion leads to ventilation perfusion mismatch, leading to hypoxia. *(See Figure 1; see more on pulmonary contusion in the section dedicated to this entity.)*

Elderly and Pediatric Patients. The presence of rib fractures in older individuals carries significant morbidity and mortality. An elderly patient with a rib fracture resulting from blunt chest trauma has twice the morbidity and mortality of younger patients.[4,5] For each additional rib fracture, mortality increases by 19%.[4] Similarly, pediatric patients who present with rib fractures should alert the practitioner to possible child abuse. Although a rib fracture could occur in a pediatric patient who sustains blunt chest trauma, it is very rare. Its presence portends the possibility of concomitant severe injuries. Thus, geriatric and pediatric patients with multiple rib fractures warrant extreme vigilance.

The initial evaluation of a patient with a rib fracture involves palpation for localized pain, crepitus, subcutaneous emphysema, and deformities. In simple rib fractures, chest radiography alone will suffice. The purpose of the study is not to visualize the rib per se, but to evaluate for possible coexisting complications such as pneumothorax. Patients who sustain rib fractures from significant trauma to the thoracic cage, in addition to chest radiography, require computed tomography (CT) of the chest to qualify the extent of intrathoracic organ involvement (e.g., hemopneumothorax or aortic, bronchial, esophageal, cardiac, or diaphragmatic injuries).

Initial therapeutic intervention for an isolated rib fracture focuses on adequate ventilation and analgesia for pain control. Many therapeutic regimens are available for pain management. These modalities include opioid analgesics, transcutaneous electric nerve stimulation, non-steroidal anti-inflammatory drugs, and regional nerve blocks (intercostals, epidural, interpleural, and thoracic paravertebral).[6,7]

In patients with simple flail chest and minimal or no lung contusion, analgesia or intercostal nerve blocks with chest physiotherapy may be adequate. In patients with flail chest and moderate to severe pulmonary contusion, evidence of hypoxia, and signs of shock, mechanical ventilation should be initiated. Mechanical ventilation should be considered in individuals with flail chest and shock, three or more associated injuries, severe head injury, underlying pulmonary disease, fracture of eight or more ribs, or age older than 65 years.[1] Bergeron et al showed that after adjusting for severity, co-morbidity, multiple rib fractures, and age, patients older than 65 years had five times the odds of dying when compared to those younger than 65 years.[8] In patients with flail chest and two or more injuries, early mechani-

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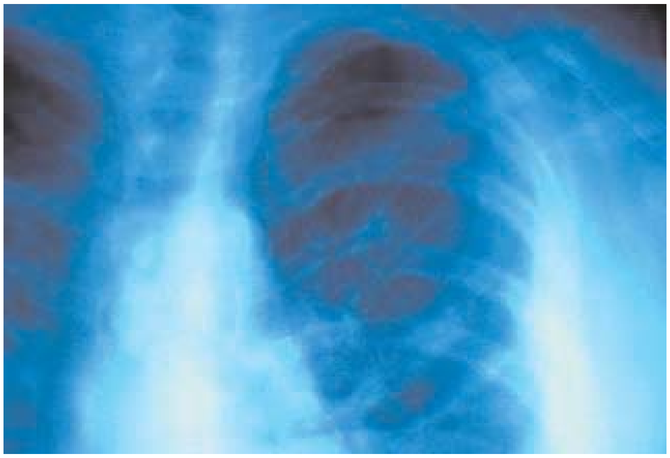
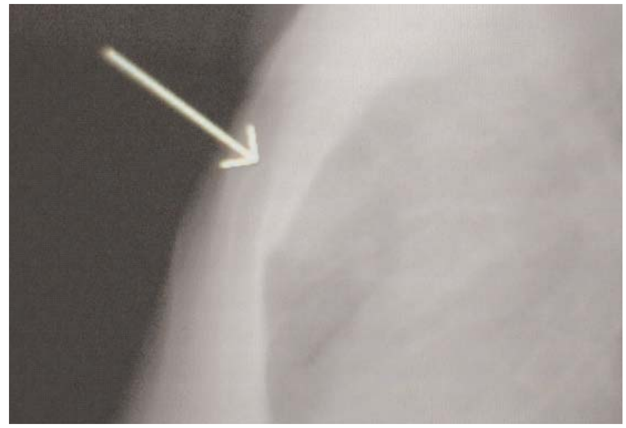
Figure 1. Rib Fractures with Pulmonary Contusion



The above image demonstrates multiple rib fractures and a pulmonary contusion.

Figure 2. Sternal Fracture with Posterior Displacement



A 78-year-old man who sustained a seatbelt injury in a motor vehicle accident. The arrow shows a sternal frature with posterior displacement. (Image courtesy of A. Adewale, MD.)

cal ventilatory support has been shown to reduce the mortality from 69% to 7%.[1]

The disposition of a patient with a rib fracture resulting from blunt chest trauma should be individualized. Patients with isolated rib fractures may be discharged home safely with good analgesia and an incentive spirometer. In patients with multiple rib fractures, admission often is recommended for pain control and to minimize the potential associated morbidities. All geriatric patients with multiple rib fractures require admission for management.[4,5,8]

Sternal Fracture. Sternal fractures commonly result from a direct blow to the chest wall during motor vehicular accidents or from falls, and account for approximately 8% of thoracic injuries.[9] The majority of these injuries result from direct contact with the steering wheel or from seatbelt compression. The presence of a sternal injury should alert practitioners to the possibility of an underlying cardiac or pulmonary injury. However, in the absence of concomitant injuries and other comorbidities, the incidence of morbidity is only about 5.4%,[9] and mortality is approximately 0.7-0.8%.[1,9] Previously, fractures of the sternum were believed to be harbingers of underlying cardiac injury; however, multiple recent studies[9-12] have proven otherwise. It is prudent to remember anecdotal cases of ventricular wall rupture associated with a sternal fracture.[13] *(See Figure 2.)*

The initial diagnostic modalities for sternal fracture are posterior-anterior and lateral chest radiographs. Although some studies have reported better diagnostic yield with sternal ultrasound and bone scintigraphy,[14,15] chest radiography still remains the initial diagnostic aid.

The extent of the evaluation and management should be guided by the severity of the injury and co-morbid factors. Most studies recommend a baseline electrocardiogram (ECG) and creatinine kinase (CK-MB),[1,11,12] with a 2-D echocardiogram reserved for those patients whose clinical presentation warrants the diagnostic test. Althought there are significant variations in institutional management of sternal fractures, the disposition of these patients should be individualized. Most patients with an isolated sternal fracture and no significant underlying medical problems may be discharged safely with appropriate analgesia.[10-12]

Thoracic Spine Injury. The thoracic spine supports the posterior segment of the thoracic cage. The first 10 vertebrae are fixed owing to their articulation with the thoracic cage.[1] Thoracic spine fractures often result from a fall from a height, a motor vehicle collision (especially with ejection), or a motorcycle accident.[16] Motorcycle accidents are the most common mechanisms because of the forced hyperflexion of the thoracic spine.[17,18] Fractures of the thoracic spine usually result from an excessive force, and an anatomically narrow thoracic spinal canal leads to a high incidence of associated neurologic complications.[1,2] Thoracic spinal fractures include anterior wedge compression, burst, chance, and fracture dislocation injuries. *(See Figure 3.)*

The initial evaluation of a patient with a possible spinal injury involves palpation of the spine for step off or subluxation, deformity, or midline tenderness. In most minor blunt chest trauma patients, an ordinary thoracic spine radiograph is adequate for evaluation. However, in severe multi-trauma patients with decreased levels of consciousness, severe alcohol intoxication, or respiratory distress requiring mechanical ventilation, it may be difficult to adequately assess the thoracic spine with routine radiographs. In these patients, CT of the spine has been shown to have better sensitivity, specificity, and negative and positive predictive values when compared to routine thoracic spine radiography.[19-21] Hence, in severe multi-trauma patients, CT scan of the thoracic spine should be the diagnostic study of choice.

The initial therapeutic management of a potential thoracic spinal injury associated with chest trauma involves spinal immobilization until radiographic clearance. However, because of the high incidence of spinal cord involvement associated with thoracic spine injury, the early utility of steroids is very controver-

Figure 3. Thoracic Spinal Fracture

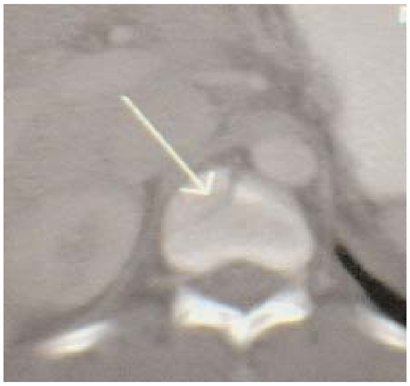
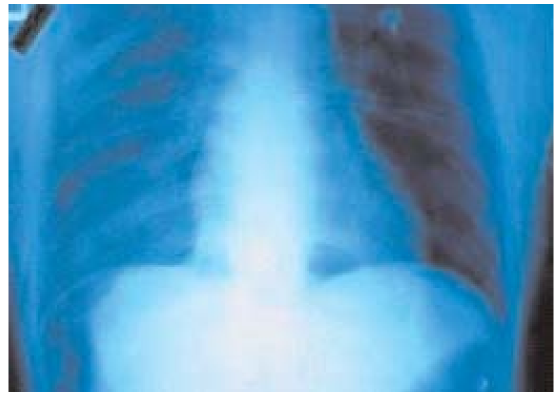


Image courtesy of A. Adewale, MD.

Figure 4. Hemopneumothorax



The chest radiograph above demonstrates a hemopneumothorax in a 49-year-old male involved in a motor vehicle collision.

sial. While some studies support the utilization of corticosteroids,[22,23] others do not consider it as a mandated standard of care, but rather as a treatment option.[24,25] Currently, the National Acute Spinal Cord Injury Studies (NASCIS) advocates the early use of steroids.

Intrathoracic Injuries

Pneumothorax. Pneumothorax results when air enters the potential space between the parietal and visceral pleura. This injury can be caused by either penetrating or blunt trauma.[2] The most common cause of pneumothorax in blunt trauma is a lung laceration with air leak.[2] It is a common complication of chest trauma, and a recognizable cause of preventable death.[26,27] About 6.7% occur without rib fractures, while the incidence increases with number of rib fractures.[28]

For the normal functioning of the pulmonary apparatus, the thorax is completely filled by the lungs, which are held to the chest wall by surface tension between the pleural surfaces. The presence of air in the pleural space leads to eventual collapse of the lungs. This collapse results in a ventilation/perfusion (V/Q) mismatch, because the blood perfusing the non-ventilated area is not oxygenated.[2] This ultimately results in respiratory distress and hypoxia. Early in the course of a pneumothorax or a small pneumothorax, the patient may be asymptomatic. However, as the intrathoracic pressure increases because of air in the pleural space, a simple asymptomatic pneumothorax may become a life-threatening tension pneumothorax.

The diagnosis of pneumothorax often is accomplished by clinical examination and chest radiography. However, in a severely traumatized patient, supine chest radiography may miss a pneumothorax.[29] Adjunctive diagnostic aids, such as ultrasonography and CT scan, have been shown to have both high sensitivity and specificity in diagnosing pneumothorax.[29,30]

The management of simple pneumothorax is expectant. However, when mechanical ventilation is indicated, prophylactic chest tube thoracostomy should be performed to prevent tension pneumothorax. In acutely dyspneic patients with hemodynamic instability after chest trauma, rapid needle decompression followed by tube thoracostomy is the standard of care.

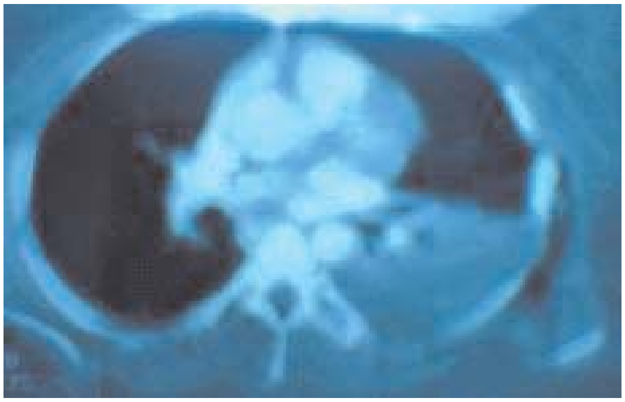
Hemothorax. Hemothorax commonly results from lacerations to the lungs, intercostal vessels, or the internal mammary artery due to either blunt or penetrating trauma.[1,2] Patients with large hemothoraces often are dyspneic, with some degree of respiratory distress because of restricted ventilation. *(See Figure 4.)*

The diagnosis of hemothorax should be made promptly. Although the majority of these injuries are diagnosed during the initial phase of assessment and management of the patient, there have been reported cases of delayed manifestation up to eight hours after initial presentation.[31] The initial diagnostic method is the chest radiograph, and a hemothorax that is large enough to be apparent on chest radiograph should be evacuated. However, in a supine trauma patient, up to 1000 mL of blood could be missed on a radiograph.[1] Studies showing the utility of ultrasound have produced mixed results.[32,33] The use of CT scans has been shown to be the most sensitive and specific for diagnosing and accurately assessing hemothoraces in chest trauma patients.[34-36] CT adequately quantifies the extent of the injury complex and the underlying complications.

The initial management of a hemothorax involves the insertion of a large-caliber chest tube for drainage, or open thoracotomy. In the majority of patients with hemothoraces, tube thoracostomy alone is adequate and is effective in more than 80% of the cases.[37] In the presence of persistent bleeding, videothoracoscopic evaluation and treatment have been shown to be as effective as open thrpracotomy, and minimize the complications that accompany thoracotomy.[37] The presence of more than 1500 mL of blood in the initial chest tube drainage, drainage of more than 200 mL an hour for 2-4 hours, or ongoing transfusion requirements mandate surgical exploration with open thoracotomy.[2] *(See Figure 5.)*

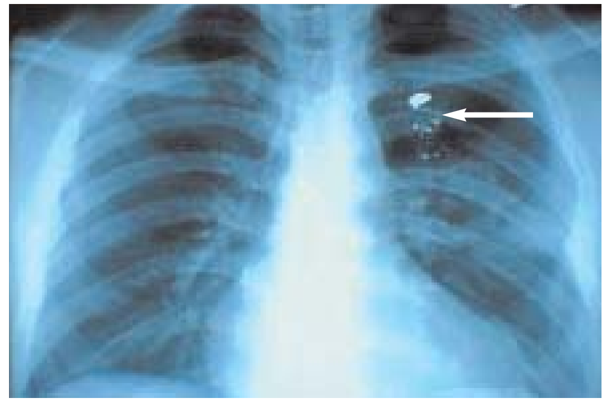
Pulmonary Contusion. Contusion to the lung parenchyma is a significant cause of morbidity and mortality resulting from thoracic trauma. It is the most common cause of lethal chest injury.[2] Usually a result of direct force to the chest wall, the injury results

Figure 5. Hemothorax and Pulmonary Contusion



A CT scan of the chest that demonstrates a hemothorax and pulmonary contusion. (Images courtesy of A. Adewale, MD.)

Figure 6. Rib Fracture from Gunshot Wound, with Pulmonary Contusion



An isolated rib fracture from a gunshot wound to the chest with a pulmonary contusion. The arrow indicates a bullet fragment.

from a coup and countrecoup effect. The transmitted force produces direct damage to the lung parenchyma and associated hemorrhage and edema. In the majority of cases, respiratory failure develops over days rather than instantaneously.[2]

The pathophysiology of pulmonary contusion is poorly understood, and only minimal advances have been made in the past 20 years.[38] The primary injury to the lung results in increased blood flow to the uninjured lung and parenchyma due to reflex decrease in pulmonary vascular resistance. This results in the extravasation of fluid into the alveoli and the interstitial spaces. With aggressive resuscitation, the level of the edema and blood in the lung parenchyma and interstitial spaces increases, thus producing a V/Q mismatch that is manifested as worsening hypoxia, hypercarbia, and acidosis.[1]

The diagnosis of pulmonary contusion is dependent on the extent of the lung parenchyma involved. Initial radiographic manifestations may be minimal or significant, depending on the individual patient. Even so, the chest radiograph should be the initial diagnostic aid, and for the majority of patients will show diffuse opacification of the involved lung parenchyma. *(See Figure 6.)* CT scan of the chest may be used in patients to adequately identify the exact lung segment involved, quantify the contusion volume, and act as a prognosticator of morbidity and mortality.[39,40]

The management of pulmonary contusion depends on the severity, associated injuries, and co-morbid conditions of the patient. The physiologic parameters that determine the severity of a pulmonary contusion in a chest trauma patient include: oxygen saturation less than 90% that is not responding to routine oxygen supplementation (nasal canula, bag-valve-mask, or facemask); PaO_2 less than 65 mmHg; persistent tachypnea or FiO_2/PaO_2 less than 300.[1,2,41]

The primary management goal is to maintain adequate ventilation. The severity of the injury determines the modality of mechanical ventilation utilized. The need for intubation should be individualized, since the majority may be managed non-invasively.[42] In a patient with a contused lung, the plateau airway pressure is increased, while static compliance and $ETCO_2$ is decreased.[43] Independent lung ventilation (ILV) now is a commonly used modality for managing pulmonary contusion, because it is effective in reducing V/Q mismatch, improving oxygenation with a setting of tidal volume (TV) and positive end-expiratory pressures (PEEP) to keep pulmonary plateau pressure below safe thresholds for barotrauma.[42,43] Presently, the use of lung protective ventilatory strategy with low TV and high PEEP has become a standard practice.[43,44] In most severe cases, placing the patient in a decubitus position (involved lung dependent position), use of nitrous oxide (because of its less dense characteristics), utility of pressure-control ventilation with paralysis, and the use of high-frequency oscillator have been shown to improve survival.

Blunt Myocardial Injury. Blunt myocardial injury (BMI) commonly is caused by high-speed motor vehicle collisions, although injuries resulting from low-velocity collisions have been reported.[1] Due to different criteria used in diagnosing BMI, the incidence is difficult to determine. However, the prevalence ranges from 3-56%, depending on the study. The mechanisms of this injury include direct blows, athletic trauma, industrial crush, blasts, rapid deceleration, or falls from a height.[1]

A direct blow to the precordium (such as the chest wall striking the steering wheel in a motor vehicle collision, or the handlebar or a guardrail in a motorcycle collision, or being struck by a high-velocity object such as a baseball) creates a force that compresses the myocardium against the spine. *(See Figure 7.)* Because of the relatively free anterior-posterior movement of the heart, the momentum generated from a rapid deceleration accident maintains the heart in a uniform, straight-line motion, resulting in a direct strike against the internal sternum.[1,2] BMI includes injuries such as cardiac contusion, cardiac concussion (commotio cordis), cardiac chamber rupture, or valvular disruption.

The manifestations of BMI originate from a direct injury to the myocardium that results in sub-endocardial hemorrhage, in turn leading to the formation of localized edema and the mobilization of the inflammatory response system. The resulting inflammatory changes cause a redistribution of coronary flow that may manifest as ischemic type chest pain.[1] The severity of the presentation is contingent upon the underlying coronary artery disease, since transient coronary redistribution can produce a total or near-total occlusion of coronary vessels that already are stenosed. The worsening stenosis may lead to signs of acute coronary syndrome or acute myocardial infarction (AMI).

The clinical manifestations of this injury, in addition to ischemic or ischemic-like chest pain, include rhythm and conduction disturbances (e.g., multiple premature ventricular contractions and premature atrial contractions, atrial fibrillation, ventricular tachycardia, etc.), tachycardia out of proportion to volume loss or pain, right bundle-branch block (RBBB), and heart failure.[1,2] Also, the clinician should be suspicious of BMI in the presence of increased central venous pressure (CVP) and the absence of obvious cause.

Presently, there is no gold standard for diagnosing cardiac contusion. The true diagnosis only can be ascertained through direct visualization of the injured myocardium. Currently utilized diagnostic aids include:

- Chest radiography—The presence of pulmonary contusion or sternal fracture on chest x-ray should lead to a high index of suspicion for BMI;
- ECG—Its utility is controversial. However, recent studies have shown its advantages as a stratification and medical decision-making tool. Velmahos et al show that "the combination of normal ECG and cardiac TnI at admission and 8 hours later rules out the diagnosis of significant blunt cardiac injury."[45]
- Cardiac enzymes—Its utility also is controversial. However, multiple recent studies[46,49-52] are beginning to show its usefulness in determining if a patient has sustained significant BMI. Mori et al showed that "abnormal titers of cardiac TnI suggesting myocardial contusion may be found in more than half of the patients with blunt chest trauma."[46]
- Echocardiography—This is a very good screening and adjunctive test for evaluating BMI. Because of the low yield and lack of specificity of transthoracic echocardiography (TTE), transesophageal echocardiography (TEE) is the modality of choice when BMI is suspected. Chirillo et al showed that TTE "has a low diagnostic yield in severe blunt chest trauma, while transesophageal echocardiography provides accurate diagnosis in a short time at the bedside."[47]
- Magnetic resonance imaging (MRI)—The ability of cardiac MRI to adequately visualize the entire myocardium and the adjoining great vessels might increase its utility in chest trauma patients. Descat et al reported two cases of myocardial contusion diagnosed using MRI.[48]

The management of BMI depends on the clinical manifestations. Although the majority of the cases are benign, some may present with AMI,[53,54] persistent hypotension, or cardiogenic shock that warrants acute intervention. Recent literature proposes the use of ECG and TnI as the initial screening tools. If the initial ECG and TnI (four hours after the injury) are normal in the absence of concurrent injuries, the patient safely may be discharged from the ED.[52] However, if the ECG or the TnI is abnormal, further workup is indicated. The patient should be admitted to a monitored unit with serial cardiac enzymes and a TEE to adequately evaluate the cardiac apparatus. In the absence of abnormalities on TEE and the normalization of cTnI, the patient should be reassessed, and the clinical parameters should determine the disposition.

Figure 7. Blunt Myocardial Injury

A 7-year-old boy who presented with dizziness and chest pain after being struck on the chest by a baseball. (Image courtesy of A. Adewale, MD.)

Commotio Cordis. Commotio cordis, or cardiac concussion, is the most common cause of traumatic death in an athlete.[55-57] It causes sudden or near cardiac death in the absence of structural abnormalities, and results from an object directly striking the chest wall at a phase of ventricular depolarization.[56] Ventricular fibrillation is the most commonly induced arrhythmia. The survival rate is very low when the condition is not recognized. With the popularity of automatic electric defibrillators in public places, the survival from this phenomenon could be improved, since the ventricular fibrillation may respond to rapid defibrillation.

Esophageal Injury. Traumatic esophageal injury is very uncommon.[2,57] When it occurs, injury to the esophagus most commonly results from penetrating trauma; however, it also can result from blunt injury to the lower thoracic cavity. Upper esophageal injury may accompany lower cervical or upper thoracic spine injuries, while distal injuries are rarely caused by blunt chest trauma.[57,58] The mechanism of this injury is the forceful expulsion of gastric contents into the esophagus caused by a severe blow to the lower chest wall or upper abdomen. The resulting increase in intragastric pressure being transmitted to the esophagus results in a linear tear of the lower esophagus. This tear leads to the leakage of gastric contents into the mediastinum.[2]

The clinical presentation of the disease is similar to that of an esophageal tear secondary to persistent, profuse retching. In a severely traumatized patient, the diagnosis initially may be missed, and delayed diagnosis may lead to increased morbidity and mortality.

Esophageal rupture should be suspected in a patient with blunt trauma and one of the following features:

- Left hemothorax or pneumothorax without rib fracture;
- A significant blow to the lower sternum or epigastria with pain out of proportion to the apparent injury;
- Presence of particulate matter in chest tube drainage; or
- Presence of pneumomediastinum.

The diagnosis of esophageal perforation is very challenging. If suspected, a non-ionic contrast esophagogram should be performed.[1] However, this study carries a high false-negative rate, and in patients with a high degree of suspicion for this injury, flexible esophagoscopy in conjunction with esophagogram may increase diagnostic yield.

The management of esophageal perforation is very controversial and is guided by the location of the perforation (hypopharynx, cervical, thoracic, or distal). While some authors propose surgical management for all perforations,[59-63] some propose conservative management based on Cameron's criteria (i.e., minimal signs of sepsis, disruption contained within the mediastinum, drainage of cavity back into the esophagus, and minimal symptoms).[64] According to Rios et al, "Conservative treatment should be considered for patients meeting Cameron's criteria, since their evolution is favorable with low morbidity and mortality."[64]

Despite technical and nutritional advances, the mortality rate for esophageal injuries ranges from 5-25% for those treated within 12 hours, to 25-66% for those treated after 24 hours.[1] Regardless of the management approach, esophageal perforation is a life-threatening condition that requires early diagnosis and management to minimize the morbidity and mortality.

Diaphragmatic Injury. Blunt injury to the diaphragm is uncommon, occurring in about 0.8-8% of hospitalized chest trauma patients.[65] The incidence of the laterality of this injury varies depending on the study. However, most studies report the incidence of left-side injury to be between 60-90%.[1,66-68] This probably is because of the protection provided by the liver on the right and the possible left posterior lateral weakness of the diaphragm.[1,2] Because of the significant contribution of the diaphragm to normal ventilation, injury to this structure may lead to significant respiratory compromise.

This injury often initially is missed unless the defect is large enough to cause acute herniation of the abdominal viscera into the thoracic cavity. In this instance, the chest radiograph will show the gastrium in the thoracic cavity or a coiled nasogastric tube in the thoracic cavity. Smaller defects may cause a delay in diagnosis until the abdominal viscera slowly migrates into the thoracic cavity, causing compression of the adjacent lung leading to either bowel strangulation or tension pneumothorax.[1]

Diaphragmatic injuries create a diagnostic dilemma. The chest radiograph is diagnostic in most cases of large ruptures; however, in the small defects, the injury often is missed or the chest radiograph is misinterpreted as showing an elevated diaphragm, acute gastric dilatation, a loculated pneumohemothorax, or a sub-pulmonary hematoma.[2] The general consensus for the modality of choice for diagnosis is the helical CT; multiple studies have shown helical CT scan with sagittal and coronal reconstruction to be very sensitive in diagnosing diaphragmatic injury.[65,69,70] In one study, sensitivity approaches 84%, with specificity of 77%.[70] The most accurate modality for diagnosis is MRI. MRI is capable of directly acquiring both coronal and sagittal images, and allows the evaluation of the entire diaphragm, and shows the exact site and size of rupture in all cases as reported in one study.[69] However, its usefulness is limited in the acutely traumatized patient. Its utility is beneficial in otherwise stable patients with blunt chest trauma with a high index of suspicion for isolated diaphragmatic injury. With technological advances in the field of thoracic surgery, videothoracoscopic evaluation of the diaphragm in the hand of an experienced surgeon is emerging as a diagnostic modality.[71,72]

The management of diaphragmatic injury mostly is surgical. Most trauma patients with suspected diaphragmatic injury undergoing exploratory laparotomy for any intra-abdominal injury should have the diaphragm evaluated for possible defect. However, with the advances in laparoscopic techniques, thoracolaparoscopic repair is becoming the modality of choice for repair of an isolated diaphragmatic injury.[71] Isolated diaphragmatic injury in the absence of other surgical injuries carries low mortality, and Bergeron et al showed that operative repair can be deferred without appreciable increase in mortality if no other indications mandate surgery.[72]

Blunt Aortic Injury. Blunt traumatic thoracic aortic injury (BAI) is a rare, but very lethal, condition. It often occurs as a result of a decelerating injury from high-speed motor vehicle collision (low-speed in older population) or a fall from a height.[1] The incidence of BAI is about 6.8 per 100,000 motor vehicle occupants, with a steady increase with increasing age.[73] Approximately 80-90% of the patients die at the scene,[1,73] and up to 50% of the remaining patients die within 24 hours if not promptly diagnosed and treated.[1] Of the survivors who make it to the hospital, the ultimate survival rate is lower for patients who are older than 60 years.[73]

Three mechanical factors contribute to aortic rupture. These factors include shearing stress, bending stress, and torsion stress.[1] As deceleration occurs, a gradient is created between the mobile aortic arch and the immobile descending aorta. This gradient places the aortic isthmus under tension, and the resultant shearing stress can lead to rupture or tear opposite the fixation site.[1] Bending stress results from the hyperflexion of the aortic arch produced by the downward traction exerted by the heart, and torsion stress results from anterior posterior compression of the chest, with the heart displaced to the left combined with an intravascular pressure wave transmitted to the aorta.[1] When combined, these three forces produce maximum stress to the inner surface of the aorta at the ligamentum arteriosum, which is the point of greatest fixation.[1]

The clinical presentation of BAI is vague, since specific signs and symptoms often are absent.[2] Most patients with free rupture, as elucidated earlier, die at the scene. However, patients with contained rupture who make it to the hospital may exhibit transient hypotension, dysphagia, hoarseness, or acute dyspnea.[1,74] Although clinical factors (e.g., multiple rib fractures, flail chest,

Table 1. Radiographic Findings Suggestive of Aortic Injury[1,2]

- Widened mediastinum
- Aortic knob obliteration
- Rightward deviation of the trachea
- Obliteration of the aorto-pulmonary window
- Left mainstem bronchus depression
- Rightward deviation of the esophagus
- Paratracheal stripe widening
- Presence of apical cap
- Left hemothorax

Figure 8. Thoracic Aortic Dissection

CT-scan of a 78-year-old female involved in a motor vehicle accident. The arrow shows a thoracic aortic dissection. (Image courtesy of A. Adewale, MD.)

pulse deficits, or hoarseness without laryngeal injury) may be suggestive of BAI, about one-third of patients with this injury have no external signs of thoracic injury on initial physical examination.[1]

The diagnosis of BAI requires a high index of suspicion. A chest radiograph remains the most appropriate initial screening modality, with a negative predictive value approaching 95%.[75] The supine chest x-ray may not show the classic findings; however, the presence of a widened mediastinum mandates further investigation. On chest x-ray, the presence of a widened mediastinum and a hemothorax in a patient with transient hypotension should increase the suspicion of aortic injury.[74] *(See Table 1.)*

The radiographic algorithm utilized after the initial chest x-ray should be dictated by the patient's clinical condition. Although aortography still is the recognized gold standard, fast spiral helical CT scan has emerged as a diagnostic study that potentially will supplant aortography.[76] Multiple studies[77-80] have shown spiral CT angiography to have a sensitivity of 96-100%, specificity of 98-100%, and accuracy of 99-100%. In the hemodynamically stable patient, contrast-enhanced helical CT has a critical role in the exclusion of BAI and prevents unnecessary thoracic aortography.[80]

The role of TEE in the evaluation of BAI has been well documented.[75,77] Since the specificity and sensitivity of TEE are similar to those of helical CT, the indication for TEE is for the hemodynamically unstable patient with suspected BAI.[75,77] Although TEE and CT have similar diagnostic accuracy, TEE allows for the diagnosis of associated cardiac injuries, and is more sensitive than CT for the identification of intimal or medial lesions of the thoracic aorta.[77] *(See Figure 8.)*

While aortography still is the recognized gold standard for diagnosing BAI, in the case of equivocal aortographic findings, intravascular ultrasound (IVUS), with sensitivity approaching 92%, and specificity of 100%, could be used as an adjunctive diagnostic aid.[81] The role of MRI in the evaluation of trauma patients remains indeterminate. Despite sensitivity and specificity approaching 100% for aortic injury, MRI's utility in trauma patients is not feasible logistically because of the requirements of a metal-free environment and the long period of time that the patient must lie in isolation in a quiet room.[1]

The management of BAI typically is surgical following the initial resuscitation using the American College of Surgeons Advanced Trauma Life Support protocol. Emergent surgical intervention is the accepted standard of care. However, in some selected cases, such as patients deemed to be at high operative risk because of associated injuries or pre-existing medical conditions, or in stable patients for whom conditions for surgery are not ideal, delayed surgical intervention may be warranted.[1] The surgical approach utilized is institutionally dependent.

Tracheobronchial Injury. Injury to the tracheobronchial area occurs very rarely and often is associated with other injuries.[84] Although this injury potentially is fatal, it often is overlooked on initial assessment.[2] The reported mortality for tracheobronchial injury (TBI) has fallen from 36% in the 1950s to less than 9% in the 1970s.[85] The mechanism of this injury results from the effect of rapid deceleration on a relatively mobile bronchial structure and its fixed proximal segments.[1] The majority of these injuries occur within 2 cm of the carina, or at the origin of the lobar bronchi.[1,86]

The common clinical presentations of TBI are signs of respiratory distress (dyspnea, stridor, or hemoptysis), subcutaneous emphysema, and sternal tenderness.[1,87,88] The presence of pneumomediastinum, pneumothorax, widened mediastinum, or deep cervical emphysema on chest radiograph may suggest TBI.[1,2,84,88]

The diagnosis of TBI requires a high index of suspicion. The morbidity and mortality increases if not diagnosed early.[87] The initial screening modality is the chest radiograph, which often demonstrates the signs suggestive of TBI (as described in the paragraph above). According to one study, the CT scan of the chest demonstrated similar findings to the chest radiograph, but failed to confirm the diagnosis.[89] The presence of the "fallen lung" sign (a collapsed lung in a dependent position hanging on the hilum by its vascular attachment) on CT scan is highly suggestive of TBI.[84] Tracheobronchoscopy is the definitive diagnostic modality of choice.[1,2,84,88] On bronchoscopy, the injury pattern visualized is a transverse tear in the main bronchus involved or a disruption at the origin of an upper lung bronchus, while the trachea shows a vertical tear in the membranous portion near its attachment to the tracheal cartilages.[1]

Since most patients with TBI present with pneumothorax or tension pneumothorax, initial needle decompression with subsequent thoracostomy tube placement is required. The presence of persistent air leak with proper chest tube placement and drainage is highly suggestive of TBI until proven otherwise.[1,2,89]

The presence of persistent hypoxia despite intubation and chest tube placement mandates the use of temporizing opposite main stem bronchus intubation to provide adequate oxygenation,[2] and also minimizes the effect of the ventilation and perfusion mismatch. In some instances, blind endotracheal intubation may be difficult, owing to anatomic distortion from pharyngeal injuries, paratracheal hematoma, or the TBI itself. For these patients, immediate operative intervention is required.[2] However, in the more stable patients, acute surgical intervention could be delayed until inflammation and edema resolve.[2,90] The definitive surgical treatment involve the reestablishment of the anatomic continuity of the tracheobronchial tree if the lesion affects more than one-third of the circumference.[86]

Finally, independent of mechanism and anatomic location of injury, delay in diagnosis is the single most important factor influencing outcome.[91]

Penetrating Chest Injury. Penetrating chest injuries (PCIs) are more common in urban medical centers. Most of these injuries involve firearms and knives. Injuries involving the cardiac, vascular, and transmediastinal structures are the most lethal, with prehospital mortality approaching 86% for cardiac injuries, and 92% for extrapericardial vasculatures.[92] Of patients who make it to the hospital alive, only about 5-15% require thoracotomy.[1] Of those who survive to the hospital, the mortality for those with cardiac injury is 21.9%, and 1.5% for those without cardiac injury.[93] Survival rates from stab wounds generally are much higher than those from gunshot wounds.[1,93]

The injury pattern in PCI may include extensive lung laceration *(See Figure 9),* a sucking chest wound, or mediastinal traversing injuries. A sucking chest wound acts as a one-way valve that allows air to enter the pleural cavity during inspiration and none to leave during expiration. This eventually leads to an expanding or tension pneumothorax.[1] In the prehospital setting, it is imperative that the wound be covered with occlusive dressings on only three sides. This allows air to escape the pleural cavity during expiration and, thus, prevents development of a tension pneumothorax. On arrival to the ED, the wound should be examined and covered completely with occlusive dressing, with simultaneous insertion of a chest tube at a site other than the initial injury location.[1,2]

Wounds that traverse the mediastinum may involve the great vessels, heart, tracheobronchial tree, or the esophagus.[2] The overall mortality for these injuries approaches 20%.[2] The evaluation of these injuries in hemodynamically stable patients can be performed non-operatively.[94] Trauma ultrasound may be used for assessing pericardial tamponade, spiral CT for evaluating transmediastinal injuries, and organ-specific studies (e.g., esophagogram, aortography, bronchoscopy, thoracolaparoscopic evacuation of retained hemothorax, or repair of diaphragmatic injury) are minimally invasive management techniques for stable PCI patients.[94]

Figure 9. Penetrating Chest Injury

This penetrating chest injury was sustained by a motorcyclist who struck an embankment in a high-speed collision (Image courtesy of A. Adewale, MD.).

In hemodynamically unstable patients, there should be a high index of suspicion for exsanguinating thoracic hemorrhage, tension pneumothorax, or pericardial tamponade.[1,2] In this situation, a bilateral chest tube thoracostomy is warranted to decompress possible hemopneumothorax and document volume of blood in chest tube drainage.[2] The performance of ED thoracotomy is mainly for evacuation of pericardial blood, direct control of exsanguinating hemorrhage, open cardiac massage, or cross-clamping the descending aorta to slow blood loss below the diaphragm and increase perfusion to the brain and heart.[1,2] With a bleak survival report for emergent ED thoracotomy, each facility should develop a uniform guideline for performance of this procedure. *(See* Trauma Reports *2003;4:1-12 for a thorough discussion of ED thoracotomy.)* Recent studies[95,96] show that the presence of signs of life on arrival to the hospital, in addition to the mechanism of injury and location of major injury, should be the determinants of the indications for emergent thoracotomy.

Conclusion

Thoracic cavity trauma carries significant morbidity and mortality because of the vital structures it involves. With technologic advances, most of these injuries now can be evaluated with minimally invasive diagnostic aids. The evaluation and management of injuries involving this cavity should be individualized, with special consideration for the pediatric and geriatric population.

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CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

CE/CME Questions

1. The most commonly injured structure(s) in the thorax is/are:
   A. the sternum.
   B. the clavicle.
   C. the ribs.
   D. the lungs.

2. The indication for surgical exploration or videothoroscopic evaluation of hemothorax in a chest trauma patient is:
   A. initial chest tube drainage of 500 mL.
   B. the drainage of 100 mL/hr of blood for 2-4 hrs.
   C. spontaneous resolution of bleeding.
   D. initial chest tube drainage of more than 1500 mL.

3. The "fallen lung sign" is suggestive of which injury?
   A. Tension pneumothorax
   B. Esophageal tear
   C. Tracheobronchial injury
   D. Hemopneumothorax

4. One of the physiologic parameters that determine the severity of pulmonary contusion is:
   A. initial oxygen saturation less than 95%.
   B. initial PaO_2 less than 65 mmHg.
   C. initial PaO_2/FiO_2 less than 400.
   D. initial respiratory rate of 42.

5. The gold standard for diagnosing cardiac contusion is:
   A. electrocardiogram.
   B. cardiac enzymes.
   C. echocardiogram (TEE or TTE).
   D. There is no gold standard for diagnosing cardiac contusion.

6. The most commonly induced arrythmia in commotio cordis (cardiac concussion) is:
   A. ventricular tachycardia.
   B. asystole.
   C. ventricular fibrillation.
   D. supraventricular tachycardia.

7. Esophaphageal rupture should be suspected in blunt trauma patients with which of the following features?
   A. Multiple rib fractures
   B. Left hemothorax or pneumothorax without rib fractures
   C. Lack of particulate matter in chest tube drainage
   D. Persistent retching

8. A coiled nasogastric tube in the thoracic cavity is indicative of which of the following injuries?
   A. Blunt aortic injury
   B. Tracheobronchial injury
   C. Diaphragmatic injury
   D. Pulmonary contusion

9. Regarding the diagnosis of blunt aortic injury, which of the following statements is *not* true?
   A. Aortography is still the gold standard.
   B. CT scan angiography is as specific and sensitive as aortography.
   C. Magnetic resonance imaging is the most specific and has replaced aortography as the gold standard.
   D. Transesophageal echocardiography is quick, sensitive, specific, and enables evaluation of the cardiac apparatus.

10. Which of the following statements regarding transmediastinal injuries (stab or gunshot) is true?
    A. A gunshot wound to the heart has a better survival rate.
    B. A stab wound to the heart has a better survival rate.
    C. Survival rates for stab wounds and gunshot wounds to the chest are the same.

CME Objectives

Upon completing this program, the participants will be able to:

a.) Recognize or increase index of suspicion for thoracic trauma;

b.) Identify how to correctly and quickly stabilize and manage thoracic trauma;

c.) Employ various diagnostic modalities for thoracic trauma; and

d.) Recognize both likely and rare complications that may occur.

Answer Key

1. C	6. C
2. D	7. B
3. C	8. C
4. B	9. C
5. D	10. B

In Future Issues: **Trauma Ultrasound**