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INSIDE

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Etomidate Again Linked to Adrenal Suppression

By Nathan Shapiro, MD, MPH,
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Dr. Shapiro is on the speaker's bureaus of Eli Lilly and Edwards Life Sciences and is a researcher for Biosite.

Source: Malerba G, et al. Risk factors of relative adrenocortical deficiency in intensive care patients needing mechanical ventilation. *Intensive Care Med* 2005:31:388-392.

ETOMIDATE IS A COMMONLY USED ANESTHETIC INDUCTION agent in the emergency department. Its predictable dosing and favorable hemodynamic profile make it an appropriately popular choice to use as part of a rapid sequence induction cocktail. However, emerging literature also attributes increased mortality risk to adrenal suppression, especially in patients with sepsis. To complete the story, there also are studies that show steroid supplementation in critically ill septic patients with adrenal suppression may reverse these effects and provide a mortality benefit in these patients. This investigation reports on the risk of adrenal suppression in recently intubated patients in intensive care units.

The specific goal of this study was "to study the factors associated with relative adrenocortical deficiency in mechanically ventilated, critically ill patients." This single center, prospective, observational study looked at patients who were likely to need mechanical ventilation for at least 24 hours. Exclusion criteria were prior mechanical ventilation for more than 6 hours, a stay in another intensive care unit, or the need for steroids. Patients underwent a corticotropin stimulation test (i.e., cortisol measurement at baseline and 60 minutes after receiving 250 micrograms of corticotropin) and were considered nonresponders (i.e., adrenal suppression) when the absolute increase in cortisol level was less than 90 micrograms/liter.

The 62 patients in the study had a mean age of 63 years, mixed etiologies of illness (e.g., cardiogenic shock, coma, sepsis), and were fairly ill according to their severity of illness scores. On a univariate

basis, the risk factors for adrenal suppression as defined by an inadequate response to a corticotropin stimulation test were male sex, worse severity of illness scores, lower mean arterial blood pressure, lower creatinine clearance, vasopressor agent use, and a single bolus of etomidate. Thus, if the patient was male, more critically ill, or had received etomidate, there was an unadjusted association with adrenal suppression at 24 hours. Finally, on a multivariate analysis, a single bolus of etomidate was an independent risk factor (OR 12.2, 95% CI 3.0 50) for adrenal suppression while female gender was protective (OR 0.13, CI 0.03 - 0.57). There was not enough statistical power to comment on mortality risk of etomidate, but 70.4% of nonresponders died compared with 31.4% of responders ($p = 0.005$).

The authors concluded that a single dose of etomidate in critically ill patients could be a major factor in reduced adrenal function at 24 hours. They cautioned that the implications of these findings call for a larger investigation to look at the effect on outcomes that this adrenal suppression may have. Essentially, they called for a re-appraisal of the risk-benefit ratio for etomidate in the critically ill patient. ❖

COMMENTARY

This is a provocative single center investigation in a relatively limited number of patients from a heterogeneous patient population. The study found an association between etomidate use and failure to respond to a corticotropin stimulation test at 24 hours. That being said, there is ample opportunity for confounding where more critically ill (i.e., hypotensive) patients may have been preferentially given etomidate due to its negligible effects on hemodynamic stability. There also are a number of other opportunities for unmeasured confounding in this nonrandomized study. Finally, there is an association with adrenal suppression, but the effect on mortality was not analyzed.

So, is this study definitive evidence that we should not use etomidate for induction anymore? What about use in septic patients? To keep things in perspective, this is another piece of circumstantial evidence to add to the data in this field. We know that adrenal suppression is associated with worse outcomes, but we do not really know why. We know that septic and other critically ill patients often are adrenally suppressed, but we do not know how. Now, we think that etomidate is associated with short-term adrenal suppression, but we do not know the importance of this fact. We also have to weigh the benefit of rapid airway control using an agent with a stable hemodynamic profile, where there are few other alternatives with this property. We do know of the dangers of poor perfusion and profound hypotension; in some cases this needs to be avoided, even at the risk of short-term adrenal suppression. There are three approaches until more data are available: 1) don't react too quickly—wait for outcomes-based studies; 2) avoid etomidate and select an alternative agent when safe to do so; and 3) initiate steroid supplementation in patients who likely will be affected by adrenal suppression, followed by corticotropin stimulation testing, and halt steroids after adrenal suppression is resolved. Although perhaps more questions are raised than answered in this study, it is important to be aware of the potential downstream effects of the use of etomidate as an induction agent in emergent airway treatment, and to be cognizant of these effects until we have more definitive studies to guide our choices in the future.

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PCI: Better Late than Never!

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Dr. Perron reports no relationships with companies having ties to the field of study covered by this CME program.

Source: Schomig A, et al. Mechanical reperfusion in patients with acute myocardial infarction presenting more than 12 hours from symptom onset. *JAMA* 2005;293:2865-2872.

PERCUTANEOUS CORONARY INTERVENTION (PCI) HAS developed into the clear choice as the reperfusion strategy in acute myocardial infarction (MI) when weighed against fibrinolysis. When used within 12 hours of symptom onset, PCI has demonstrated increased myocardial salvage, preservation of left ventricular function, and overall improved survival as compared with intravenous fibrinolytic strategies. Further, studies that looked at intravenous fibrinolysis beyond 12 hours from symptom onset have shown no benefit to this modality; in fact, results showed a trend toward harm. What generally is unknown is whether there is any benefit to a late PCI reperfusion strategy in the subset of acute MI patients presenting more than 12 hours after symptom onset. Observational studies have hinted that there may be benefit, but prospective data are lacking. These late presenters (i.e., patients presenting more than 12 hours from symptom onset) have been reported to constitute between 8% and 32% of all patients presenting with acute MI.

The purpose of this prospective, open-label, randomized, controlled study carried out in Italy, Germany, and Austria was to determine if there was benefit to PCI performed for acute MI in patients who presented more than 12 hours after symptom onset. As fibrinolysis was not an option for those patients not randomized to receive PCI, these patients were randomized to conventional conservative treatment. The primary outcome measure was prospectively defined as left ventricular (LV) infarct size. Secondary outcome measures included death, recurrent MI, or stroke at 30 days.

All patients received standard care including aspirin and an initial dose of glycoprotein IIb/IIIa inhibitor, and heparin. All patients also received beta-blockers, as well as angiotensin converting enzyme inhibitors and statins as indicated. All patients were admitted to telemetry for at least 48 hours. Patients in the control group continued taking heparin for 24 hours, and ultimately had a symptom-limited stress test prior to discharge. Patients assigned to PCI were taken to the cardiac catheterization laboratory immediately. If indicated, these patients had angioplasty and/or stenting; these patients received abciximab following their procedures.

The study enrolled 365 patients between May 2001 and December 2004; 182 were randomized to the PCI arm and 183 randomized to the control group. The results showed significantly smaller infarct size in the PCI group (8%, IQR 2%-16%) as compared with controls (13%, IQR 3%-27%). There was a trend toward improved secondary outcome measures in the PCI group (4.4%) as compared with controls (6.6%), but this did not reach statistical significance (relative risk 0.67, $p = 0.37$).

The authors of the study concluded that PCI— even when used at 12 or more hours after onset of symptoms—results in smaller infarct size in acute MI patients. ❖

COMMENTARY

The fundamental question being asked by this multicenter, multinational trial is: Regarding PCI for AMI, is late better than never? The answer would seem to be a cautious "yes" at least for infarct size. Now the astute clinician likely (and rightly) will ask first whether the 5% reduction in LV infarct size is clinically significant. The answer again seems to be yes, based upon other studies. Other authors have reported that even a 3% difference in infarct size results in reduced 30-day mortality.[1]

The reader might then question why there was not a significant difference in 30-day composite outcomes in this study. While the relative risk of reaching one of the three composite endpoints was reduced by 33% in the treatment arm, this factor did not reach statistical significance. The authors (and an accompanying editorial)[2] pointed out that this study was never powered to find such a difference. Far more patients would need to be enrolled to demonstrate (or disprove) this.

I don't think that this relatively small study is enough to make PCI an absolute requirement (i.e., a Class I indication) for all comers with acute MI regardless of time from symptom onset. I do think it should allow the clinician to feel comfortable pushing the interventional cardiologist toward doing the procedure when the only limitation identified is time from symptom onset.

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Head Positioning in Patients With Acute Ischemic Stroke: Does it Matter?

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Dr. Abbuhl reports no relationships with companies having ties to the field of study covered by this CME program.

Source: Wojner-Alexander AW, et al. Heads down: Flat positioning improves blood flow velocity in acute ischemic stroke. *Neurology* 2005;64:1354-1355.

THE PURPOSE OF THIS STUDY WAS TO DETERMINE IF middle cerebral artery flow velocity could be augmented by simple head-of-the-bed (HOB) positioning during acute stroke while avoiding arterial flow compromise as might occur with an increase in intracranial pressure (ICP). Transcranial Doppler (TCD) was used to measure mean flow velocity (MFV) and indirectly estimate flow changes in association with HOB positioning. In a repeated measures design, the effect of 30°, 15°, and 0° HOB elevation on residual arterial flow signals was measured in 20 awake patients within the first 24 hours of acute ischemic stroke symptoms. Patients with hemorrhagic or posterior circulation stroke were excluded from the study, and no patient received thrombolytic therapy prior to, or at the time of, TCD measurements.

The mean age of the patients was 60 ± 15 years with 14 men and 6 women. Median National Institute of Health Stroke Scale score at the time of initial TCD was 14 points (range 3 to 24 points). All patients had middle cerebral artery MFV improvement with lowering the head position. The mean difference in MFV from HOB 30° to HOB 0° was -8.2 cm/s ±8.2 (95% CI -12.0 to -4.28). On average, the middle cerebral artery MFV increased 20% (12% from 30° to 15° and 8% from 15° to 0°; $p \leq 0.025$). No significant difference was measured in mean arterial pressure and heart rate for any HOB position change throughout the intervention. Pulsatility index, a measure of resistance to flow, remained unchanged at each HOB position. Immediate neurologic improvement occurred in three patients after lowering the head position. ❖

COMMENTARY

This study found that decreasing HOB elevation from the 30° to the 0° position resulted on average in a 20% increase in the mean velocity of the residual arterial blood flow at the affected artery following acute ischemic stroke. At the same time, the pulsatility index remained unchanged within low resistance parameters suggesting no increase in resistance due to a potential increase in ICP. While this study is limited by its small size, the results suggested that zero-degree head positioning may improve residual flow in the affected middle cerebral artery. One explanation is that simple gravitational force may augment the local perfusion pressure gradients. It also is possible that the velocity increase may be due to improved collateral flow.

It should be cautioned, however, that TCD MFV is only a surrogate marker of cerebral blood flow. There is no direct way of measuring blood flow volume with TCD, and the velocity measurement is limited to the horizontal portion of the middle cerebral artery. Ultimately, all these measurements are only surrogate markers for the most important outcome: clinical functional status. A large study comparing these simple HOB maneuvers with clinical outcomes would be helpful in guiding both prehospital and emergency department management. Certainly, not all patients can tolerate zero-degree positioning due to poor cardiopulmonary status and/or the risk of pulmonary aspiration, and patients with hemorrhagic stroke might do worse. The optimal beneficial time frame for this position and the question of any further benefit with the Trendelenburg position are unknown.

Yet, until we have more data, this study—along with a prior paper[1] that directly measured ICP and middle cerebral artery flow velocity in patients with large hemispheric stroke—suggested that cerebral perfusion pressure may be maximized by simple zero-degree HOB positioning in patients with acute ischemic stroke. It will be interesting to see if the next set of American Heart Association/American Stroke Association guidelines recommend a flat position if tolerated.

References

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Special Feature

Heat Stroke

By William Brady, MD, FACEP, FAAEM,
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Heat illness includes several distinct syndromes, presenting across a range of severity; two distinct syndromes include heat exhaustion and heat stroke. Heat exhaustion, the least concerning of these illnesses, is seen in patients who have been exposed excessively to high ambient temperatures for prolonged time periods. Heat exhaustion is seen in persons working in high heat conditions as well as recreational athletes. Heat exhaustion is associated with a favorable outcome, usually with removal of the patient from the high heat environment. Heat stroke results from an inability to control one's body temperature, essentially, a failure of the body's thermoregulatory mechanism. Heat stroke, if not rapidly diagnosed and appropriately managed, can result in death as well as significant long-term disability.

Etiology

Two different varieties of heat stroke are seen in clinical medicine: classical and exertional.[1] Classical heat stroke is seen in the older patient with significant long-term medical problems; the exertional subtype is encountered in the younger, healthier population after intense physical activity.[2] Classical heat stroke tends to occur in epidemic fashion during periods of high heat index, a function of both ambient temperature and humidity (e.g., the summer heat wave). Affected patients are usually older with poor conditioning, have significant comorbidity, and have an absence of cooling systems. Psychiatric syndromes with related medications (e.g., anticholinergic drugs, including neuroleptic medications) greatly increase the risk of heat stroke in affected individuals.

Exertional heat stroke commonly occurs in younger, healthy individuals when endogenous heat production overwhelms the thermoregulatory mechanisms. This homeostatic failure typically occurs when the patient is physically active in a warm, humid setting. Military personnel, athletes, and industrial workers are typical patients. Exertional heat stroke occurs sporadically throughout the summer.

Numerous factors—both endogenous and exogenous—can predispose an individual to heat stroke, including patient characteristics (e.g., advanced age, lack of acclimatization, physical activity, obesity, or poor conditioning), medical issues (e.g., substance abuse, dehydration, cardiovascular disease, and history of heat illness), concurrent medication use (e.g., antipsychotic, antidepressant, and cardioactive agents) and environmental factors (e.g., high humidity, high ambient temperature, or inadequate ventilation).[3,4]

Pathophysiology

The pathophysiology of heat stroke is linked to methods of bodily heat dissipation. Heat can be removed from the body by convection, radiation, conduction, and/or evaporation. Convection is the transfer of heat to a circulating fluid or gas while radiation is the transfer of heat to a portion of the environment that is not directly in contact with the object. Conduction is direct transfer of heat from the body to another object; evaporation is the transfer of heat by vaporization of liquid. Convection, radiation, and conduction require a thermal gradient between the transferring body and the environment; therefore, as the ambient temperature approaches 99º F, the mechanisms of heat loss become less effective; evaporation then assumes the major responsibility for heat dissipation.[3] Furthermore, as humidity levels approach 90%, evaporation becomes much less effective in dissipating heat.[1] Thus, a high heat index (both elevated ambient temperature and humidity) is a particularly likely risk scenario for heat stroke.

Clinical Presentation

Heat stroke usually presents as an acute illness; the initial manifestations are altered mentation, high temperature, and ineffective diaphoresis. A minority of patients, however, can present with a transient, prodromal syndrome similar to heat exhaustion. These patients will manifest nausea, weakness, headache, and confusion; such patients then progress onto the typical heat stroke. The classic triad of symptoms includes central nervous system (CNS) dysfunction, hyperthermia, and hypohydrosis; as is true with most classic triads, this complete constellation of findings is encountered less often. CNS dysfunction is a universal finding in heat stroke victims; in fact, the clinical onset of heat stroke often is marked by either a loss of or alteration in consciousness. Any form of CNS dysfunction can be encountered, yet confusion progressing to extreme irritability followed by unresponsiveness is the norm. Convulsions (both focal and generalized), papilledema, muscular rigidity, and dystonic movements are other manifestations of neurologic dysfunction.

Hyperpyrexia is an important finding in the patient with heat stroke. It is difficult, however, to pinpoint an exact temperature at which heat stroke occurs. Prehospital cooling as well as the potential inaccuracies of peripherally obtained body temperatures further complicate the notation of specific temperatures as a criterion for the diagnosis. In fact, a lower temperature does not preclude the diagnosis.

The third classic finding in the heat stroke patient is

hot, dry skin. Strict adherence to this criterion will lead to many missed cases; dry skin is a late phenomenon of heat stroke. In fact, most patients with heat stroke will present with continued, profuse sweating. The lack of anhydrosis does not rule out the possibility of heat stroke.[1-4]

Other organ systems are affected. The cardiovascular system invariably is involved with manifestation ranging from compensatory sinus tachycardia with nonspecific ST-segment changes on the electrocardiogram, to overt cardiac failure with acute coronary syndrome. Hepatic damage is an almost constant feature of heat stroke and is manifested by elevations in the transaminases levels, with jaundice appearing 24-72 hours after heat stroke onset. Hepatic synthetic dysfunction is manifested by coagulation abnormalities. Acute renal failure complicates 30% of exertional heat stroke cases but is much less common in the classical heat stroke patient. Rhabdomyolysis and hypotension are additional stresses placed on the renal system that may precipitate renal failure.[1-3]

Treatment and Outcome

Heat stroke is a medical emergency; irreversible damage may occur quickly if cooling measures are not started immediately. As abnormalities in the ABCs are addressed, cooling measures must be initiated. Several cooling methods are available, including cool/ice water immersion and evaporative techniques. The most commonly used method—ice water tub immersion—results in a rapid reduction of core body temperature with a cooling rate of up to 16º C/min. Cool water—rather than ice water—immersion also may be used and generally is less uncomfortable with a lower rate of cooling.[1,2,4] The evaporative technique uses a combination of aerosolized water and fanning applied to the disrobed patient. Evaporative cooling methods may be employed easily in the ED using aerosolized tepid tap water and a fan over a disrobed patient; this approach likely represents the most appropriate method of core temperature reduction in the heat stroke patient. Other methods of cooling have been used to reduce core temperature (e.g., ice pack application, cooling blankets, peritoneal dialysis, and cardiopulmonary bypass); none of these methods has been proven to be superior to the immersion and evaporative approaches. As cooling occurs, the body temperature must be monitored. When body temperature reaches 38.5º C, cooling measures should be modified to avoid hypothermic overshoot.[2]

Other management issues include the support and maintenance of an adequate airway with appropriate oxygenation and ventilation; endotracheal intubation with mechanical ventilation often is required due to aspiration and convulsion management. Cardiovascular support most often is accomplished with intravenous fluids; it is uncommon to require vasopressor infusions. Seizures are frequent and are managed in standard also will address agitation and control shivering associated with cooling; older therapies with chlorpromazine should be avoided due to the potential for increased heat production. Surveillance for, and treatment of, rhabdomyolysis and the associated renal failure with appropriate laboratory testing and intravenous fluids is encouraged. Antipyretic agents play no role in the management of heat stroke unless complicated by an infectious syndrome after hospitalization.

The outcome of heat stroke is related to numerous factors including the degree of hyperthermia, the duration of hyperthermia, and co-morbidities. Investigators suggested that mortality in heat stroke is inversely related to the rapidity of effective cooling of the patient; the absolute peak of body temperature elevation is not so important prognostically.[5] Duration of hyperthermia is another major factor in the outcome of the heat stroke patient; morbidity and mortality markedly increases with prolonged periods of elevated body temperature.[6] ❖

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Physician CME Questions

17. Which of the following is a recognized effective cooling technique in classic heat stroke?

A. Intravenous olanzapine by continuous infusion
B. High-dose acetaminophen
C. Evaporative cooling with fans and tepid water
D. Iced peritoneal lavage

18. Which of the following factors was found to be a risk factor for adrenal suppression in critically ill, mechanically ventilated patients?

A. Myocardial infarction
B. A single dose of etomidate
C. Obesity
D. Hyperglycemia

19. Patients presenting with acute myocardial infarction more than 12 hours after symptom onset who received percutaneous coronary intervention have been demonstrated to have a reduction in _____ versus controls.
A. Infarct size
B. Post-infarction dysrhythmia rate
C. Mortality rate
D. Left ventricular aneurysm formation

20. In the study looking at mean flow velocity (MFV) and head-of-the-bed (HOB) positioning in acute ischemic stroke:
A. all patients had posterior inferior cerebellar artery MFV improvement with lowering the head-of-the-bed position.
B. patients with hemorrhagic stroke and posterior circulation stroke were included in the study.
C. transcranial Doppler was used to MFV.
D. functional clinical scores improved significantly in patients with HOB at 0° compared with those at 30°.

21. The outcome of patients presenting to the ED with heat stroke is related to all of the following issues, *except:*
A. Duration of hyperthermia
B. Magnitude of hyperthermia
C. Etiology of hyperthermia
D. Time to definitive care including cooling

22. The diagnosis of heat stroke is supported by all of the following conditions, *except*:
A. Elevated body temperture
B. Altered mental status
C. Pancreatitis
D. Inadequate diaphoresis

Answers:
17. C
18. B
19. A
20. C
21. C
22. C

CME Objectives

To help physicians:
- Summarize the most recent significant emergency medicine-related studies;
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Class IV Symptoms Post-MI

By Ken Grauer, MD, Professor and Associate Director, Family Practice Residency Program, College of Medicine, Universityof Florida, Gainesville.
Dr. Grauer is the sole proprietor of KG/EKG Press.

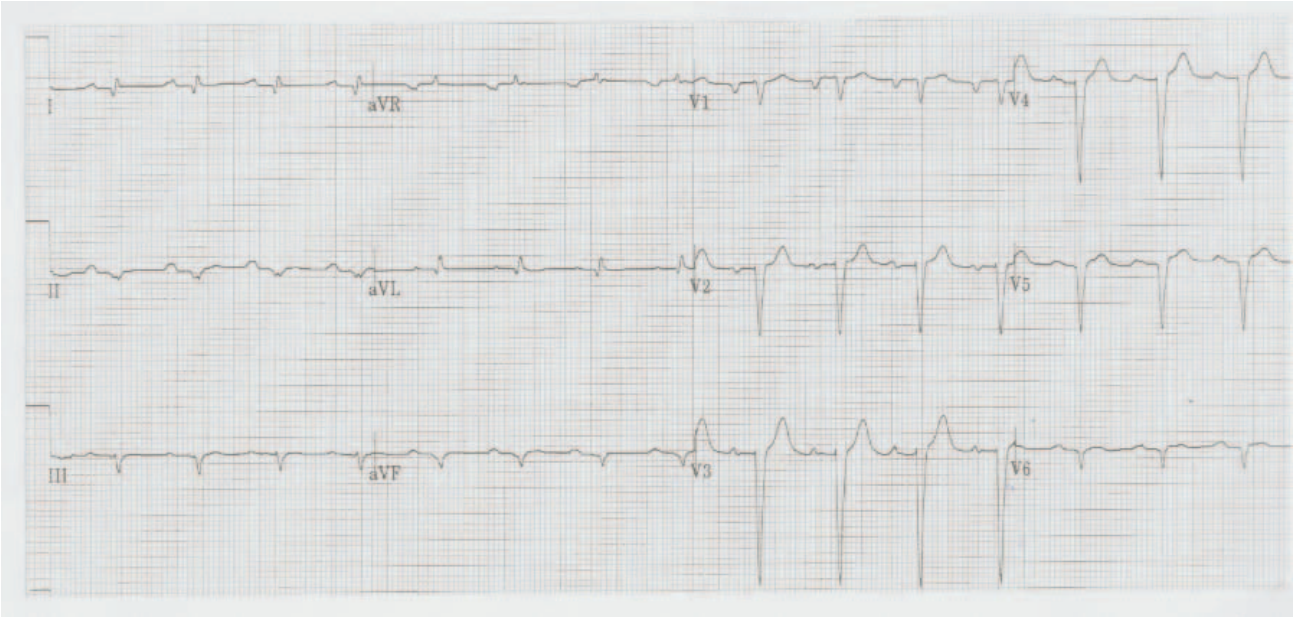

Figure. 12-lead ECG recorded from a 60-year-old man with coronary artery disease, heart failure, and hypotension.

The electrocardiogram (ECG) in the Figure was obtained from a 60-year-old man with a history of two prior myocardial infarctions. His most recent infarction was 8 months earlier, and was remarkable for an extended course complicated by severe heart failure. His major symptoms since that time have been shortness of breath, progressively increasing fatigue on minimal exertion, and hypotension. He has not had angina. How would you interpret his most recent ECG in view of this clinical picture? What types of interventions might be most likely to prolong his survival?

Interpretation: The ECG shows sinus rhythm at a heart rate of just under 100 beats/minute. QRS amplitude is reduced in the limb leads. All intervals are normal. A deep negative component to the P wave in lead V1 is consistent with left atrial enlargement (LAE), but there is no other evidence of chamber enlargement. ST-T waves do not show acute changes. Instead, the most remarkable finding on this tracing is that this patient has essentially "Q'ed out." Other than tiny initial r waves in leads III, V2, V3, and V4—the QRS complex is predominately (or totally) negative in virtually all leads, except I and aVL. Even in these two leads, Q waves are present and R-wave amplitude is significantly less than usually is seen in lateral leads. The overall ECG picture is consistent with the clinical history suggesting severe (probably end-stage) heart failure from an ischemic cardiomyopathy. Loss of R-wave amplitude in conjunction with diffuse Q/QS waves and/or deep rS complexes is most probably the result of extensive myocardial damage, which has led to persistent hypotension with Class IV symptoms of low cardiac output and congestive heart failure. Obviously, optimizing medical management is essential. However, interventions, such as cardiac transplantation, use of a ventricular assist device, and/or placement of an implantable cardioverter-defibrillator (ICD), may be more likely to increase long-term survival in this unfortunate patient who is otherwise at high risk of developing cardiogenic shock or ventricular fibrillation as a terminal event. ❖

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Trauma complicates 6% to 7% of all pregnancies.[1] Emergency department (ED) physicians and nurses will find themselves frequently caring for pregnant women who have suffered a variety of traumatic injuries, ranging from minor to life-threatening. These cases will be complicated by a variety of issues, including pregnancy-associated physiologic changes, imaging/radiation risks, limitations in medication use, and fetal monitoring/tocometry. Emergency health care providers must be comfortable with the unique issues surrounding the evaluation and treatment of pregnant trauma patients and approach each pregnant woman in a systematic fashion.

— The Editor

Trauma in Pregnancy

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Peer Reviewer: **Jeffrey F. Linzer, Sr., MD, FAAP, FACEP,** Assistant Professor of Pediatrics and Emergency Medicine, Emory University, Atlanta.

Epidemiology

Trauma is the leading cause of death among women of childbearing age. Trauma requiring hospitalization complicates 0.4% of pregnancies. Overall maternal morbidity and mortality after trauma are not increased by the gravid state; however, pregnancy alters the injury patterns.[2] Motor vehicle crashes are the leading cause of trauma in pregnancy, followed by physical violence and falls.[2-4] Maternal morbidity from trauma during pregnancy is related to an increased propensity to develop disseminated intravascular coagulation (DIC) due to placental factors, an increased rate of fracture complications, and increased abdominopelvic blood flow, leading to increased blood loss and development of retroperitoneal bleeding.[5] In addition, the presence or risk of preterm labor and the need for fetal monitoring are justifications for increased maternal hospitalization. Motor vehicle crashes have been identified as the most common cause of preterm labor requiring admission.[3]

Trauma is the most common cause of nonobstetric maternal death in pregnancy. (Maternal death is defined as death during pregnancy or within 42 to 90 days after delivery.[6-10]) In some areas of the United States, injuries are the most common cause of

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maternal death.[6] In recent studies comparing rates and causes of maternal death, the number of deaths from obstetric causes is decreasing.[7,8] In contrast, deaths from injuries (including unintentional and suicidal) account for more than half of nonobstetric deaths, and the number resulting from homicide is increasing.[9]

Fetal Morbidity and Mortality. Even mildly injured (Injury Severity Score [ISS] 1-8) pregnant women are at risk for placental abruption, and their fetuses are at risk for hypoxia, respiratory distress syndrome, and death.[11] Less commonly reported fetal injuries sustained in motor vehicle collisions include brain trauma and spinal fracture.[12,13] When the fetal head is engaged in the pelvis or maternal pelvic fractures are present, the likelihood of fetal skull and brain injuries is increased.[14] The fetus is at greatest risk of suffering injury or death during the time immediately following trauma. Rare case reports document severe intrauterine fetal neurologic injury undetected during trauma evaluation.[15]

Maternal death is the most common cause of trauma-associated fetal death. Following maternal trauma, fetal death rates have been reported between 4% and 61%, depending upon length of follow-up and injury severity.[16] Motor vehicle crashes are the predominant traumatic mechanism causing fetal death, distantly followed by penetrating trauma (caused by firearms) and falls. The prevalence of fetal injury and loss is difficult to determine, because of underreporting of these events in vital statistics reports and injury surveillance systems.[17]

Multiple studies have attempted to identify maternal factors predictive of poor fetal outcome. Fetal death has been associated with an increased maternal ISS and abnormal maternal physiology on presentation, including shock. Severe truncal injuries also have been associated with increased fetal loss.[2] In one large multi-institutional study, fetal deaths occurred in 50% of patients with an ISS more than 25.[18] Fetal loss also was associated with maternal shock (e.g., systolic blood pressure < 90) or a fetal heart rate less than 110 bpm. The most frequent fetal complication was premature labor; 5.9% of patients in this study delivered prematurely, 95% delivering viable neonates. Placental abruption occurred in 3.5% of patients and was associated with a 54% mortality. Other complications included premature rupture of membranes and uterine perforation or rupture. Overall, 72.3% of the 372 patients studied did not have fetal complications.[18] In another study, patients with placental abruption had a much lower ISS (< 2). In this population, placental abruption occurred in 6.8% of patients. An ISS of 4 was associated with fetal death.[19] Together, these studies illustrate that ISS alone should not be used to determine risk of placental abruption or fetal death; women with relatively minor injuries can sustain adverse pregnancy outcomes.

Physiology

The emergency care provider should be familiar with the physiologic changes of pregnancy (*Table 1*). These changes cause a pregnant woman's response to injury to be different from what is expected in a nongravid female. Trauma resuscitation of a pregnant female without consideration of these physiologic changes may contribute to high fetal death rates.[20]

Cardiovascular. Plasma volume is increased significantly during pregnancy. By the end of the first trimester, plasma volume has increased 40% to 50%.[21] Red blood cell mass also increases, but to a lesser degree, resulting in a dilutional anemia. This increased blood volume provides maternal protection against blood loss during delivery. However, an injured pregnant woman can lose up to 2000 mL of blood without developing any signs of hemodynamic instability.[21] Trauma care providers must be vigilant in their search for possible bleeding, even when the patient exhibits normal vital signs.

Blood pressure and heart rate also are affected by pregnancy. Baseline blood pressure decreases, secondary to progesterone-related vascular relaxation. During the second trimester, baseline systolic and diastolic blood pressures are lowered by 5 to 15 mmHg, making interpretation of blood pressure difficult in the presence of hemodynamic compromise. Baseline heart rate during pregnancy increases 15 bpm, complicating evaluation for occult hemorrhage. There are multiple case reports of pregnant women with presumably normal vital signs after trauma, who suffered significant internal injuries, including uterine rupture and placental abruption. Because of these factors, the care provider must have a high level of suspicion for injury.

Elevated blood pressure has different implications for pregnant and nonpregnant trauma patients, as it may be an indicator of pre-eclampsia and eclampsia. (The clinical triad that heralds eclampsia comprises seizures, elevated blood pressure, and proteinuria.) Pre-eclampsia also has been associated with transient blindness.[22] If a woman in the third trimester presents to the ED

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Table 1. Normal Physiologic Changes of Pregnancy Relevant to Trauma Care

PARAMETER	CHANGE	IMPLICATION
Maternal blood volume	Increased	Attenuated initial response to hemorrhage
Cardiac output	Increased	Increased metabolic demands
Uterine enlargement	Enlarged	Propensity for supine hypotension from aortocaval compression
Functional residual volume	Decreased	Hypoxemia from atelectasis more likely
Gastrointestinal motility	Decreased	Greater risk of aspiration
Minute ventilation	Increased	Compensated respiratory alkalosis, diminished buffering capacity

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with altered mental status after being involved in a motor vehicle collision or after being found after a presumed or witnessed seizure, eclampsia should be considered as a cause of the incident. Appropriate laboratory studies include complete blood count, blood urea nitrogen, creatinine, liver function tests, and a coagulation profile. Eclampsia is treated with a magnesium sulfate drip. Definitive treatment is delivery of the fetus, as placental factors contribute to the disease. An obstetrician should be involved in the decision-making process when managing a trauma patient with potential eclampsia.

Cardiac output increases 40% during pregnancy. During the third trimester (> 24 weeks), supine positioning of the patient will cause significant aortocaval compression, resulting in a 25% reduction in cardiac output and dropping systolic blood pressure by as much as 30 mmHg.[23] This condition can be alleviated by placing the patient in a left lateral tilt position, which can be accomplished by placing a towel, bag of saline, or wedge under the backboard or under the patient if she has no evidence of spinal injury.[21] Uterine blood flow at term approaches 600 mL per minute. Rapid exsangination can occur from uterine bleeding.[24]

Pulmonary. Respiratory changes in pregnancy also affect the patient's response to trauma. Minute ventilation increases due to increased tidal volume, resulting in a compensated respiratory alkalosis with increased excretion of bicarbonate. Pregnant patients tolerate acidosis poorly because they have little buffering reserve capacity. Functional residual volume also is decreased, increasing the patient's propensity to develop hypoxia. Atelectasis contributes to hypoxia in the supine pregnant patient as a result of increased abdominal girth. Because of these factors, after becoming apneic, a pregnant woman will become hypoxic more rapidly than a nonpregnant woman.

Pregnant patients are at increased risk of pulmonary embolism, because of changes in coagulation factors and venous stasis. In patients who report syncope, shortness of breath, or chest pain prior to the traumatic event, pulmonary embolism should be considered as a possible factor leading to the injury.

Other. Gastrointestinal motility is decreased by circulating progesterone.[24] Thus, many pregnant women have increased gastric reflux and are at an increased risk for aspiration.

Glomerular filtration rate and creatinine clearance are increased in pregnancy. These increases lower baseline serum creatinine levels (< 0.9 mg/dL) and blood urea nitrogen levels (<15 mg/dL), and modest increases may represent significantly impaired renal function.[20]

Pregnancy induces a hypercoagulable state by increased hepatic production of clotting factors, increasing maternal risk of thromboembolism.[24] An increase in the baseline level of fibrinogen in a pregnant patient is important when evaluating for the presence of DIC.[21]

Initial Management

Prehospital. Emergency medical services personnel are usually the first care providers who have contact with pregnant trauma patients. The most important initial factor in fetal outcome is the status of the mother; therefore, maternal resuscitation should come before attempts to assess the fetus. If the patient is of childbearing age but does not appear pregnant, she should be asked if she could be pregnant. If the estimated fetal age is more than 20 weeks, a hospital with a neonatal intensive care unit is preferred, but may not be available.[23] Fetal gestational age more than 20 weeks, may be estimated by a uterine fundal height between the umbilicus and xiphoid.[25] After the patient is placed on the backboard, the board should be placed in the left lateral decubitus position using blocks, towels, or a liter bag of saline. When supine, pregnant patients develop hypoxia easily and should receive supplemental oxygen. Two large-bore intravenous lines should be placed and aggressive crystalloid fluid resuscitation begun. En route to the trauma facility, all treatment should be guided by maternal status, not by suspected fetal distress.

Primary Survey. In the trauma center's receiving unit, initial management of the pregnant patient should focus on the maternal primary survey (evaluation of the airway, breathing, circulation, disability, and exposure or environment).[26] All women capable of childbearing should be tested for pregnancy. Supplemental oxygen should be given to achieve a hemoglobin saturation level greater than 90%.[1] If there is airway occlusion or danger of not protecting the airway adequately, rapid sequence intubation should be performed. Special considerations for intubation include physiologic and pharmacological issues. Induction and narcotic medications cross the placenta; paralytic agents are larger molecules and do not cross the placenta.[27] Although no consensus has been published regarding the selection of induction agents during pregnancy, etomidate and succinylcholine commonly are used. Success also has been reported without compli-

cations using ketamine and succinylcholine.[28] The individual performing the intubation must be prepared for rapid desaturation resulting from decreased functional reserve capacity, despite adequate preoxygenation. During the intubation attempt, the airway mucosa may be edematous and friable. Every attempt should be made to decrease aspiration risk by applying cricoid pressure and immediately placing a gastric tube, because of decreased gastric motility and increased aspiration risk. If a chest tube is required, it should be placed one to two interspaces higher than usual, owing to elevation of the diaphragm by the gravid uterus.[21] Prior to the circulatory evaluation, the patient should be placed in the left lateral decubitus position to prevent aortocaval compression, while maintaining appropriate spinal immobilization. Crystalloid (lactated Ringer's solution or normal saline) resuscitation should be given as a 3:1 blood loss replacement.[1]

Secondary Survey. The secondary survey should include evaluation of fetal heart rate. Fetal heart rate can be heard by Doppler at 10-14 weeks and with a conventional stethoscope at 20 weeks' gestation. A heart rate slower than 120 bpm or faster than 160 bpm should raise suspicion of fetal distress.[29] If the fetus is viable (> 23 weeks), continuous monitoring should begin at this time.[23]

In addition to the routine portions of the secondary trauma survey, several aspects of the physical examination apply specifically to pregnancy. First, in an unresponsive patient, or one from whom a history cannot be obtained, gestational age must be determined. The umbilicus marks the fundal height that correlates with 20 weeks' gestation. Gestational age beyond 20 weeks' can be estimated by measuring the distance in centimeters from the symphysis pubis to the uterine fundus. Next, the uterus itself should be palpated; it should be palpable but soft. Fetal parts may be palpable as small protusions but should not have discreet form. A firm uterus (with resistance like that of a basketball) is indicative of contraction or tetany. If a discreet uterus cannot be palpated in a woman known to be pregnant, uterine rupture should be considered, especially if individual fetal parts are palpable through the abdominal wall.

The perineal area should be assessed for leakage of fluid (clear or green stained with meconium), bleeding, prolapsed umbilical cord, or fetal parts. If there is leakage but it is not known whether the fluid is urine or amniotic fluid, amniotic fluid placed on a slide, allowed to dry, and then viewed by microscope will exhibit a classic ferning pattern. In addition, amniotic fluid placed on nitrazine paper will turn the paper blue.

Depending upon availability, optimally an obstetrician should perform the internal vaginal examination. In patients with vaginal bleeding, a speculum or bimanual examination should not be performed, because of the possibility of placenta previa (placenta covering the cervical os) and the risk of severe hemorrhage. The Apt test, described later in this article, should be considered to determine the source of any vaginal bleeding. In cases of fetal hemorrhage, exsanguination occurs rapidly, and the time from onset of fetal vaginal bleeding and fetal death is only 1 or 2 minutes. In patients without vaginal bleeding, a careful sterile speculum examination may be performed to assess cervical dilation, to identify or exclude internal trauma, and to look for fluid or amniotic membranes in the vaginal vault or for fetal presenting parts. A sterile bimanual examination should be avoided in preterm patients with leakage of fluid, because the procedure may increase the risk of fetal infectious complications.

Routine examination findings may be altered in pregnant trauma patients. Peritoneal signs may be absent in a pregnant woman despite significant intraperitoneal hemorrhage, owing to stretching of abdominal musculature making the peritoneum less sensitive to irritation.[30] In addition, many pregnant patients have low back pain caused by increased forces on the joints, which may present as paraspinal muscle spasm.[31]

History. The history of the trauma should be obtained simultaneously with the secondary survey. In addition to routine questions about the mechanism and cause of injury, loss of consciousness, location of pain, medical history, and allergies, questions specific to pregnancy should be asked. Information regarding gestation and parity should be obtained.

If the patient has delivered in the past, the mode of delivery (cesarean section or vaginal delivery) should be determined. In addition, any complications of pregnancy (current or previous) should be discussed. If the patient knows her Rh status and received RhoGAM (Ortho-Clinical Diagnostics) during the current pregnancy, this should be documented.

Laboratory Studies. Routine laboratory studies generally should be obtained, including complete blood count, basic metabolic panel, hepatic function studies, coagulation studies, toxicology screen, urinalysis, and type and screen.[25,32] In addition, the fetal DEX test, or Kleihauer-Betke test, should be considered. This quantitative test assesses for fetal blood cells in the maternal circulation. The results can be used to determine the required dose of RhoGAM. In addition, this test frequently is used to monitor patients for ongoing fetal-maternal hemorrhage during prolonged fetal monitoring after trauma.

If a patient is experiencing vaginal bleeding after trauma, it is important to determine whether the bleeding is fetal or maternal in origin. Total fetal blood circulation is very small; thus, if fetal blood is being lost, exsanguination can be rapid with minimal blood loss. Causes of fetal blood loss include umbilical cord avulsion and laceration of a vessel. Maternal vaginal bleeding can be secondary to placenta previa (placental tissue over the cervical os). A placental abruption can present with vaginal bleeding, but usually the blood is trapped behind the placenta. In either case, rapid diagnosis and definitive treatment are necessary to preserve fetal viability.

At the bedside, the Apt test can be performed to distinguish between maternal and fetal sources of vaginal bleeding. To perform this test, blood is placed on a 4 x 4-inch gauze and sodium hydroxide is applied. If the blood changes to dark brown, it is maternal blood, which oxidizes; blood that remains red is fetal.[33] In both cases, prompt obstetric evaluation is necessary.

Diagnostic Testing

Diagnostic testing in trauma patients routinely includes ultrasound evaluation, plain radiographs, and computed tomography (CT) imaging. Care providers must attempt to limit radiation in pregnant patients, and all patients who are able to give consent should be aware of radiation risks prior to undergoing any imaging. In addition, pregnancy may make the films from some of these modalities more difficult to interpret.

Ultrasound. Ultrasound is a valuable tool in the evaluation of nonpregnant and pregnant trauma patients. In emergency ultrasound guidelines published by the American College of Emergency Physicians,[34] evaluation of patients in the second and third trimesters focuses on detecting fetal cardiac activity. In the pregnant trauma patient, standard trauma ultrasound (focused assessment with sonography for trauma [FAST]) technique should be performed.[34] In addition, longitudinal and transverse views of the uterus should be obtained.[35] Uterine views should be used to establish fetal heart rate; to assess the placenta for abnormalities; and to look for gross structural fetal abnormalities, fetal movement, and the presence of amniotic fluid.

Ultrasound evaluation for free intraperitoneal fluid after blunt abdominal trauma is reportedly up to 90% sensitive in nonpregnant and pregnant patients alike, including patients in their third trimester.[5,37] One study advocates including an assessment for intrauterine pregnancy in the FAST study for all patients of childbearing age.[38] In the study population, identification of pregnancy on FAST scan significantly decreased radiation exposure in patients at more than 8 weeks' gestational age. Pregnancies of less than 8 weeks' gestation were not identified by the transabdominal FAST scan; a pregnancy test was required.[38] Bochicchio and colleagues suggested consideration of the rapid bedside urine pregnancy test in addition to serum pregnancy testing to have results before the patient is exposed to any radiation.[39]

Classically, placental abruption is visualized as a retroplacental hematoma (i.e., hypoechogenic area between the placenta and uterine wall) on ultrasound images. However, successful identification by ultrasound occurs in only 50% of trauma patients with placental abruption. Thus, an ultrasound study should not be used to exclude an abruption.[33,37]

Imaging/Radiation Risks. Radiation in pregnancy has three potentially harmful effects: 1) teratogenesis and cell death, 2) carcinogenesis, and 3) germ cell mutations or genetic effects.[40]

Cell death due to high doses of radiation is presumably an all-or-none phenomenon. Prior to implantation, at 2 to 4 weeks' gestation, it appears that cell death occurs universally. In contrast, a wide range of teratogenic effects of radiation has been documented. Human teratogenic risks of radiation include microcephaly, growth restriction, and mental retardation. During the organogenesis period (4-10 weeks' gestation), the fetus is at greatest risk of developing birth defects. The fetus is at greatest risk of radiation-induced mental retardation at 10 to 17 weeks' gestation, during the period of neurologic development. Radiation levels above 20 rads have been correlated with mental retardation in a dose-related fashion. After 17 weeks' gestation, there are very rare cases of radiation-induced effects.[37,40,41]

The risk of developing childhood leukemia is only mildly increased by fetal radiation exposure from 1 in 3000 in the general pediatric population to 1 in 2000 exposed children.[40] This risk is not related to radiation dose and has been associated with low doses of ionizing radiation. Ultrasound utilizes sound waves and has no documented adverse fetal effects. However, resolution limits usefulness in the trauma setting to evaluating for free fluid and fetal activity. CT scans expose patients to radiation, yet are considered safe below 5 cumulative rads.[40] In addition, contrast materials are not radioactive and therefore may be given during pregnancy.[41,42] Magnetic resonance imaging presents no known risks to the fetus but generally is not recommended in the first trimester.[40] In the future, it may become an important imaging modality in pregnant patients. Gadolinium crosses the placenta and should not be given.[41,43]

There is no increased risk of fetal anomalies, growth restriction, or spontaneous abortion with a radiation exposure of less than 5 rads (the maximal cumulative dose of ionizing radiation to which a pregnant woman should be exposed).[44] However, the Committee on Obstetric Practice of the American College of Obstetricians and Gynecologists states that any concern about fetal radiation exposure and risks should not alter the decision to obtain medically indicated maternal radiographic studies and that risks, although real, are not an indication for therapeutic abortion.[40]

Standard initial trauma radiographic studies usually expose patients to near-maximum total pregnancy radiation. Bochicchio and colleagues determined that a standard head, abdominal, and pelvic helical CT scan delivers approximately 4.5 rads.[39] In a study involving 3,976 women of childbearing age, the investigators documented that 114 (2.9%) were pregnant. Thirteen of the pregnancies were incidental, either unknown to the woman or known by the woman but unable to be conveyed to the trauma team. Fetal mortality in the incidental group was 77%. Newly diagnosed pregnancies ($N = 9$) were earlier in gestation (6.9 ± 4.2 weeks) than known pregnancies unable to be made known to the trauma team (20.5 ± 5.8 weeks). These women with newly diagnosed pregnancies received more than 5 rads of radiation and had 100% fetal mortality (three by induced abortion, six by spontaneous abortion).[39] This study raises the question whether immediate point-of-care testing (e.g., bedside urine human chorionic gondaotropin [hCG]) should be reinstituted in an attempt to decrease these fetal losses. However, the care provider must determine whether the medical indication for the test outweighs the significant potential for fetal loss. Of note, controversy exists regarding the 4.5 rads of radiation noted in the aforementioned study, and institutional variance may exist.[45]

Prior to counseling pregnant patients, the emergency physician should discuss radiation exposure with a radiation physicist at his or her institution. It is then important to be honest with patients and families about the risks associated with radiation. Trauma care providers should have figures available to quickly calculate an estimated fetal radiation exposure to explain those risks to the family.[46] Table 2 is provided as a radiation risk reference.

Table 2. Estimated Fetal Exposure for Various Diagnostic Imaging Methods

EXAMINATION TYPE	EST. FETAL DOSE PER EXAM (RADS)	NO. OF EXAMS REQ FOR A CUMULATIVE 5-RAD DOSE
Plain Films		
Skull	0.004	1250
Dental	0.0001	50000
Cervical spine	0.002	2500
Upper or lower extremity	0.001	5000
Chest (2 views)	0.00007	71429
Abdominal (multiple views)	0.245	20
Thoracic spine	0.009	555
Lumbosacral spine	0.359	13
Pelvis	0.040	125
Hip (single view)	0.213	23
CT scans (slice thickness, 10 mm)		
Head (10 slices)	<0.050	>100
Chest (10 slices)	<0.100	>50
Abdomen (10 slices)	2.600	1
Lumbar spine (multiple views)	3.500	1
Environmental (for comparison)		
Background radiation (cumulative doses over 9 months)	0.100	N/A

Modified from Toppenberg KS, et al. Safety of radiographic imaging during pregnancy. *Am Fam Phys* 1999;59:1813.

Fetal Monitoring/Tocometry

Intrapartum fetal heart rate (FHR) monitoring helps the physician identify acidosis, fetal tachycardia, hypoxia, and umbilical cord compression.[47] Fetal cardiotocographic monitoring for both FHR and contractions is recommended for at least 4 hours after trauma; after 4 hours of continuous monitoring without complications, outcomes among pregnant women are similar to those of uninjured control patients.[30,48] Some authors advocate up to 48 hours of continuous monitoring, based upon reports of placental abruption during that length of time after maternal injury. One study identified patients requiring at least 24 hours of monitoring by uterine irritability (at least 6 contractions per hour), abdominal or uterine tenderness, vaginal bleeding, hypovolemia, or nonreassuring fetal heart monitoring.[49] Obstetricians at specific institutions frequently have a standard length of monitoring if there are no notable complications.

Prior to the mid-third trimester, it is very difficult to obtain prolonged fetal heart rate tracings, although continuous monitoring is recommended beyond 20 weeks' gestation. To assess fetal well-being, a tocometer belt is applied to the gravid abdomen and the tracing is assessed for contractions or uterine irritability; intermittent fetal heart tones are documented. After blunt abdominal trauma, uterine irritability may signify placental abruption.[50,51]

In third-trimester pregnancies, fetal cardiac monitoring and tocometry constitute an established method of determining fetal well-being. A normal tracing shows FHR of 120 to 160 bpm with good variability (*Figure 1*). Fetal tracings that should cause concern include those indicating decreased variability in FHR corresponding with early fetal distress (*Figure 2*) or acidosis (*Figure 3*), or late decelerations caused by significant fetal distress such as abruption (*Figure 4*) or fetal bradycardia (FHR < 120 bpm).

Although a normal FHR tracing is associated with good fetal outcome, the reverse is not always true.[51,52] For the sake of this discussion, any FHR tracing abnormalities should be brought to the attention of the obstetrician immediately. Unfortunately, in one study, fewer than 15% of EDs had cardiotocographic equipment, yet 92% of emergency residents reportedly were taught the indicators of fetal distress.[53] This disparity may contribute to the finding that most teaching institutions do not begin continuous fetal monitoring within the first 30 to 60 minutes after presentation, which warrants concern because abruption usually occurs shortly after injury.[53]

Complications of Trauma During Pregnancy

Fetal injury can separated into two categories: direct fetal injury and indirect fetal injury caused by placental disruption, uterine injury, maternal shock, or preterm labor.

Prior to 12 weeks' gestation, the uterus is a pelvic organ and relatively protected from abdominal trauma. After the uterus becomes an abdominal organ, it is more susceptible to injury. In addition, increased uterine blood flow makes significant hemorrhage more likely. Some studies show a decreased incidence of injury to other abdominal organs because of the gravid uterus.

Placental Abruption. Placental abruption is the most common cause of fetal demise after blunt abdominal trauma. Placental abruption is the shearing of the placenta from the uterine implantation site. In trauma, this condition likely is secondary to continued forward propulsion of the uterus, with muscular elasticity, without placental adaptation to the change in location and form. Hemorrhage occurs into the new space between the uterus and placenta. As bleeding increases, the expansion can continue to shear the placenta from the uterine wall.

Placental abruption complicates up to 40% to 50% of cases of severe blunt abdominal trauma or major trauma and 1% to 5% of cases of minor trauma.[5,21,23,25,32] The incidence of abruption is not predicted by the speed or force of trauma. Similarly, injury severity scores do not correlate well with the likelihood of abruption.[19] Abruption frequently occurs soon after the trauma incident, but it can occur up to 48 hours after the initial trauma. One multicenter

Figure 1. Normal Fetal Heart Rate Variability

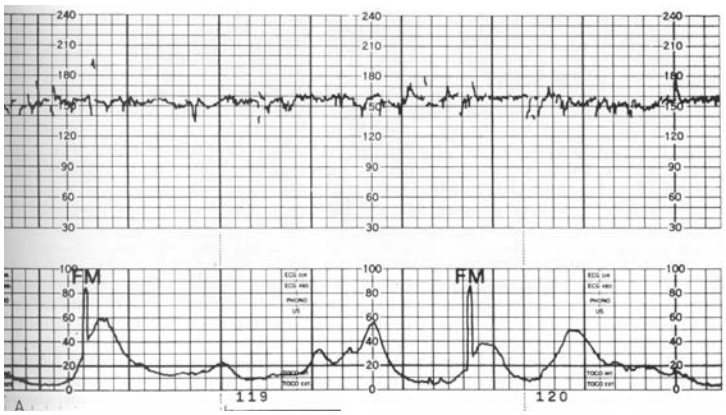

Figure 1. Example of normal fetal heart rate variability with accelerations greater than 15 bpm and longer than 15 seconds.

Reprinted with permission: Druzin ML, et al. Antepartum fetal evaluation. In: Gabbe SG, et al, eds. *Obstetrics: Normal and Problem Pregnancies*, 4th ed. New York:Churchill Livingstone;2002:325.

Figure 2. Fetal Movements

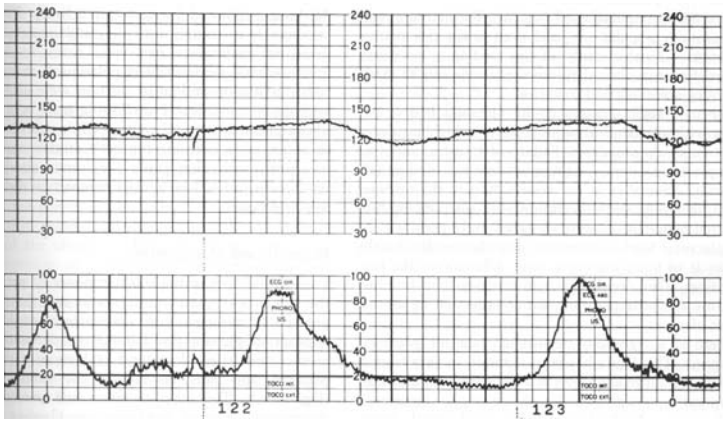

Figure 2. *Top*: Fetal movement (breaks in continuity of tracing) and no periodic changes. *Bottom:* Corresponding tocometer tracing.

Reprinted with permission: Benedetti TJ. Obstetric hemorrhage. In: Gabbe SG, et al, eds. *Obstetrics: Normal and Problem Pregnancies.* 4th ed. New York: Churchill Livingstone;2002:511.

study of admitted pregnant trauma patients reported a 3.5% incidence of abruption with resulting 54% mortality.[18] Disseminated intravascular coagulation occurs in up to 30% of patients with traumatic abruption.[53]

Evaluation for abruption includes a careful physical examination to identify the classic flat, board-like abdomen and vaginal bleeding. Vaginal bleeding caused by blood collecting in the potential space between the uterus and the placenta may or may not be present. Serial evaluations of fundal height may be performed at 15-minute intervals; a rising fundus correlates with concealed abruption.[14] As previously discussed, ultrasound has been utilized to identify and follow placental abruption; however, ultrasound should not be used to exclude the diagnosis.

Assessment of fetal well-being by cardiotocographic monitoring is the most sensitive test for abruption. In a previously mentioned multicenter study, cardiotocographic abnormalities (bradycardia or fetal distress) indicated abruption and led physicians to perform successful cesarean deliveries,[18] the definitive treatment for abruption with fetal distress. If the fetal status is reassuring on the cardiotocographic monitor, the patient can be monitored for a prolonged period and managed conservatively by the obstetrician.

Uterine Rupture. Uterine rupture, tearing of the uterine wall with release of the fetus into the abdominal cavity, occurs in less than 1% of pregnant trauma patients. However, due to the associated very poor maternal and fetal outcomes, uterine rupture is one of the most feared complications of trauma in the pregnant patient. Fetal mortality is 100%, and associated maternal mortality is 10%. The most common site of uterine rupture is the fundus, the superior portion of the uterus. Rupture is more common in patients with a history of previous cesarean delivery. Uterine rupture has been reported with lap belt use without shoulder restraint, airbag deployment, and pelvic fractures.[21,27] The mechanism of rupture in lap belt and air bag use likely is increased fundal pressure. The association between pelvic fractures and uterine rupture may be related to overall force of impact.

Penetrating Trauma. The position of the gravid uterus frequently prevents maternal visceral injury from penetrating abdominal trauma. The gravid uterus displaces the small bowel into the upper abdominal cavity, protecting it from direct abdominal trauma. However, the bladder is displaced out of the pelvis, increasing its susceptibility to injury. Injury patterns in penetrating trauma include direct fetal injury, placental injury or abruption, and uterine damage leading to preterm delivery. There are also case reports of uterine rupture caused by gunshot.[54] One study spanning 16 years of civil war in Lebanon showed maternal visceral injuries were present with entrance in the upper abdomen and back and absent if the entrance wound was below the fundus. However, half of cases with penetration below the fundus were associated with fetal demise.[55] Bullet wounds usually warrant surgical exploration. However, the management is controversial, and some advocate a conservative surgical approach to penetrating wounds, delaying laparotomy.[25,56]

Preterm Labor. In women who sustain severe trauma early in pregnancy, spontaneous abortions are almost universal. Interestingly, preterm labor or spontaneous abortion has been associated with trauma distant to the gravid uterus.[44] Trauma presumably results in the release of cytokines, which stimulate uterine contractions. Contractions developed in 28% of pregnant women with major blunt abdominal trauma in the series reported by Williams and colleagues.[57] Preterm labor leading to preterm delivery is actually very rare, accounting for less than 5% of fetal complications.[58]

If the fetus is viable, tocolysis should be discussed with the obstetrician but is not recommended until placental abruption has been excluded.[14] Even after abruption is excluded, routine med-

Figure 3. Acidosis and Decreased Fetal Heart Rate Variability

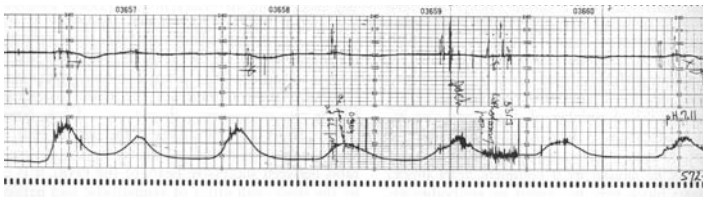

Figure 3. Acidosis with decreased fetal heart rate variability and late decelerations.

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Figure 4. Late Decelerations

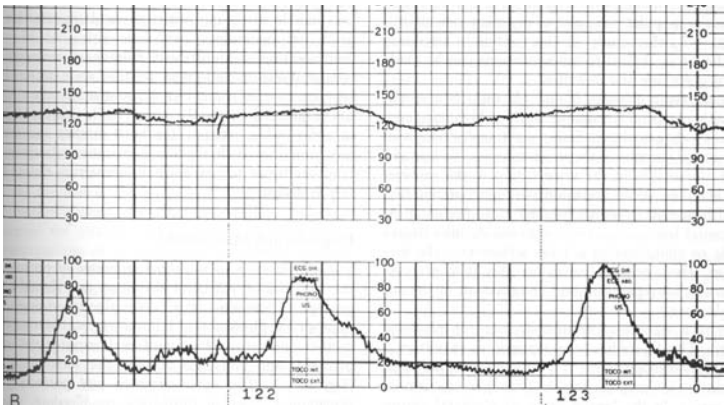

Figure 4. *Top*: Late decelerations with abruption. *Bottom:* Corresponding tocometer tracing.

Reprinted with permission: Benedetti TJ. Obstetric hemorrhage. In: Gabbe SG, et al. eds. *Obstetrics: Normal and Problem Pregnancies*. 4th ed. New York:Churchill Livingstone;2002:511.

ications used by obstetricians to treat preterm labor, including magnesium sulfate, terbutaline, and indomethacin, have side effects that may adversely affect the management of the pregnant trauma patient. Magnesium sulfate decreases respiratory effort and at high doses causes hypotension and arrhythmias. Beta-agonists (e.g., terbutaline) cause cardiac stimulation and even hypotension, confounding the evaluation of occult hemorrhage. Indomethacin may be contradicted in patients with head injury or occult bleeding because it affects platelet function.[23]

Fractures. Pelvic and acetabular fractures during pregnancy are associated with poorer outcomes than in nonpregnant patients.[60] Pregnancy predisposes the mother to significant retroperitoneal bleeding due to increased blood flow and severe hypovolemic shock.[1] Currently, there are different modes of managing pregnant patients with pelvic fractures including delayed fixation. Additional research likely will be completed in this area to determine the most successful route of treatment. Pelvic fractures are not an absolute contraindication to vaginal delivery.[1]

Disposition

Pregnant trauma patients without significant maternal injuries are not eligible for immediate discharge. Beyond 24 weeks' gestation, at least 4 hours of fetal monitoring is recommended. One study identified gestational age more than 35 weeks, assault, and pedestrian collision as risk factors associated with poor outcomes. Some centers monitor 24 hours, based upon persistently increased risk of fetal complications, including abruption beyond 6 hours, as previously discussed. One study advocates using the emergency observation unit for fetal monitoring if personnel are trained in interpretation of monitors and obstetrics staff is available for consultation.[61] In this case, care providers must be trained in interpretation of monitoring and have direct contact with an obstetrician. In academic centers, patients frequently are transferred to labor and delivery for prolonged monitoring if the acute trauma-related maternal issues have been addressed. If the patient requires ongoing trauma resuscitation unit care or trauma admission, there is variability in fetal monitoring practices. Depending upon gestational age and availability of obstetricians, patients may be monitored on the trauma floor by obstetrics nurses or followed by the trauma team on an obstetrics unit.

At the time of discharge after fetal monitoring or after maternal evaluation in the previable fetus, it is important to tailor the discharge instructions to the patient. Routine injury-specific discharge instructions should be given. In addition, the patient should follow up with her obstetrician as soon as possible. Patients should be instructed to return to the ED immediately if they experience any change in fetal activity, vaginal bleeding, abdominal pain or contractions, or leakage of fluid.

Special Considerations

Domestic Violence. Emergency care providers must have a high index of suspicion for domestic violence in pregnant trauma patients. Pregnancy is a significant risk factor for abuse, and studies have shown worsening patterns of abuse in pregnancy.[62] The 1985 National Family Violence Study reported 154 of 1000 pregnant women were assaulted by their partners in the first four months of pregnancy.[63,64] In addition, studies have established that the frequency of intentional injury sustained during pregnancy is increasing.[65] Studies have shown that direct interview questioning regarding abuse is the most effective way to identify that abuse is taking place.[66]

During pregnancy, the risk of homicide increases among battered women. A regional study by Krulewitch showed that maternal mortality resulting from violent death is underreported and thus goes underrecognized.[67] The gravid abdomen is one of the most common locations of partner-induced trauma. In addition, head and neck, breast, and genital injuries are common.[14,68]

Many studies have identified associations between domestic violence during pregnancy and adverse pregnancy outcomes. Adverse outcomes include placental abruption, spontaneous abortion, uterine rupture, preterm labor, hemorrhage, and low birth weight.[69] One study identified an association between com-

plications in pregnancy and physical violence, including partner-inflicted physical harm and being involved in a fight. The prevalence of physical violence in this study was 11.1%.[70] Overall, studies have shown inconsistent results regarding influence of violence on low birth weight and preterm birth.[71,72] These studies may be confounded by characteristics that commonly are associated with physical violence and with adverse pregnancy outcome, including young age, poverty, unmarried status, unwanted pregnancy, and substance abuse.[69] One study concluded that abuse was suspected or known prior to more than half of violent maternal deaths.[73] When eliciting a patient's history, it is important to remember the effect of domestic violence on pregnancy and ask questions regarding safety at home. Beyond the current pregnancy, abuse during pregnancy has been associated with future maternal homicide by the abusive partner.[74] The trauma care provider should be aware of appropriate documentation in suspected or alleged cases of abuse.[75]

Fetomaternal Hemorrhage and RhoGAM. All pregnant trauma patients should have a type and screen performed to determine their Rh antigen status, regardless of gestational age. Despite common beliefs that alloimmunization is rare in the early first trimester, even 38-day-old fetuses have detectable RhD antigen, and at 6 weeks Rh antigen is developed fully.[76] Minor trauma can be associated with significant fetomaternal hemorrhage. In one study, 28% of minor trauma cases, defined as a stable patient without need for surgery or admission and presenting with only contusions or superficial lacerations, experienced significant hemorrhage.[77] In trauma patients who are Rh negative, administration of 300 mg of RhoGAM (anti-D immunoglobulin) within 72 hours after onset of fetomaternal hemorrhage will prevent alloimmunization caused by up to 30 mL of Rh-positive fetal blood (15 mL of fetal cells). It has been established that anti-D immunoglobulin should be given to all Rh-negative patients with abdominal trauma.[78]

The Kleihauer-Betke test is used to quantify fetomaternal hemorrhage greater than 0.5 mL. The test has little utility in the acute trauma setting, because maternal fetal hemorrhage volume as low as 0.15 mL has elicited an antigen response; thus, all Rh-negative patients should be considered RhoGAM candidates, even if their Kleihauer-Betke test result is zero. The test can be used by the obstetrician to follow hemorrhage and to determine if additional doses of RhoGAM are indicated.

Medications. Acetaminophen is the only class A medication (i.e., determined safe in pregnant humans by controlled studies). All other medications used in pregnancy are class B (i.e., presumed to be safe, no evidence of risk) or class C (i.e., risk cannot be ruled out). Most medications used in the trauma setting are class B and C medications. Acute trauma-related pain usually is managed with opiates, which cross the placenta freely. Because the fetus absorbs opiates, decreased fetal heart rate variability and decreased fetal movements may be noted. These effects may confound evaluation of fetal well-being. Tetanus immunization is safe during pregnancy.[79]

Antibiotic use in the immediate trauma setting is frequently necessary for the management of open wounds or fractures. A first-generation cephalosporin often is used in this setting. Briggs and colleagues suggested that teratogenicity can be associated with cefaclor, cephalexin, and cephadrine but not with other cephalosporins.[80] Other antibiotics considered safe for use in pregnancy should be administered whenever possible. These include older penicillins (e.g., ampicillin and amoxicillin) and macrolides (e.g., erythromycin). Sulfonamides have no documented teratogenic effects;[81] however, they should not be used in the third trimester because of the increased risk of neonatal kernicterus. Antibiotics not recommended for use during pregnancy include the aminoglycosides and tetracyclines.[24] An update on the use of antibiotics during pregnancy was published by Niebyl.[82]

The use of acid-suppressing drugs (e.g., cimetidine, omeprazole, and ranitidine) is considered safe during pregnancy.[83]

Antiemetic agents safe for use during pregnancy include diphenhydramine (antihistamine) and phenothiazines such as promethazine, prochlorperazine, and thorazine.[84] Ondansetron has not been evaluated for safety.

In the hypotensive pregnant trauma patient, aggressive crystalloid fluid resuscitation and blood transfusion are the mainstays of therapy. Vasopressors reduce uterine blood flow and should be avoided if possible. If shock is unresponsive to volume resuscitation, ephedrine or dopamine should be considered. Ephedrine classically is used in nontrauma laboring or cesarean-section patients for hypotension without associated uteroplacental insufficiency and with possible increased uterine blood flow during contractions.[85,86] Low-dose dopamine (up to 5 mcg/kg/min) also has limited effect on placental blood flow, but dopamine at doses greater than 10 mcg/kg/min has been shown to decrease placental blood flow.[86]

Cardiac Arrest and Perimortem Cesarean Section. Cardiac arrest is complicated if the fetus is viable because there are two patients to consider during resuscitative efforts. Advanced cardiac life support should be performed; however, the effectiveness of cardiopulmonary resuscitation (CPR) is limited significantly by the physiologic changes of pregnancy, including decreased stroke volume while in the supine position. CPR also is limited significantly by the required left lateral positioning of the pregnant patient. Uterine evacuation has been shown to increase cardiac output by approximately 60%.[87] Emergent cesarean delivery of a viable fetus (> 23 weeks' gestation) should be performed after 4 minutes of unsuccessful maternal cardiac resuscitation. CPR should not be stopped to perform the cesarean delivery. The fetus should be delivered within 5 minutes of maternal resuscitation for optimal maternal and neonatal survival rates. There are case reports of delivery of viable infants after delayed perimortem delivery, but the most consistent infant survival rate (up to 70%) relies upon delivery within the first 5 minutes after arrest.[88]

The procedure may be completed within 5 minutes with a large scalpel and hemostats for clamping the umbilical cord. Sterile technique and draping generally are not used because they increase time to delivery. A vertical incision is made from the epigastric region to the symphysis pubis, following the darkened linea nigra. When the uterus has been exposed, a midline vertical uterine incision is initiated at the fundus with the scalpel and

completed with blunt-tipped scissors (e.g., bandage scissors), extending to the bladder reflection. If an anterior placenta is encountered, it should be incised. If the fetus is in the vertex (head-down) position, the operator's hand is placed into the cavity and under the fetal head, drawing it caudad out from the pelvis and through the uterine incision; the body will follow. Then, the umbilical cord should be double clamped and cut and the neonate handed to the team assigned to neonatal resuscitation. At this point, it is important to reassess the mother's vital signs. Delivery usually improves the response to resuscitative efforts significantly.[89]

Conclusion

The management of the pregnant trauma patient is a difficult and complex task and requires coordination between a variety of health care providers, including those in the prehospital, ED, trauma, operating room, and labor and delivery areas.

Several basic principles are paramount in the care of these patients. The first is the concentration of initial evaluation and treatment on maternal injuries; maternal death is the most common cause of trauma-associated fetal death.

The second principle is the importance of involvement of obstetricians and labor and delivery personnel in the care of these patients. This factor is especially important when the stage of fetal viability has been reached.

Finally, domestic violence reaches across our society and is increasing. The pregnant patient is especially at risk, and all health care workers need to be aware and ready to assist the victims of domestic violence and their unborn children.

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CE/CME Objectives

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

a.) discuss conditions that should increase suspicion for traumatic injuries;
b.) describe the various modalities used to identify different traumatic conditions;
c.) cite methods of quickly stabilizing and managing patients; and
d.) identify possible complications that may occur with traumatic injuries.

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.

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CE/CME Questions

1. What is the most common cause of trauma-associated fetal death?
   A. Fetal brain trauma
   B. Fetal spinal fracture
   C. Maternal death

2. Patients in the third trimester of pregnancy should be transported to the hospital in the left lateral tilt position. The benefit of this position is:
   A. Improved functional residual capacity
   B. Increased comfort for the patient
   C. Prevention of aortocaval compression

3. Examination of a pregnant patient's abdomen reveals a fundal height at the level of the umbilicus. What is the estimated gestational age?
   A. 16 weeks
   B. 20 weeks
   C. 24 weeks

4. Serial examinations of fundal height performed on a pregnant patient reveal a rising fundus. This finding indicates which of the following conditions?
   A. Concealed placental abruption
   B. Placenta previa
   C. Preterm labor

5. Which complication of traumatic injury in the pregnant patient is associated with 100% fetal mortality?
   A. Placental abruption
   B. Placenta previa
   C. Uterine rupture

6. The most effective way to identify that a pregnant woman is a victim of domestic violence is:
   A. Direct interview questioning
   B. Fetal monitoring
   C. Physical examination

7. A pregnant patient with abdominal trauma should be given RhoGAM if:
   A. she is Rh negative and in any stage of pregnancy.
   B. she is Rh negative and in the second trimester or later.
   C. she is Rh negative and in the third trimester.

8. Which of the following regarding placental abruption is true?
   A. It is not associated with minor trauma.
   B. It never occurs more than 6 hours after trauma.
   C. It is the most common cause of fetal demise after blunt abdominal trauma.

9. The goal during postmortem cesarean section is to deliver the fetus within how many minutes after maternal arrest?
   A. 5 minutes
   B. 10 minutes
   C. 15 minutes

10. In a pregnant patient with vaginal bleeding, what test can determine if the blood is fetal or maternal in origin?
    A. Apt test
    B. Ferning test
    C. Fetal DEX test

Answers 1. C; 2. C; 3. B; 4. A; 5. C; 6. A; 7. A; 8. C; 9. A; 10. A

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THOMSON™
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Dear *Trauma Reports* Subscriber:

Trauma Reports provides you with evidence-based information and best practices that help you make informed decisions concerning treatment options and medical practices. Our intent is the same as yours - the best possible patient care.

The objectives of *Trauma Reports* are to:

1. Discuss conditions that should increase suspicion for traumatic injuries;
2. Describe the various modalities used to identify different traumatic conditions;
3. Cite methods of quickly stabilizing and managing patients; and
4. Identify possible complications that may occur with traumatic injuries.

Each issue of your newsletter contains questions relating to the information provided in that issue. After reading the issue, answer the questions at the end of the issue to the best of your ability. You can then compare your answers against the correct answers provided in an answer key in the newsletter. If any of your answers were incorrect, please refer back to the source material to clarify any misunderstanding.

Enclosed in this issue is an evaluation form. Please complete it and return it to us in the enclosed envelope. Please make sure you sign the attestation verifying that you have completed the activity as designed. Once we have received your completed evaluation form, we will mail you a certificate of completion.

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On behalf of Thomson American Health Consultants, we thank you for your trust and look forward to continuing our educational partnership.

Sincerely,

Brenda L. Mooney

Vice-President/Group Publisher
Thomson American Health Consultants