

Emergency Medicine Reports

The Practical Journal for Emergency Physicians

Volume 33, Number 20 / September 10, 2012

www.emreports.com

Authors:

Larissa I. Velez, MD, Assistant Professor of Emergency Medicine, University of Texas Southwestern, Dallas.

Melanie J. Lippmann, MD, Department of Emergency Medicine, University of Texas Southwestern, Dallas.

Janna Welch, MD, Assistant Residency Director, University of Texas Southwestern, Dallas.

Gilberto A. Salazar, MD, Department of Emergency Medicine, University of Texas Southwestern, Dallas.

Peer Reviewer:

Frank LoVecchio, DO, Emergency Medicine Department, Maricopa Medical Center, Phoenix, AZ.

Statement of Financial Disclosure

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Farel (CME question reviewer) owns stock in Johnson & Johnson. Dr. Stapczynski (editor) owns stock in Bristol Myers Squibb. Dr. Schneider (editor), Dr. Velez (author), Dr. Lippmann (author), Dr. Welch (author), Dr. Salazar (author), Dr. LoVecchio (peer reviewer), Ms. Mark (executive editor), and Ms. Hamlin (managing editor) report no financial relationships with companies related to the field of study covered by this CME activity.

Rhabdomyolysis: Review and Update

In the past few weeks, I have seen several patients with rhabdomyolysis. One of the more memorable patients was a person who had too much to drink at a party, and the guests restrained him with duct tape to keep him from driving. After a night of struggling to get free, he developed dark urine and was admitted to the hospital.

We have also seen rhabdomyolysis from self-poisoning and as a side effect of medication. Although the references suggest this disease is somewhat uncommon, it may be that we overlook some cases.

— Sandra M. Schneider, MD, Editor

Introduction

Rhabdomyolysis is a syndrome with far-reaching systemic sequelae. The release of intracellular components into the vascular space following striated muscle injury produces tissue necrosis, end-organ damage, and metabolic derangements. The incidence of rhabdomyolysis is approximately two cases per 10,000 person-years in North America.¹ The mortality rate is estimated to be 8%.²

Rhabdomyolysis was first described in the medical literature after the London bombings during World War II, and began to be fully recognized following studies on military recruits as early as the 1970s. Olerud published a study in 1976 detailing exertional rhabdomyolysis in Marine Corps recruits in 1976.³ The syndrome has received much more attention in the last 30 years, and now we have a much better understanding of its pathophysiology.

Striated muscle injury is at the core of mechanisms producing vast systemic insults in rhabdomyolysis. Central to the development of the syndrome is intracellular ATP depletion.⁴ This triggers a cascade of cellular disruptions, such as Na⁺ K⁺ ATPase pump dysfunction, impaired calcium transport, and release of free oxygen radicals.⁵ Intracellular calcium levels increase in unregulated fashion, leading to persistent muscle contraction.⁶ Eventually, there is cell death. Intracellular components such as myoglobin, creatine kinase (CK), lactate dehydrogenase (LDH), uric acid, aldolase, and electrolytes (e.g., potassium) are released into the bloodstream.⁴

Rhabdomyolysis is a systemic event. It involves virtually every organ system. Disseminated intravascular coagulation (DIC), compartment syndrome, electrolyte abnormalities, cardiac arrhythmias, and acute renal failure form part of a large list of potentially life-threatening complications from rhabdomyolysis.⁷

The kidneys are exquisitely sensitive to myoglobin. Not only do the renal tubules sustain significant damage from tissue necrosis and obstruction, but there is also a direct toxic effect of ferrihemate, a byproduct of myoglobin breakdown. Hypovolemia and aciduria appear to compound the effect of myoglobin, resulting in acute renal failure. It is estimated that 30-50% of rhabdomyolysis patients develop acute renal failure.⁸ Seven percent to 10% of all cases of acute kidney injury in the United States are due to rhabdomyolysis.⁶

Similar mechanisms may be responsible for the vascular insult and tissue

Executive Summary

- Rhabdomyolysis is caused by the breakdown of muscle tissue. Frequent causes include trauma (especially crush injuries), drugs (particularly statins), insect stings, and infections.
- While classically rhabdomyolysis presents with muscle tenderness, weakness, and dark urine, often patients present without all of the classic symptoms.
- Acute renal failure is the most common complication of rhabdomyolysis, but electrolyte abnormalities of hyperkalemia, hypocalcemia, and hyperuricemia are seen.
- The mainstay of treatment is aggressive fluid resuscitation with either normal saline or lactated Ringer's. Bicarbonate and mannitol have been used, but there are some data to suggest they may not be essential.

necrosis that affects the cardiovascular system and brain, leading to the cardiopulmonary and neurological findings associated with rhabdomyolysis.

There are many causes of rhabdomyolysis. Exercise has traditionally been considered the main culprit. Release of CK during strenuous exercise is a well-documented phenomenon.⁹ However, it is important to remember that there are many other causes of muscle injury. Immobilization, medications, environmental exposures, infection, and work-related hazards (e.g., electrical injury) are well known to cause muscle injury, tissue ischemia, and necrosis. It is crucial to remember that rhabdomyolysis is often multifactorial, and each cause can compound the effects of the other.⁸

Clinical Presentation

The assessment of the patient with rhabdomyolysis must take into consideration the systemic nature of the syndrome. The patient with rhabdomyolysis may present with classic signs and symptoms, including myalgias, weakness, and dark-colored urine. However, only approximately 50% of patients with rhabdomyolysis experience myalgias or weakness. Emergency physicians should be aware that even an obtunded patient may have rhabdomyolysis.

Knowledge of the patient's activities, behavior, and environment prior to the ED visit should trigger the addition of rhabdomyolysis to the list of differential diagnoses. A history should include a thorough medication review looking for medications associated with rhabdomyolysis.

Table 1: Major Causes of Rhabdomyolysis

Exertional <ul style="list-style-type: none">• Genetic predisposition
Trauma
Drugs
Toxins/venoms
Infection
Environmental <ul style="list-style-type: none">• Heat or cold
Electrolyte abnormalities
Food

The physical examination may vary widely, and may even be normal. However, fever, tachycardia, hypertension, hypotension, tachypnea, evidence of immobilization, agitation, central nervous system (CNS) depression, seizure activity, edema, and increased muscle tone may be found. Patients with altered mental status and rhabdomyolysis may be particularly difficult to diagnose. The emergency physician should consider the diagnosis when the history is suggestive.

The laboratory workup is essential in determining optimal management of the patient suspected of having rhabdomyolysis. A CK level of 5000 IU/L, or five times the normal level, is considered by most to be rhabdomyolysis, although a definitive number varies in the published literature.¹⁰ The CK should be monitored serially, as its rise may be evidence of ongoing muscle injury. An electrolyte panel, including renal function, magnesium, phosphorus, and calcium should be obtained. A bedside capillary blood glucose is extremely useful in ruling out hypoglycemia. An arterial blood gas is

helpful in determining serum pH. Serum and urine myoglobin may be obtained as part of the workup. The patient's coagulation may be useful, as DIC is a severe complication of rhabdomyolysis. Obtain a complete blood cell count with differential, blood culture, and urine culture when infection is highly suspected as the cause of rhabdomyolysis. Chest radiography may help delineate the cause when a pulmonary source is suspected in patients with rhabdomyolysis. A lumbar puncture should be considered in the patient with rhabdomyolysis who have a clinical suspicion of bacterial meningitis.

Cardiac ischemia is a potential complication of rhabdomyolysis, particularly in patients with comorbidities or those known to abuse sympathomimetic drugs. An electrocardiogram and cardiac markers suffice as an initial cardiac workup. A toxicologic workup aids in delineating possible triggers for muscle injury, but a positive test should not stop the search for other possible causes. Panels testing for drugs of abuse are widely available, but other offending agents, such as salicylates,

alcohol, and opiate narcotics, must be considered. When altered mental status is present, a CT of the brain should be considered as an adjunct when intracranial pathology enters the differential diagnosis.

Causes of Rhabdomyolysis

Exertional. The most common causes of non-traumatic rhabdomyolysis are hypermetabolic exertional stress injury. Strenuous exercise will increase serum CK in normal humans. Exertional rhabdomyolysis, or “hyper-CK-emia” occurs in individuals who have a sudden increase in overall levels of physical activity and can demonstrate CK levels more than five times normal. There is an increased incidence in males, African-Americans, and individuals with high muscle mass.⁹ The majority of these individuals do not have muscle soreness, weakness, and myoglobinuria, and have CK levels that rapidly return to normal. When hyperCK-emia and myoglobinuria occur in a patient with muscle soreness and weakness, the patient has exertional rhabdomyolysis. A common factor in exertional rhabdomyolysis cases is repetitive exercise or exertion beyond when fatigue would compel an individual to normally stop (for example, new military recruits and participants in long-distance running events). It is exacerbated by high ambient temperatures.

Exertional rhabdomyolysis occurs when exertional energy requirements exceed ATP production. Depletion of ATP within the myocyte during exertion causes a release of calcium into the cell and, therefore, cellular necrosis. The myocytes then become permeable, swollen with fluid, and leak their components into the intracellular matrix. This results in intravascular volume depletion from third spacing and lactic acidosis. There is a resultant rapid increase in serum creatinine. The prognosis for exertional rhabdomyolysis is better than for other forms of rhabdomyolysis and less often results in acute kidney injury. However, exertional rhabdomyolysis exacerbated by severe

Table 2: Hereditary Conditions Associated with Rhabdomyolysis

<p>Carnitine Metabolism Disorders</p> <ul style="list-style-type: none"> • Carnitine palmitoyl transferase (CPT2) deficiency • VLCAD (very long chain acyl CoA deficiency) <p>Adenosine monophosphate deaminase deficiency (AMPD) McArdle’s disease (glycogen storage disease type 5) Malignant hyperthermia Phosphorylase kinase deficiency Duchenne muscular dystrophy 11-hydroxylase deficiency Phosphofructokinase deficiency</p>

Table 3: Trauma Conditions Associated with Rhabdomyolysis

<ul style="list-style-type: none"> • Crush injury • Compartment syndrome • Physical torture and abuse • Exercise • Heat stroke • High voltage electrical injury • Lightning • Elevated ambient temperature (heat exposure) • Low ambient temperature (cold exposure)

heat injury will increase risk of renal injury.

Anyone may develop exertional rhabdomyolysis when under enough mechanical and environmental stress. However, exertional rhabdomyolysis can also be triggered by genetic influences that predispose the individual to the development of exercise and heat-related illness. These genetic abnormalities cause abnormal intracellular skeletal muscle calcium regulation via disorders of carbohydrate metabolism, lipid metabolism, or mitochondrial disorders. Recognized genetic causes include McArdle’s disease, CPT2 deficiency, and AMPD deficiency.⁹ In McArdle’s disease, the individual lacks the enzyme to break down muscle glycogen to continue to fuel cells after circulating ATP are spent. Carnitine palmitoyl transferase (CPT2) deficiency causes an increase in open-state probability of RYR1 calcium channels, so there is a much lower threshold for high calcium levels within cells to cause cell breakdown.

In adenosine monophosphate deaminase deficiency (AMPD), a critical enzyme for muscle energy metabolism is present in abnormally low levels, therefore decreasing exercise capacity and shortening the time to tissue ischemia. Polymorphic variations in angiotensin-converting enzyme, CK muscle isoform, and myosin light-chain kinase have also been associated with exertional rhabdomyolysis.⁹ Hereditary causes are listed in Table 2.

Trauma. Traumatic injury is the most common cause of rhabdomyolysis.¹¹ Table 3 lists the common trauma causes of rhabdomyolysis. The two types of trauma that result in rhabdomyolysis include crush and electrical injuries. Physical beating has also been associated with rhabdomyolysis.¹² Electrical injuries may be caused by lightning or high-voltage electrical current. Due to the short duration of exposure, lightning injuries do not cause significant burns or muscle breakdown. In contrast, high-voltage electrical injuries

commonly cause rhabdomyolysis in patients who survive the initial insult. In such cases, rhabdomyolysis cannot be predicted by the size of external wound or site of electrical current entry.^{8,13} Up to 10% of patients with severe electrical injuries will develop rhabdomyolysis.¹⁴

Crush injury is the most common traumatic cause of rhabdomyolysis, as it is seen in natural disasters such as earthquakes and landslides, or in war zones, in which individuals are trapped under fallen buildings.¹⁴ Epidemics of traumatic and crush injuries have been described following massive earthquakes.^{15,16} Crush injuries occur when a patient is trapped or compressed under the weight of external forces. Due to external pressure compressing the affected limb, there is inadequate blood pressure to deliver blood and oxygen to the tissue. Intramuscular compartment pressure may rapidly exceed arterial blood pressure, resulting in muscle tamponade, compartment syndrome, and muscular necrosis within the first 30 minutes of injury.¹⁴ Severe crush injury may result in transient flaccid paralysis without spinal cord injury secondary to an increase in compartment pressures and ischemia to peripheral nerves.

Decreased blood flow and lactic acidosis from tissue ischemia causes the release of vasodilatory nitric oxide in crushed muscle. As a result, there is rapid swelling within muscle compartments once the injured limb is released from entrapment. This massive third spacing often causes sudden hypotension, hypocalcemia, and hyperkalemia.¹⁴ Pre-renal azotemia from hypotension, along with lactic acidosis and hyperCKemia, result in oliguria and acute kidney injury. Renal failure causes further sudden increases in potassium levels. The cardiotoxic effects of hyperkalemia are aggravated by hypocalcemia from muscle breakdown. Hypovolemic shock and arrhythmias are the most common early causes of death.¹⁷

Drug-related. The list of drugs associate with rhabdomyolysis is extensive, including more than

200 medications.¹⁸ It is estimated that drugs and medications cause about one-third of the cases of adult rhabdomyolysis.¹⁸

There are some common mechanisms for drug-induced rhabdomyolysis:¹⁹

- Inadequate delivery of oxygen and nutrients to the tissue.¹⁹ This can be due to pressure ischemia from prolonged immobilization that can occur with any central nervous system (CNS) depressant. Examples include the narcotics, general anesthetics, benzodiazepines (BDZ), tricyclic antidepressants (TCAD), antihistamines, ethanol, barbiturates, and carbon monoxide (CO). The syndrome can also be due to increased pressure within specific compartments, as when the person passes out in an awkward position.

Some drugs cause significant vaso-spasm or vasoconstriction that can restrict blood flow. One example is vasopressin. Other drugs result in a “functional anemia” due to the production of abnormal hemoglobins that cannot transport and/or deliver oxygen to the tissues. Carboxyhemoglobin and methemoglobin are examples of such drugs. Finally, drugs that cause hemodynamic shock have the potential to cause rhabdomyolysis.

- Excessive energy use by the muscle.¹⁹ Any drug that results in excessive exertion, delirium, agitation, and seizures can cause rhabdomyolysis. This group of drugs is extensive and includes the sympathomimetics, LSD, and PCP. Isoniazid (INH), strychnine, theophylline, and lithium are some of the drugs known to result in seizures and status epilepticus. The withdrawal from ethanol, BDZ, and gamma hydroxybutyrate (GHB) can also cause both agitation and seizures.

Drugs that cause movement disorders like dystonias and choreo-athetosis are also in this category, such as the phenothiazines and butyrophenones.

Depolarizing neuromuscular blockers like succinylcholine have been associated with rhabdomyolysis in children.

- Metabolic poisons, which are drugs that interfere with the production or use of ATP.¹⁹ Drugs in this category include the inhibitors of the electron transport chain (cyanide [CN], hydrogen sulfide (HS), CO, and phosphine); the uncouplers of oxidative phosphorylation (salicylates and chlorophenoxy herbicides; and the inhibitors of glycolysis [sodium fluoroacetate]). Rarer agents in this group include the heavy metals like mercury, selenium, copper, and tetra ethyl lead.

- Potassium depletion.¹⁹ Patients in this category include those taking diuretics or mineralocorticoids (like licorice), and toluene users. The mechanism for rhabdomyolysis in hypokalemia is thought to be due to inadequate cell release of potassium, which helps with vasodilation during exercise.

- Other miscellaneous mechanisms.¹⁹ **Ethanol.** Ethanol is thought to account for about 20% of cases of myoglobinuria.¹⁹ The mechanism for this is not clear, but could in part be behavioral and also due to a local toxic effect.

HMG-CoA reductase inhibitors (statins). Statins are the drug class most commonly associated with drug-induced rhabdomyolysis.⁴ This is probably because they are one of the most widely prescribed drug classes.²⁰ The myopathies from statins are a spectrum of disease, ranging from isolated CK elevation with no symptoms, to pain or weakness with little CK elevation, to profound rhabdomyolysis.

Rhabdomyolysis due to statins is thought to be due to inhibition of HMG-CoA reductase, which leads to decreased levels of ubiquinone (Coenzyme Q).²⁰ Ubiquinone is a key player in electron transport chain, and is also an intracellular antioxidant.²¹ However, the exact mechanism has not been elucidated.

Higher serum statin concentrations have been associated with a higher likelihood of rhabdomyolysis.²² Statins undergo glucuronidation in the liver. Drugs like gemfibrozil, which have a similar metabolism, can increase serum statin levels.²⁰

Subsequently, most statins are metabolized by cytochrome oxidase 3A4 (CYP 3A4), so co-administration of drugs such as macrolide antibiotics, non-dihydropyridine calcium channel blockers, and protease inhibitors may also increase blood concentrations of statins and precipitate rhabdomyolysis.²⁰

There is no consensus on screening for rhabdomyolysis in patients taking statins, but patients taking other drugs that can result in elevated statin levels, or patients on high-dose simvastatin, should be recognized as at high risk for developing the syndrome.^{20,22}

Propofol. Propofol is a widely used sedative and short-acting anesthetic due to its very favorable neurologic profile and its quick “on and off” properties.²³ Propofol is toxic to the mitochondria and elevates malonyl-carnitine levels, which results in inhibited fatty acid transport. It also uncouples oxidative phosphorylation and inhibits the respiratory chain.⁴ The toxicity of propofol is also thought to be mediated to beta antagonism and catecholamine inhibition.²⁴

The Propofol Infusion Syndrome (PRIS) was first termed by Bray in 1998. In this series, he described 18 children who developed bradycardia leading to asystole with at least one of the following: metabolic acidosis, rhabdomyolysis or myoglobinuria, lipemic plasma, and enlarged or fatty liver. Most patients had a respiratory illness. The mortality in this report was 83%.²⁵ In more recent reports, the mortality from PRIS has been 18%.²⁶ Between 1996-2000, the first adult cases were reported. In 2001, a report first showed a dose relationship in the development of PRIS in adult head-injured patients.²⁷ In this series, patients taking doses of less than 5 mg/kg/hr did not develop PRIS; those taking doses between 5-6 mg/kg/hr had a 17% incidence; and those taking doses greater than 6 mg/kg/hr had a 31% incidence.

Risk factors for the development of PRIS include: severe head injury; respiratory illness; young age; large total cumulative dose; high

catecholamine and glucocorticoid levels; low carbohydrate/high fat intake; critical illness; and inborn errors of fatty oxidation.²⁷

The proposed adult criteria for PRIS are:

- Age between 18-55 years;
- Progressive heart failure with arrhythmias;
- Two of the following: metabolic acidosis, hyperkalemia, or evidence of muscle cell destruction;
- Exclusion of other causes for symptoms.²³

Cardiac dysfunction or a Brugada-like ECG may be the first clue of PRIS. This is thought to be due to NA channel blockage or unmasking of a genetic channel defect.²³

A recently published report, the largest one to date, gives an incidence of 1.1% for PRIS. Most patients developed symptoms by the first 24 hours. In this study, most patients (91%) were receiving vaso-pressors. Interestingly, only 18% of patients were receiving propofol at a rate greater than 5 mg/kg/hr doses, demonstrating that PRIS can occur at low doses.²⁶

To prevent the development of PRIS, when possible propofol infusions should be less than 4 mg/kg/hr and they should not be continued longer than 48 hours.²³ However, cases of PRIS have been reported even after propofol has been used for procedural sedation.²³ The propofol infusion should be stopped and the patient appropriately resuscitated. Case reports have shown some success with hemodialysis, hemoperfusion, and extracorporeal membrane oxygenation (ECMO).^{4,23}

Drugs of Abuse. Virtually all the sympathomimetics have been associated with rhabdomyolysis. Most notably, cocaine has been reported to cause rhabdomyolysis in about 5% of all cocaine-related visits in one ED study.²⁸ As expected, the degree of agitation was the best predictor for the development of rhabdomyolysis. Patients with cocaine-induced rhabdomyolysis have been reported to have high mortality.²⁹ The mechanism for cocaine-induced rhabdomyolysis is thought to be multifactorial,

including agitation, seizures, vasoconstriction, and other causes of skeletal muscle dysfunction like neuroleptic malignant syndrome (NMS), serotonin syndrome (SS), and malignant hyperthermia (MH).

The newer synthetic amphetamines like MDMA and N-benzylpiperazine have also been reported to result in rhabdomyolysis. Finally, the synthetic cathinone derivatives, such as “bath salts” (MDPV and mephedrone), have also been associated with rhabdomyolysis.^{30,31}

Table 4 lists the drugs that have been associated with rhabdomyolysis.

Toxins/Venoms. Rare cases of rhabdomyolysis have been documented after red fire ant bites, massive stings from Africanized bees, wasp stings, and following snake bites.³²⁻³⁵ The spread of Africanized honey bees across the southern United States has increased the incidence of massive stings. When the bee colony feels threatened by human activity, the bees swarm in defense. In contrast to anaphylaxis secondary to a bee or wasp sting with laryngeal constriction, bronchospasm, and hypotension, rhabdomyolysis occurs secondary to massive envenomation from hundreds of stings. Melittin, the pain-inducing compound in bee venom, in conjunction with phospholipase A2, compromise red blood cell membrane integrity. The mast-cell degranulating protein, hyaluronidase, and neurotoxic apamin allow bee venom to infiltrate tissues and propagate cell apoptosis, resulting in rhabdomyolysis and renal failure.³⁵

Fire ant venom contains formic acid, which in large quantities acts as an inhibitor of the mitochondrial cytochrome oxidase complex.³³ Rhabdomyolysis may occur secondary to tissue ischemia from the formic acid. Severe scorpion stings from certain species (most notably the *Centruroides* species, known also as the “bark scorpion”) are reported to result in elevated CK, but in patients who also suffered severe neurotoxic and cardiotoxic effects.³⁶ Bites from pit vipers with hemotoxic or myotoxic venom may

Table 4: Medication/Drug Causes of Rhabdomyolysis¹⁹

- Ethanol
- Cocaine and other sympathomimetics
- Anticholinergic agents
- Sedatives and hypnotics
- Metabolic poisons (cyanide, carbon monoxide)
- Colchicine
- Steroids
- Zidovudine
- Statins
- Propofol (infusion)
- Daptomycin
- Sunitinib and Imatinib
- Leflunomide
- Serotonin syndrome
- Neuroleptic malignant syndrome

also cause rhabdomyolysis, which in conjunction with disseminated intravascular coagulation (DIC), hypotension, and hemorrhage may result in renal failure.³² Table 5 lists common envenomations associated with rhabdomyolysis.

Infection. Many infections have been related to the development of rhabdomyolysis. In adults, infections may represent only about 5% of total cases of rhabdomyolysis, but in the pediatric population, infection is the most common cause of rhabdomyolysis.¹⁸ In at least one adult cohort study of patients with sepsis, the presence of rhabdomyolysis carried a mortality of 59%.³⁷ Table 6 summarizes infectious causes of rhabdomyolysis.

Other. There are many additional causes of rhabdomyolysis, including tumors, endocrine, electrolyte derangements, hereditary, and dietary. All are listed in Table 7.

Complications of Rhabdomyolysis

As rhabdomyolysis is a multi-organ disease, its complications are many. The most common and obvious complication is acute renal failure (ARF).^{19,40} The association was first reported by Bywaters in 1941.⁴¹ It is thought that rhabdomyolysis causes 5-8% of all cases of ARF in hospitals. Renal failure occurs when released myoglobin, which is partly bound to alpha-2 globulin, dissociates into

ferrihemate and globin. This occurs predominantly at low serum pH. Ferrihemate results in direct toxicity to the kidney. Additionally, there is alteration of renal blood flow (RBF), and tubular obstruction due to precipitation of heme pigment casts and uric acid crystals.^{18,19} Other aspects of rhabdomyolysis that contribute to ARF include: intravascular volume depletion with renal hypoperfusion and ischemia; oxidative stress to the kidney from iron-mediated free radical formation; myoglobin-induced nitric oxide scavenging; circulation of inflammatory mediators; and activation of innate immune system.⁴² The ARF can be oliguric or non-oliguric.

Nearly half of patients with rhabdomyolysis develop hyperkalemia.⁴³ Potassium is released from damaged muscle, and its clearance is impaired from fall in glomerular filtration rate (GFR) and decreased renal function.^{19,40} Profound hyperuricemia, as high as 36 mg/dL, has also been reported.^{19,40} Purines released from injured muscle are converted to uric acid by the liver. Due to the falling renal function, uric acid levels may rise.¹⁹ Other electrolytes are also deranged in rhabdomyolysis, including hypocalcemia, found in 63% of patients.⁴⁰ Both calcium and phosphorus are sequestered and deposited in injured muscle. Later, once ARF ensues, these low levels resolve, and hypercalcemia can develop.¹⁹

Disseminated intravascular

coagulation (DIC) has been reported to be associated with rhabdomyolysis. It could result from temperature elevation or from systemic release of substances such as plasminogen activator or thromboplastin.^{19,40} Metabolic (lactic) acidosis is also common and has many contributors, including hypocalcemia, renal failure, and strenuous exertion leading to anaerobic metabolism.^{19,40}

Rarer complications include hepatic damage, cardiomyopathy, ECG changes, arrhythmias, and cardiogenic shock.⁴⁰ Respiratory failure, delayed compartment syndromes, and peripheral neuropathies have also been reported.¹⁹

Laboratory Evaluation

Creatine kinase (CK) is the most sensitive indicator of rhabdomyolysis. Most authors will use a CK greater than five times the upper limit of normal as a definition for rhabdomyolysis, but there is no absolute number that defines the syndrome.¹⁰ The CK starts increasing within 12 hours from injury, peaks at 1-3 days, and declines 3-5 days after the insult ceases (declines by about 39% from the previous day's value).^{10,11} It should be noted that these criteria and elevations are variable and poorly studied, especially in heat-related illness and drug-induced rhabdomyolysis. Once 100 grams of muscle are damaged, myoglobin is released into the circulation.

The renal threshold for myoglobin is 15 mg/dL. Once this threshold is reached, myoglobinuria ensues. Myoglobinuria can be detected using the orthotolidine reaction, in which myoglobin is detected as blood in urine. This test is very sensitive.¹⁹ Visible urine discoloration occurs at myoglobin levels of more than 1,000 mg/L. At alkaline pHs, myoglobin is red or pink; at acidic pHs, it is dark red-brown (from ferrihemate). But, it is important to note that the discoloration also depends on urine flow and GFR and is therefore very variable.¹⁹

As discussed before, the electrolytes, particularly hyperkalemia, hypocalcemia, and hyperuricemia are

also clues to the presence of rhabdomyolysis. Both the creatinine and the BUN are elevated in rhabdomyolysis, but the normal ratio of 10:1 decreases to about 6:1.¹¹

Management

Intravenous Crystalloid Fluid Resuscitation. The cornerstone for the successful treatment of rhabdomyolysis and the prevention of ARF is prompt and aggressive intravenous isotonic crystalloid resuscitation.^{16,44} Fluid sequestration in injured skeletal muscles of rhabdomyolysis patients leads to intravascular volume depletion, and repletion of up to 10-12 liters per day is often needed to maintain adequate urine output.^{6,45}

Numerous studies have confirmed an increased incidence of acute kidney injury (AKI) when fluid initiation is delayed or not aggressive.^{14,45,46} Thus, prompt administration of large-volume crystalloid repletion is important. Aggressive fluid resuscitation can improve long-term patient outcomes and prevent progression to ARF. Similarly, pre-hospital EMS providers can assist by beginning the process while en route to the ED. Maintaining a high index of suspicion for the development of rhabdomyolysis in the appropriate setting of presumed skeletal muscle injury is key to early and effective treatment.

Following an initial fluid bolus of 20 cc/kg, intravenous fluids should be continued at a rate of approximately 400-500 cc/hr.^{6,47} Placement of a urinary catheter is necessary to closely monitor urine output, which should be maintained at a target of 2-3 cc/kg/hr (or approximately 200-300 cc/hr).^{6,45,47} Volume overload and pulmonary edema is infrequent, even after infusion of these large volumes. In older and more frail patients, however, volume overload can occur. It is important that these patients be monitored for signs of pulmonary edema.⁴⁵

No large, randomized, controlled trials exist to demonstrate the superiority of 0.9% normal saline (NS) versus lactated Ringer's (LR) as the solution of choice for volume

Table 5: Envenomations Associated with Rhabdomyolysis¹⁹

<ul style="list-style-type: none"> • Ants (fire ants in particular) • Bees • Centipedes • Wasps • Scorpions • Snakes (hemotoxic and myotoxic snakes)
--

Table 6: Infections Associated with Rhabdomyolysis

<p>Viral</p> <ul style="list-style-type: none"> • Influenza (H1N1; A; B) • Coronavirus • Herpesvirus • HIV • Dengue • Parainfluenza • Varicella • West Nile encephalitis • Mononucleosis (Epstein-Barr) • Cytomegalovirus (CMV) • Coxsackie
<p>Bacterial</p> <ul style="list-style-type: none"> • <i>Staphylococcus aureus</i> • <i>Salmonella typhi</i> • <i>Pseudomonas aeruginosa</i> • <i>Mycoplasma pneumoniae</i> • <i>Bacillus cereus</i> • <i>Clostridium tetani</i> • <i>E. coli</i> • <i>Listeria monocytogenes</i> • <i>Legionella pneumophila</i> • Tularemia • Tetanus
<p>Protozoan</p> <ul style="list-style-type: none"> • <i>P. vivax</i> (malaria)

resuscitation in rhabdomyolysis patients. However, because large volumes of NS lead to the development of a metabolic acidosis, some authors advocate LR.^{6,47} In a small study that randomized 28 patients to NS versus LR resuscitation for the treatment of rhabdomyolysis induced by doxylamine intoxication, Cho and colleagues found that in order to maintain an alkaline urine (pH > 6.5), the addition of sodium bicarbonate was required more frequently in the NS group as compared to the

LR group. They thus concluded that LR is a superior crystalloid in this setting.⁴⁷ However, no patients in either group developed renal failure necessitating dialysis, and time to CK normalization (recovery) did not differ significantly between the two groups. Most case series of patients with crush injuries use normal saline as the fluid of choice.⁴⁵ Particularly in the early ED phase of volume resuscitation, the choice of NS versus LR is likely less important.

During the initiation of treatment

Table 7: Miscellaneous Causes of Rhabdomyolysis

Electrolytes
<ul style="list-style-type: none">• Hypokalemia• Hypernatremia• Hypocalcemia• Hypophosphatemia
Food
<ul style="list-style-type: none">• Quail ingestion (coturnism)³⁸• Mushrooms• Licorice• Red yeast rice (<i>Monascus purpureus</i>)³⁹
Endocrine
<ul style="list-style-type: none">• Thyrotoxicosis• Hyperaldosteronism• DKA
Other
<ul style="list-style-type: none">• Status asthmaticus• Massage• Polymyositis• Dermatomyositis• Neurosarcoidosis• Sjögren's syndrome

for significant rhabdomyolysis, patients should be placed on a cardiac monitor, and electrolytes checked frequently due to the risk of dysrhythmias relating to metabolic derangements, particularly hyperkalemia. Hemodynamic monitoring may also be required to avoid fluid overload in susceptible patients. Patients with significant rhabdomyolysis should be admitted to a monitored bed setting for these reasons. The rigorous treatment of hyperkalemia, which typically first begins early in the disease process, is paramount and may require dialysis if severe.

With the goal of preventing ARF, it is also important to minimize patient exposure to additional potential renal insults, including intravenous contrast material, nephrotoxic antibiotics, and other nephrotoxic medications such as ACE inhibitors and NSAIDs.⁶ It is also imperative to search for and treat the underlying cause of rhabdomyolysis, and to exclude or treat compartment syndrome in affected injured muscle groups.

Urine Alkalinization Using Sodium Bicarbonate and Mannitol Osmotic Diuresis. Despite the common practice of urine alkalinization to a pH greater than 6.5 using sodium bicarbonate and forced diuresis with mannitol, no randomized, controlled trials support their superiority to adequate fluid resuscitation alone for the treatment of rhabdomyolysis and prevention of ARF.^{6,11} Most of the data to support their use come from the cardiothoracic and the renal transplant literature.⁴⁵

Homsy and colleagues performed a retrospective review of rhabdomyolysis patients admitted to an ICU setting and found no difference in the rate of ARF in patients receiving saline, sodium bicarbonate, and mannitol versus those receiving saline alone.⁴⁶ Similarly, in a retrospective analysis of trauma ICU patients with rhabdomyolysis, Brown et al noted no difference in the rates of ARF as defined by a creatinine greater than 2.0 mg/dL (22% vs. 18%), need for dialysis (7% vs. 6%), or mortality

(15% vs. 18%) among patients receiving fluid resuscitation with the addition of sodium bicarbonate and mannitol compared to those who received only volume replacement.⁴⁸ These authors concluded the standard practice of bicarbonate and mannitol therapy should be reconsidered for the treatment of rhabdomyolysis.

While the only clear disadvantage of urine alkalinization with sodium bicarbonate is exacerbation of the hypocalcemia associated with the initial phase of rhabdomyolysis, mannitol has been found to actually worsen renal failure if it is used without adequate fluid resuscitation or if used late in the course of rhabdomyolysis.^{6,45} One possible additional benefit of sodium bicarbonate is the concomitant treatment of hyperkalemia and of metabolic acidosis.

Hemodialysis

Despite appropriate fluid therapy, approximately 10-50% of rhabdomyolysis patients progress to develop ARF.^{11,14,44,48} In a retrospective review of 2,083 trauma ICU patients, CK levels of 5000 U/L and higher were found to be associated with an increased risk of developing of ARF.⁴⁸ In such cases of rhabdomyolysis-induced ARF complicated by oliguria, severe hyperkalemia, acidosis, or volume overload, hemodialysis (HD) is often required. Either daily HD or continuous hemofiltration can be used successfully to correct fluid and electrolyte abnormalities resulting from such processes as the release of excess potassium and urea from necrotic skeletal muscle. HD has also been shown to remove large amounts of myoglobin when used before ARF is established.⁴⁹ Continuous venovenous hemofiltration may be more advantageous in critically ill patients with hemodynamic instability, as it is not associated the hypotension and dysrhythmias caused by the rapid fluid shifts of HD.^{14,44} The use of plasmapheresis for removal of myoglobin has been shown to be beneficial in a few case reports, but its routine use is not indicated.⁵⁰

Experimental Therapies

Isolated case reports have documented the success of high-dose corticosteroids for rhabdomyolysis unresponsive to fluid resuscitation² and the possible benefit of rasburicase, a urate oxidase enzyme, for the treatment of hyperuricemia in patients with rhabdomyolysis and renal failure.⁵¹ Small case series, case reports, and experimental models support the use of antioxidants and free-radical scavengers such as pentoxifylline and vitamins C and E, in addition to deferoxamine for the prevention of myoglobinuric renal failure.^{6,11,14} Another animal model showed decreased CK release from muscle after exposure to ethanol, cocaine, and electricity with the use of dantrolene sodium.⁵² However, none of these therapies is proven by controlled studies, and, thus, sufficient evidence of their efficacy is lacking.

In summary, early and aggressive crystalloid fluid resuscitation remains the mainstay of treatment of rhabdomyolysis and the prevention of rhabdomyolysis-associated renal failure. The addition of sodium bicarbonate should be considered in patients with crush injury and in those with severe metabolic acidosis, but its routine use is not supported by published evidence.

Conclusions

Rhabdomyolysis is a complex syndrome that has a multitude of causes. The classic description of muscle pain, muscle weakness, and dark urine is seldom completely present, so a high index of suspicion must be maintained for those at high risk of the syndrome: a history of heavy exertion, trauma (especially crush injuries), agitation, motor excitation, and seizures. However, other causes should also be considered, such as infections and drugs. In the ED, the insult should be identified if possible and promptly removed.

Aggressive, early isotonic crystalloid resuscitation is central to the prevention of ARF, the most common complication from rhabdomyolysis. In patients with metabolic

acidosis and/or hyperkalemia, sodium bicarbonate can be used, although there is no definitive evidence showing its benefit.

References

1. Continuous renal replacement therapy (CRRT) for rhabdomyolysis (Protocol). John Wiley and Sons, Ltd.; 2010. Accessed August, 2012.
2. Antoon JW, Chakraborti C. Corticosteroids in the treatment of alcohol-induced rhabdomyolysis. *Mayo Clinic Proceedings* 2011;86(10):1005-1007.
3. Olerud JE, Homer LD, Carroll HW. Incidence of acute exertional rhabdomyolysis. Serum myoglobin and enzyme levels as indicators of muscle injury. *Arch Intern Med* 1976;136(6):692-697.
4. Hohenegger M. Drug induced rhabdomyolysis. *Curr Opin Pharmacology* 2012;12(3):335-339.
5. Tein I. Metabolic myopathies. *Semin Pediatric Neurol* 1996;3(2):59-98.
6. Bosch X, Poch E, Grau JM. Rhabdomyolysis and acute kidney injury. *N Engl J Med* 2009;361(1):62-72.
7. Khan F. Rhabdomyolysis: A review of the literature. *Netherlands J Med* 2009;67(9):272-283.
8. Desai B. Rhabdomyolysis: Evaluation and Emergent management. *Emergency Medicine* 2012;11-16.
9. Landau ME, Kenney K, Deuster P, Campbell W. Exertional rhabdomyolysis: A clinical review with a focus on genetic influences. *J Clin Neuromuscular Dis* 2012;13(3):122-136.
10. Khan FY. Rhabdomyolysis: A review of the literature. *Netherlands J Med* 2009;67(9):272-283.
11. Huerta-Alardin AL, Varon J, Marik PE. Bench-to-bedside review: Rhabdomyolysis — an overview for clinicians. *Crit Care* 2005;9(2):158-169.
12. Bowley DM, Buchan C, Khulu L, Boffard KD. Acute renal failure after punishment beatings. *J Royal Society of Medicine* 2002;95(6):300-301.
13. Desai B. Emergent management of lightning injuries. *Emergency Medicine* 2011: 7-13.
14. Malinoski DJ, Slater MS, Mullins RJ. Crush injury and rhabdomyolysis. *Crit Care Clin* 2004;20(1):171-192.
15. Better OS, Stein JH. Early management of shock and prophylaxis of acute renal failure in traumatic rhabdomyolysis. *N Engl J Med* 1990;322(12):825-829.
16. Sanadgol H, Najafi I, Rajabi Vahid M, et al. Fluid therapy in pediatric victims of the 2003 bam, Iran earthquake. *Prehospital Disaster Med* 2009;24(5): 448-452.
17. Oda J, Tanaka H, Yoshioka T, et al. Analysis of 372 patients with crush syndrome caused by the Hanshin-Awaji earthquake. *J Trauma* 1997;42(3):470-475; discussion 475-476.
18. Elsayed EF, Reilly RF. Rhabdomyolysis: A review, with emphasis on the pediatric population. *Pediatr Nephrol* 2010;25(1):7-18.
19. Curry SC, Chang D, Connor D. Drug- and toxin-induced rhabdomyolysis. *Ann Emerg Med* 1989;18(10):1068-1084.
20. Rivers C, Nelson L. Diffuse muscle weakness: The toxicologist's approach. *Emergency Medicine* 2011:22-26.
21. Thompson PD, Clarkson P, Karas RH. Statin-associated myopathy. *JAMA* 2003;289(13):1681-1690.
22. Egan A, Colman E. Weighing the benefits of high-dose simvastatin against the risk of myopathy. *N Engl J Med* 2011;365(4):285-287.
23. Wong JM. Propofol infusion syndrome. *Am J Therapeutics* 2010;17(5):487-491.
24. Fong JJ, Sylvia L, Ruthazer R, et al. Predictors of mortality in patients with suspected propofol infusion syndrome. *Crit Care Med* 2008;36(8):2281-2287.
25. Bray RJ. Propofol infusion syndrome in children. *Paediatric Anaesthesia* 1998;8(6):491-499.
26. Roberts RJ, Barletta JF, Fong JJ, et al. Incidence of propofol-related infusion syndrome in critically ill adults: A prospective, multicenter study. *Crit Care* 2009;13(5):R169.
27. Cremer OL, Moons KG, Bouman EA, et al. Long-term propofol infusion and cardiac failure in adult head-injured patients. *Lancet* 2001;357(9250):117-118.
28. Brody SL, Wrenn KD, Wilber MM, et al. Predicting the severity of cocaine-associated rhabdomyolysis. *Ann Emerg Med* 1990;19(10):1137-1143.
29. Roth D, Alarcon FJ, Fernandez JA, et al. Acute rhabdomyolysis associated with cocaine intoxication. *N Engl J Med* 1988;319(11):673-677.
30. Adebamiro A, Perazella MA. Recurrent acute kidney injury following bath salts intoxication. *Am J Kidney Diseases* 2012;59(2):273-275.
31. Berney-Meyer L, Putt T, Schollum J, et al. Nephrotoxicity of recreational party drugs. *Nephrology (Carlton)* 2012;17(2):99-103.
32. Sitprija V. Animal toxins and the kidney. Nature clinical practice. *Nephrology* 2008;4(11):616-627.
33. Koya S, Crenshaw D, Agarwal A. Rhabdomyolysis and acute renal failure after fire ant bites. *J General Internal Medicine* 2007;22(1):145-147.
34. Vachvanichsanong P, Dissaneewate P. Acute renal failure following wasp sting in children. *Eur J Pediatrics* 2009;188(8):991-994.
35. Vetter RS, Visscher PK, Camazine S. Mass envenomations by honey bees and wasps. *Western J Med* 1999;170(4):223-227.
36. O'Connor A, Ruha AM. Clinical course of bark scorpion envenomation man-

aged without antivenom. *J Med Toxicol* 2012;8(3):258-262.

37. Kumar AA, Bhaskar E, Palamaner Subash Shantha G, et al. Rhabdomyolysis in community acquired bacterial sepsis — a retrospective cohort study. *PLoS One* 2009;4(9):e7182.
38. Korkmaz I, Kukul Guven FM, Eren SH, et al. Quail consumption can be harmful. *J Emerg Med* 2011;41(5):499-502.
39. Kuncl RW. Agents and mechanisms of toxic myopathy. *Curr Opin Neurology* 2009;22(5):506-515.
40. Welch RD, Todd K, Krause GS. Incidence of cocaine-associated rhabdomyolysis. *Ann Emerg Med* 1991;20(2):154-157.
41. Bywaters EG, Beall D. Crush injuries with impairment of renal function. *BMJ* 1941;1(4185):427-432.
42. Bagshaw SM, Bellomo R, Devarajan P, et al. Review article: Renal support in critical illness. *Can J Anaesthesia* 2010;57(11):999-1013.
43. Gabow PA, Kaehny WD, Kelleher SP. The spectrum of rhabdomyolysis. *Medicine* 1982;61(3):141-152.
44. Cruz DN, Bagshaw SM. Does continuous renal replacement therapy have a role in the treatment of rhabdomyolysis complicated by acute kidney injury? *Semin Dialysis* 2011;24(4):417-420.
45. Better OS, Abassi ZA. Early fluid resuscitation in patients with rhabdomyolysis. Nature reviews. *Nephrology* 2011;7(7):416-422.
46. Homsy E, Barreiro MF, Orlando JM, et al. Prophylaxis of acute renal failure in patients with rhabdomyolysis. *Renal Failure* 1997;19(2):283-288.
47. Cho YS, Lim H, Kim SH. Comparison of lactated Ringer's solution and 0.9% saline in the treatment of rhabdomyolysis induced by doxylamine intoxication. *Emerg Med J* 2007;24(4):276-280.
48. Brown CV, Rhee P, Chan L, et al. Preventing renal failure in patients with rhabdomyolysis: Do bicarbonate and mannitol make a difference? *J Trauma* 2004;56(6):1191-1196.
49. Splendiani G, Mazzarella V, Cipriani S, et al. Dialytic treatment of rhabdomyolysis-induced acute renal failure: Our experience. *Renal Failure* 2001;23(2):183-191.
50. Swaroop R, Zabaneh R, Parimoo N. Plasmapheresis in a patient with rhabdomyolysis: A case report. *Cases Journal* 2009;2:8138.
51. Lin PY, Lin CC, Liu HC, et al. Rasburicase improves hyperuricemia in patients with acute kidney injury secondary to rhabdomyolysis caused by ecstasy intoxication and exertional heat stroke. *Pediatric Crit Care Med* 2011;12(6):e424-427.
52. Pagala M, Amaladevi B, Bernstein A, et al. Dantrolene sodium reduces the enhanced leakage of creatine kinase caused by ethanol, cocaine, and electrical stimulation in isolated fast and slow

muscles of rat. *Alcoholism, Clinical and Experimental Research* 1997;21(1):63-67.

Physician CME Questions

1. Which of the following metabolic disturbances is *not* commonly seen with rhabdomyolysis?
 - A. hyperuricemia
 - B. disseminated intravascular coagulation
 - C. hyponatremia
 - D. hypocalcemia
2. Which of the following statements is true?
 - A. Urine alkalization with sodium bicarbonate decreases progression to ARF.
 - B. Forced diuresis with mannitol protects against progression to ARF.
 - C. Lactated Ringer's solution is superior to normal saline in the treatment of rhabdomyolysis.
 - D. Early and aggressive fluid resuscitation is associated with a decreased progression to ARF.
3. Creatine kinase levels most consistent with acute rhabdomyolysis are:
 - A. greater than 2 times normal
 - B. greater than 5 times normal
 - C. greater than 10 times normal
 - D. greater than 20 times normal
4. All of the following are known genetic influences that predispose the patient to exertional rhabdomyolysis *except*:
 - A. McArdle's disease
 - B. CPT2 deficiency
 - C. AMPD deficiency
 - D. beta thalassemia
5. Severe crush injury may result in transient flaccid paralysis of an isolated extremity secondary to:
 - A. acute spinal cord injury
 - B. direct trauma to peripheral nerves
 - C. increased compartment pressures with resultant ischemia to peripheral nerves
 - D. traumatic brain injury
6. Contributing causes of acute kidney injury resulting from crush injury include all of the following *except*:
 - A. hypotension
 - B. lactic acidosis
 - C. hyper-CK-emia
 - D. vasoconstriction by nitric oxide
7. Rhabdomyolysis has been associated with all of the following *except*:
 - A. fire ant bites
 - B. Africanized bee stings
 - C. brown recluse spider bites
 - D. pit viper envenomation
8. Rhabdomyolysis in Africanized bee stings occurs secondary to:
 - A. anaphylaxis

CME Instructions

HERE ARE THE STEPS YOU NEED TO TAKE TO EARN CREDIT FOR THIS ACTIVITY:

1. Read and study the activity, using the provided references for further research.
2. Log on to www.cmecity.com to take a post-test; tests can be taken after each issue or collectively at the end of the semester. *First-time users will have to register on the site using the 8-digit subscriber number printed on their mailing label, invoice, or renewal notice.*
3. Pass the online tests with a score of 100%; you will be allowed to answer the questions as many times as needed to achieve a score of 100%.
4. After successfully completing the last test of the semester, your browser will be automatically directed to the activity evaluation form, which you will submit online.
5. **Once the completed evaluation is received, a credit letter will be e-mailed to you instantly.** You will no longer have to wait to receive your credit letter.

Emergency Medicine Reports

CME Objectives

Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

- B. mast cell degranulation
 - C. formic acid release
 - D. disseminated intravascular coagulation
9. Acute kidney injury secondary to crush injury may cause the following concerning electrolyte abnormality:
- A. hyperglycemia
 - B. hypoglycemia
 - C. hyperkalemia
 - D. hyponatremia
10. What percentage of patients with severe electrical injuries will develop acute rhabdomyolysis?
- A. fewer than 1%
 - B. 3%
 - C. 10%
 - D. 30%

In Future Issues

Newer Anticoagulants in the ED

To reproduce any part of this newsletter for promotional purposes, please contact:

Stephen Vance

Phone: (800) 688-2421, ext. 5511

Fax: (800) 284-3291

Email: stephen.vance@ahcmedia.com

To obtain information and pricing on group discounts, multiple copies, site-licenses, or electronic distribution please contact:

Tria Kreutzer

Phone: (800) 688-2421, ext. 5482

Fax: (800) 284-3291

Email: tria.kreutzer@ahcmedia.com

Address: AHC Media
3525 Piedmont Road, Bldg. 6,
Ste. 400, Atlanta, GA 30305 USA

To reproduce any part of AHC newsletters for educational purposes, please contact:

The Copyright Clearance Center for permission

Email: info@copyright.com

Website: www.copyright.com

Phone: (978) 750-8400

Fax: (978) 646-8600

Address: Copyright Clearance Center
222 Rosewood Drive, Danvers, MA 01923 USA

Editors

Sandra M. Schneider, MD

Professor
Department of Emergency Medicine
University of Rochester School of
Medicine
Rochester, New York

J. Stephan Stapczynski, MD

Chair
Emergency Medicine Department
Maricopa Medical Center
Phoenix, Arizona

Editorial Board

Paul S. Auerbach, MD, MS, FACEP

Professor of Surgery
Division of Emergency Medicine
Department of Surgery
Stanford University School of
Medicine
Stanford, California

Brooks F. Bock, MD, FACEP

Professor
Department of Emergency Medicine
Detroit Receiving Hospital
Wayne State University
Detroit, Michigan

William J. Brady, MD, FACEP, FAAEM

Professor and Vice Chair of
Emergency
Medicine, Department of Emergency
Medicine,
University of Virginia School of
Medicine
Charlottesville, Virginia

Kenneth H. Butler, DO FACEP, FAAEM

Associate Professor, Associate
Residency Director
University of Maryland Emergency
Medicine Residency Program
University of Maryland School
of Medicine
Baltimore, Maryland

Michael L. Coates, MD, MS

Professor and Chair
Department of Family and
Community Medicine
Wake Forest University School
of Medicine
Winston-Salem, North Carolina

Alasdair K.T. Conn, MD

Chief of Emergency Services
Massachusetts General Hospital
Boston, Massachusetts

Charles L. Emerman, MD

Chairman
Department of Emergency Medicine
MetroHealth Medical Center
Cleveland Clinic Foundation
Cleveland, Ohio

Kurt Kleinschmidt, MD, FACEP, FACMT

Professor of Surgery/Emergency
Medicine
Director, Section of Toxicology
The University of Texas
Southwestern Medical Center and
Parkland Hospital
Dallas, Texas

David A. Kramer, MD, FACEP, FAAEM

Program Director,
Emergency Medicine Residency
Vice Chair
Department of Emergency Medicine
York Hospital
York, Pennsylvania

Larry B. Mellick, MD, MS, FAAP, FACEP

Professor, Department of Emergency
Medicine and Pediatrics
Medical College of Georgia
Augusta, Georgia

Paul E. Pepe, MD, MPH, FACEP, FCCM, MACP

Professor of Medicine, Surgery,
Pediatrics, Public Health and Chair,
Emergency Medicine
The University of Texas

Southwestern Medical Center and
Parkland Hospital
Dallas, Texas

Charles V. Pollack, MA, MD, FACEP

Chairman, Department of Emergency
Medicine, Pennsylvania Hospital
Associate Professor of Emergency
Medicine
University of Pennsylvania School of
Medicine
Philadelphia, Pennsylvania

Robert Powers, MD, MPH

Professor of Medicine and
Emergency
Medicine
University of Virginia
School of Medicine
Charlottesville, Virginia

David J. Robinson, MD, MS, FACEP

Vice-Chairman and Research Director
Associate Professor of Emergency
Medicine
Department of Emergency Medicine
The University of Texas - Health
Science Center at Houston
Houston, Texas

Barry H. Rumack, MD

Director, Emeritus
Rocky Mountain Poison and Drug
Center
Clinical Professor of Pediatrics
University of Colorado Health
Sciences Center
Denver, Colorado

Richard Salluzzo, MD, FACEP

Chief Executive Officer
Wellmont Health System
Kingsport, Tennessee

John A. Schriver, MD

Chief, Department of Emergency
Services
Rochester General Hospital
Rochester, New York

David Sklar, MD, FACEP

Professor of Emergency Medicine

Associate Dean, Graduate Medical
Education
University of New Mexico School of
Medicine
Albuquerque, New Mexico

Charles E. Stewart, MD, FACEP

Professor of Emergency Medicine,
Director, Oklahoma Disaster Institute
University of Oklahoma, Tulsa

Gregory A. Volturo, MD, FACEP

Chairman, Department of Emergency
Medicine
Professor of Emergency Medicine
and Medicine
University of Massachusetts Medical
School
Worcester, Massachusetts

Albert C. Weihl, MD

Retired Faculty
Yale University School of Medicine
Section of Emergency Medicine
New Haven, Connecticut

Steven M. Winograd, MD, FACEP

St. Barnabus Hospital
Core Faculty
Emergency Medicine Residency
Program
Albert Einstein Medical School
Bronx, New York

Allan B. Wolfson, MD, FACEP, FACP

Program Director,
Affiliated Residency in Emergency
Medicine
Professor of Emergency Medicine
University of Pittsburgh
Pittsburgh, Pennsylvania

CME Question Reviewer

Roger Farel, MD

Retired
Newport Beach, CA

© 2012 AHC Media. All rights
reserved.

Emergency Medicine Reports™ (ISSN 0746-2506)
is published biweekly by AHC Media, a division of
Thompson Media Group LLC, 3525 Piedmont Road,
N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305.
Telephone: (800) 688-2421 or (404) 262-7436.

Senior Vice President/Group Publisher:

Donald R. Johnston

Executive Editor:

Shelly Morrow Mark

Managing Editor:

Leslie Hamlin

GST Registration No.:

R128870672

Periodicals Postage Paid at Atlanta, GA 30304 and at
additional mailing offices.

POSTMASTER: Send address
changes to Emergency Medicine
Reports, P.O. Box 105109, Atlanta,
GA 30348.

Copyright © 2012 by AHC Media, Atlanta, GA. All rights
reserved. Reproduction, distribution, or translation
without express written permission is strictly prohibited.

Back issues: \$31. Missing issues will be fulfilled by
customer service free of charge when contacted within
one month of the missing issue's date.

Multiple copy prices: One to nine additional copies, \$359
each; 10 to 20 additional copies, \$319 each.

Subscriber Information

Customer Service: 1-800-688-2421

Customer Service E-Mail:
customerservice@ahcmedia.com

Editorial E-Mail:
shelly.mark@ahcmedia.com

World Wide Web page:
http://www.ahcmedia.com

Subscription Prices

1 year *with* 60 ACEP/65 AMA/39 AAFP
Category 1/Prescribed credits: \$544

1 year *without* credit: \$399
Add \$17.95 for shipping & handling

Resident's rate \$199

Discounts are available for group
subscriptions, multiple copies, site-licenses
or electronic distribution. For pricing
information, call
Tria Kreutzer at 404-262-5482.

All prices U.S. only.
U.S. possessions and Canada, add \$30
plus applicable GST. Other international
orders, add \$30.

Accreditation

AHC Media is accredited by the
Accreditation Council for Continuing
Medical Education to provide continuing
medical education for physicians.

AHC Media designates this enduring
material for a maximum of *65 AMA PRA
Category 1 Credits™*. Each issue has been
designated for a maximum of *2.50 AMA
PRA Category 1 Credits™*. Physicians
should claim only credit commensurate
with the extent of their participation in the
activity.

Approved by the American College of
Emergency Physicians for 60 hours of
ACEP Category 1 credit.

This Enduring Material activity, *Emergency
Medicine Reports*, has been reviewed
and is acceptable for up to 39 Prescribed
credit(s) by the American Academy of
Family Physicians. AAFP accreditation
begins January 1, 2012. Term of approval
is for one year from this date with the
option of yearly renewal. Each issue is
approved for 1.50 Prescribed credits.
Physicians should claim only the credit
commensurate with the extent of their
participation in the activity.

Please forward your comments on the
quality of this activity to [cmecomment@
aafp.org](mailto:cmecomment@aafp.org).

This is an educational publication
designed to present scientific information
and opinion to health professionals,
to stimulate thought, and further
investigation. It does not provide
advice regarding medical diagnosis or
treatment for any individual case. It is not
intended for use by the layman. Opinions
expressed are not necessarily those of
this publication. Mention of products or
services does not constitute endorsement.
Clinical, legal, tax, and other comments
are offered for general guidance only;
professional counsel should be sought for
specific situations.

This CME activity is intended for
emergency and family physicians. It is in
effect for 24 months from the date of the
publication.

© 2012 AHC Media. All rights reserved.

AHC Media

Major Causes of Rhabdomyolysis

Exertional

- Genetic predisposition

Trauma

Drugs

Toxins/venoms

Infection

Environmental

- Heat or cold

Electrolyte abnormalities

Food

Hereditary Conditions Associated with Rhabdomyolysis

Carnitine Metabolism Disorders

- Carnitine palmitoyl transferase (CPT2) deficiency
- VLCAD (very long chain acyl CoA deficiency)

Adenosine monophosphate deaminase deficiency (AMPD)

McArdle's disease (glycogen storage disease type 5)

Malignant hyperthermia

Phosphorylase kinase deficiency

Duchenne muscular dystrophy

11-hydroxylase deficiency

Phosphofructokinase deficiency

Trauma Conditions Associated with Rhabdomyolysis

- Crush injury
- Compartment syndrome
- Physical torture and abuse
- Exercise
- Heat stroke
- High voltage electrical injury
- Lightning
- Elevated ambient temperature (heat exposure)
- Low ambient temperature (cold exposure)

Medication/Drug Causes of Rhabdomyolysis

- Ethanol
- Cocaine and other sympathomimetics
- Anticholinergic agents
- Sedatives and hypnotics
- Metabolic poisons (cyanide, carbon monoxide)
- Colchicine
- Steroids
- Zidovudine
- Statins
- Propofol (infusion)
- Daptomycin
- Sunitinib and Imatinib
- Leflunomide
- Serotonin syndrome
- Neuroleptic malignant syndrome

Envenomations Associated with Rhabdomyolysis

- Ants (fire ants in particular)
- Bees
- Centipedes
- Wasps
- Scorpions
- Snakes (hemotoxic and myotoxic snakes)

Infections Associated with Rhabdomyolysis

Viral

- Influenza (H1N1; A; B)
- Coronavirus
- Herpesvirus
- HIV
- Dengue
- Parainfluenza
- Varicella
- West Nile encephalitis
- Mononucleosis (Epstein-Barr)
- Cytomegalovirus (CMV)
- Coxsackie

Bacterial

- *Staphylococcus aureus*
- *Salmonella typhi*
- *Pseudomonas aeruginosa*
- *Mycoplasma pneumoniae*
- *Bacillus cereus*
- *Clostridium tetani*
- *E. coli*
- *Listeria monocytogenes*
- *Legionella pneumophila*
- Tularemia
- Tetanus

Protozoan

- *P. vivax* (malaria)

Miscellaneous Causes of Rhabdomyolysis

Electrolytes

- Hypokalemia
- Hypernatremia
- Hypocalcemia
- Hypophosphatemia

Food

- Quail ingestion (coturnism)
- Mushrooms
- Licorice
- Red yeast rice (*Monascus purpureus*)

Endocrine

- Thyrotoxicosis
- Hyperaldosteronism
- DKA

Other

- Status asthmaticus
- Massage
- Polymyositis
- Dermatomyositis
- Neurosarcoidosis
- Sjögren's syndrome

Supplement to *Emergency Medicine Reports*, September 10, 2012: "Rhabdomyolysis: Review and Update." Authors: Larissa I. Velez, MD, Assistant Professor of Emergency Medicine, University of Texas Southwestern, Dallas; Melanie J. Lippmann, MD, Department of Emergency Medicine, University of Texas Southwestern, Dallas; Janna Welch, MD, Assistant Residency Director, University of Texas Southwestern, Dallas; and Gilberto A. Salazar, MD, Department of Emergency Medicine, University of Texas Southwestern, Dallas.

Emergency Medicine Reports' "Rapid Access Guidelines." Copyright © 2012 AHC Media, a division of Thompson Media Group LLC, Atlanta, GA. Editors: Sandra M. Schneider, MD, FACEP, and J. Stephan Stacpzyński, MD. Senior Vice President/Group Publisher: Donald R. Johnston. Executive Editor: Shelly Morrow Mark. Managing Editor: Leslie Hamlin. For customer service, call: 1-800-688-2421. This is an educational publication designed to present scientific information and opinion to health care professionals. It does not provide advice regarding medical diagnosis or treatment for any individual case. Not intended for use by the layman.

Trauma Reports

EVIDENCE-BASED MEDICINE FOR THE ED

Volume 13, Number 5

Sept/Oct 2012

Authors:

Michael C. Bond, MD, FACEP, FAAEM, Assistant Professor, Residency Program Director, Department of Emergency Medicine, University of Maryland School of Medicine, Baltimore.

Michael Scott, MD, Departments of Emergency Medicine and Internal Medicine, University of Maryland Medical Center, Baltimore.

T. Andrew Windsor, MD, Department of Emergency Medicine, University of Maryland Medical Center, Baltimore.

Peer Reviewer:

Robert E. Falcone, MD, FACS, Clinical Professor of Surgery, The Ohio State University College of Medicine, Columbus.

**Winner
Best Instructional Reporting**

March/Apr 2011 Issue

Specialized Information
Publishers Association

Statement of Financial Disclosure

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Dietrich (editor in chief), Drs. Bond, Scott, and Windsor (authors), Dr. Falcone (peer reviewer), and Ms. Behrens (nurse reviewer) report no relationships with companies related to this field of study. Ms. Mark (executive editor), and Ms. Hamlin (managing editor) report no relationships with companies related to the field of study covered by this CME activity.

The Roles and Risks of Whole-Body Computed Tomography Scans in the Trauma Patient

Emergency departments in the United States are frequently confronted with trauma patients with varying degrees of injury. Clinically significant injuries may be missed, with devastating consequences for the patient. Concern for not missing any potentially serious injuries has led to an aggressive diagnostic approach with a goal of not missing any injuries. The CT scan has facilitated this approach, providing substantial information guiding management. However, CT scans have risks, especially when the pan-scan approach is used. The authors review the uses, advantages, and disadvantages of the pan-scan.

— Ann M. Dietrich, MD, Editor

Background

Emergency departments (EDs) in the United States received approximately 35 million trauma-related visits in 2007.¹ According to the Centers for Disease Control (CDC), unintentional injury accounted for 123,706 deaths in the same year, making it the fourth leading cause of death overall and the primary cause of death among people between 1 and 44 years of age.²

The trauma population constitutes a remarkably high-risk cohort for emergency care providers and trauma surgeons. The principles of Advanced Trauma Life Support (ATLS) aim to provide a simple and effective standardized approach for the assessment and care of injured patients. Between the patient with minor isolated trauma and the unstable patient with multi-trauma requiring immediate surgical intervention lies a complex group of various injury patterns that represents a significant gray area for any trauma care provider. Patients who are stable after initial clinical evaluation still might have serious injuries that require an expedient evaluation performed in an organized manner to avoid the significant morbidity and mortality associated with delays in localization and intervention.^{3,4} Approximately 15% to 22.3% of trauma patients in whom injuries are missed have injuries that are clinically significant.⁵ This prevalence of missed serious injury has led to an aggressive diagnostic approach with an emphasis on high sensitivity and early identification of all injuries. Initially, this aggressiveness was illustrated by the use of diagnostic peritoneal lavage (DPL), an invasive but highly sensitive diagnostic screening tool that decreased the number of missed intra-abdominal injuries. With the advent of computed tomography (CT), emergency care providers and trauma surgeons have increasingly relied upon this technology as an integral part of trauma evaluation and resuscitation.⁶ The “traditional” imaging strategy includes plain radiographs of the chest, pelvis, and lateral cervical spine (C-spine), in accordance with ATLS guidelines; a Focused Assessment with Sonography for Trauma (FAST) exam; and selected CT scans as deemed necessary based on the physical examination, radiographs, or ultrasound assessment.⁷

The first generations of CT scanners suffered from a significant lack of

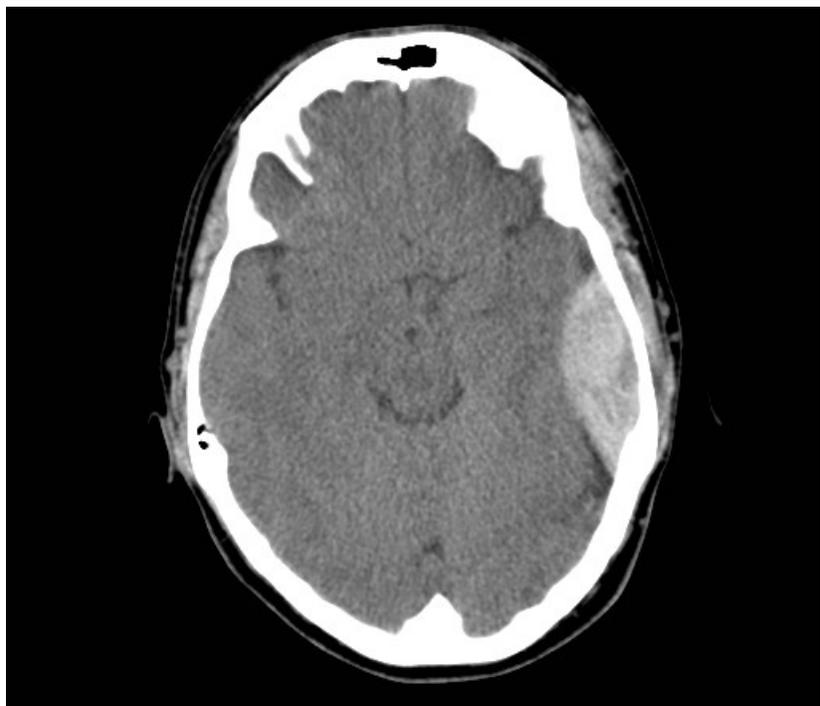
Executive Summary

- Approximately 15% to 22.3% of trauma patients in whom injuries are missed have injuries that are clinically significant. This prevalence of missed serious injury has led to an aggressive diagnostic approach with an emphasis on high sensitivity and early identification of all injuries.
- Multiple studies have validated the hypothesis that CT is superior in accuracy and reliability compared with physical examination, laboratory screening, plain radiographs, and sonography alone in the evaluation of most serious traumatic injuries.
- A typical pan-scan involves a non-contrast CT scan of the head and contrast-enhanced scans of the neck, chest, abdomen, and pelvis.
- Recent studies have shown the single-pass pan-scan to be a viable, if not superior, alternative to conventional segmental whole-body protocols. The single-pass technique has been found to be accurate and timesaving, reducing acquisition time by as much as 42.5% and decreasing radiation dose.

sensitivity and specificity and were limited by the amount of time required for the scan (separating the patient from the monitoring of the trauma resuscitation bay). The introduction of multi-slice CT scanners has both improved diagnostic accuracy and reduced the time of scanning significantly.⁸ Multiple studies have validated the hypothesis that CT is superior in accuracy and reliability compared with physical examination, laboratory screening, plain radiographs, and sonography alone in the evaluation of most serious traumatic injuries.⁹⁻¹⁶

The number of CTs performed in EDs for injury-related conditions nearly doubled between 1998 and 2007.⁶ CT now has a well-established role in the secondary evaluation of trauma, and the concept of a whole-body CT scan, or “pan-scan,” has become more accepted as an adjunct for definitive assessment of injuries during the early stages of trauma management.^{6,17,18} A typical pan-scan involves a non-contrast CT scan of the head (see Figure 1) and contrast-enhanced scans of the neck, chest, abdomen, and pelvis. Some protocols also include dedicated reconstructed views of the rest of the spine or other osseous structures. (See Figure 2.) Modern multi-detector CT scanners have the ability to produce specialized studies such as fine cuts through the facial, orbital, and temporal bones, as well as CT angiography of the body and extremities (these are not included in most

Figure 1. Epidural Hematoma



CT of a 26-year-old male on warfarin for a mechanical heart valve replacement. He fell from a ladder, sustaining an epidural hematoma. Image courtesy of University of Maryland School of Medicine Department of Emergency Medicine.

“standard” pan-scans).¹⁹ Naturally, since departmental policies and CT manufacturers differ, institutions tend to have slightly different whole-body CT protocols. Strategies for reducing scan time, improving image quality, and decreasing radiation exposure are being investigated.^{17,19,20}

Recent studies have shown the

single-pass pan-scan to be a viable, if not superior, alternative to conventional segmental whole-body protocols. A single-pass CT scan captures the neck and body portions in a single scan, usually with multi-phased contrast injection. Conventional pan-scans usually incorporate pauses and multiple overlapping scans to

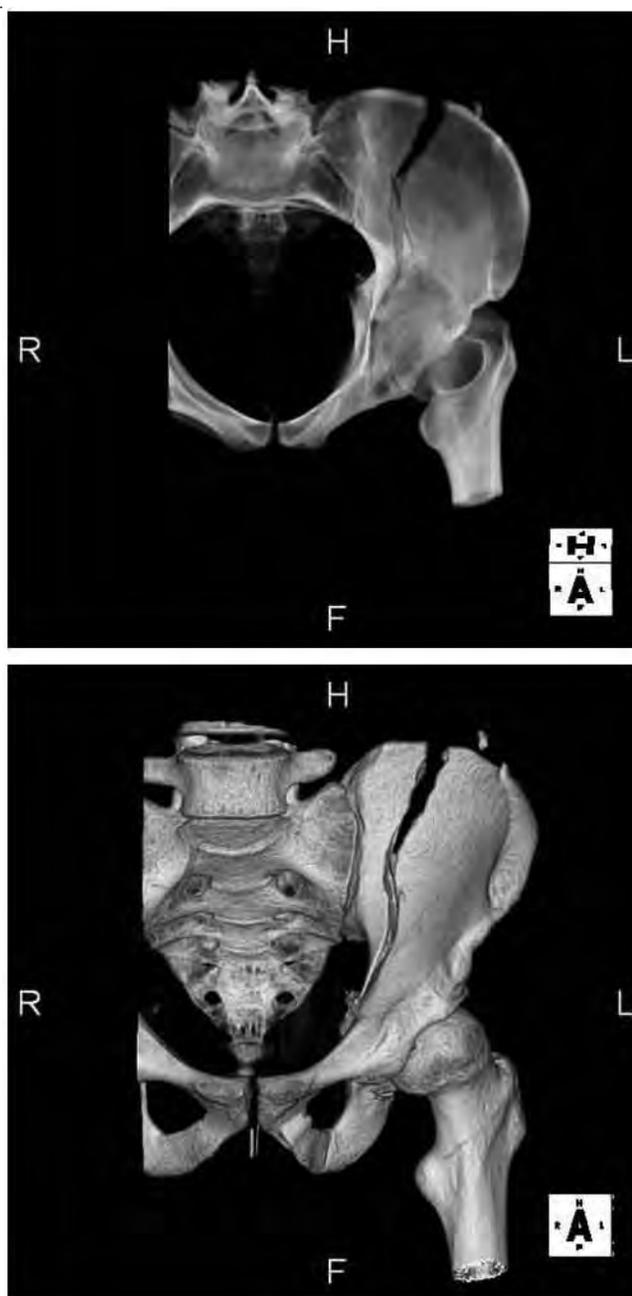
achieve separate portal and arterial phases. The single-pass technique has been found to be accurate and time-saving,^{17,21} reducing acquisition time by as much as 42.5%¹⁹ and decreasing radiation dose.²²

The immediate benefits of CT are easy to recognize: A significant amount of clinical information can be gained in a short period of time in a noninvasive manner, which aids in triaging, surgical planning, and disposition. Multiple studies have suggested that the sensitivity of CT has progressed to the point that a negative study can effectively eliminate the possibility of significant traumatic injuries, allowing patients who otherwise might have required observation for hours or days to be discharged home earlier.^{8,17,23-26} Few experts dispute the necessity of pan-scanning patients with significant physical evidence of multi-trauma (see Figure 3), those with massive blunt or penetrating injuries, and those in whom the physical examination is unreliable because of altered mental status, depressed level of consciousness, or significant distracting injuries.²⁷⁻²⁹ However, an ongoing debate focuses on the utility of the pan-scan as a standard part of the evaluation of patients with moderate trauma and of those without clinically evident injuries who have normal laboratory values and plain radiographs.^{30,31} The concerns are not without merit, as the pan-scan protocol poses several risks for the patient: radiation exposure, allergic reaction to contrast, contrast-induced nephropathy, and contrast extravasation.

Potential Benefits of Pan-Scan

Diagnostic Yield. As discussed above, the initial management of trauma patients involves an aggressive attempt to identify all injuries early. The use of whole-body CT for this purpose has been supported by studies that suggest that a pan-scan identifies more injuries and leads to a change in management more often than following selective CT protocols.^{18,32} In the study by Salim and

Figure 2. Iliac Wing Fracture

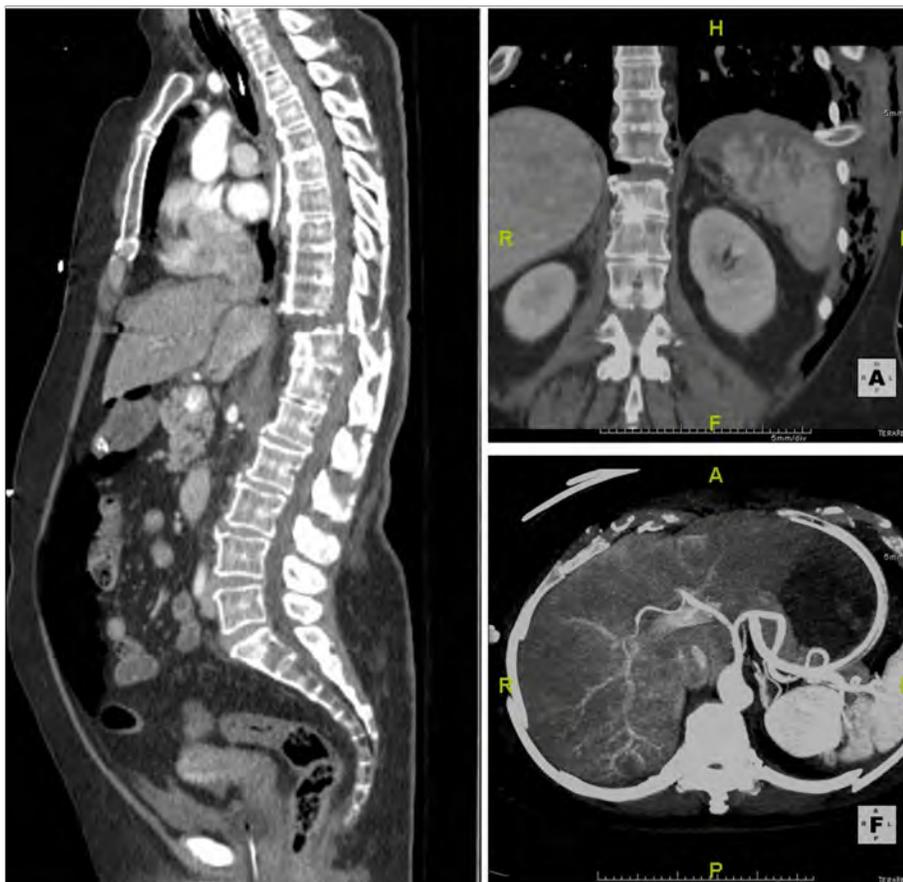


Two versions of 3D reconstructions of an iliac wing fracture in a 24-year-old male who was injured in a high-speed motor vehicle crash. Images courtesy of University of Maryland School of Medicine Department of Emergency Medicine.

colleagues,¹⁸ 18.9% of patients had a change in management as a result of a finding on whole-body CT, including earlier discharge and procedural or surgical intervention. The study's data analysis focused on the

abdominal portion of the pan-scan and found that 20.3% of patients who had a normal abdominal examination had a change in management after abdominal CT. Six of these patients required laparotomy. These

Figure 3. Pan-scan of Serious Injuries



In this 64-year-old woman who was involved in a high-speed motor vehicle crash, pan-scan identified a number of serious injuries, including spinal fracture/dislocation, grade 3 splenic laceration, and grade 5 liver injury. Image courtesy of University of Maryland School of Medicine Department of Emergency Medicine.

results suggest that CT can identify injuries in patients with normal results on the physical examination. In another study, 18 emergency physicians' clinical judgment showed relatively high sensitivity (69.9% to 100%) in excluding injuries without a pan-scan if a patient's pretest probability of injury was "very low."³³ The sensitivity of excluding injuries in specific body regions steadily decreased with higher pretest probabilities of injury, supporting the idea that the accuracy of clinician judgment worsens in the assessment of severely injured patients.

Multiple studies have failed to develop a clinical decision rule that

could completely exclude all types of intra-abdominal injuries after blunt trauma without performing CT.^{15,25,34} In two of these studies, the presence of abdominal pain or tenderness achieved 100% sensitivity in detecting intra-abdominal injuries requiring surgical intervention,^{25,30,34} even though it missed non-surgical injuries. Because the primary outcome of these studies was identifying all injuries, the clinical decision rules were viewed as failures. This illustrates two viewpoints as to what is the most important endpoint when evaluating the use of CT in trauma. Should the endpoint be finding any injury, or should it be finding only injuries that

require surgical or medical intervention? Do clinicians really need to know about an injury that does not require treatment?

Gupta and colleagues presented a study that further illustrates this debate.³⁵ They polled emergency physicians and trauma surgeons about which components of the pan-scan obtained for individual trauma patients they thought were necessary. All scans were ordered at the discretion of the trauma surgeon. The ED physicians would have ordered 35% fewer scans, but in doing so would have missed 10% of injuries. However, only 0.3% of these injuries would have led to a predefined critical action. This suggests that although CT is superior in identifying objective injuries that the physical examination and clinical suspicion might miss, very few of these injuries prove to be emergently dangerous. Of note, the authors had difficulty agreeing on the true importance of the abnormal findings. The emergency medicine authors thought the projected miss rate of 0.3% (the number of missed injuries that would have required predefined critical actions) supported a more selective use of CT based on physician judgment. The trauma surgeon authors pointed to the projected missed injury rate of 10% (the number of all missed injuries, regardless of requiring a critical action or not) as justification for more liberal use of CT.

Survival/Mortality. Although most of these studies have used injuries identified on CT or change in management based on CT as the primary outcome, some studies have indicated a possible decrease in the mortality rate with a liberal CT approach. Hutter et al³⁶ studied the effect on survival before and after the institution of a liberal pan-scan policy at a major high-volume trauma center in Germany. The study included patients who did not undergo a pan-scan due to the unavailability of the method, patients who were eligible but were not pan-scanned due to physician discretion, and eligible patients who underwent a pan-scan. Patients who actually underwent a

pan-scan had a statistically significant reduction in overall mortality, with an odds ratio (OR) of 0.17 (95% CI, 0.1–0.28) and a total risk difference of 7%. The relative impact of a patient receiving a pan-scan on the mortality rate was small compared with the effect of Injury Severity Score (ISS) or neurologic function.

Huber-Wagner and associates³⁷ suggested that the use of whole-body CT in the management of trauma patients increased the probability of survival compared with the predicted mortality rate based on the Trauma Injury Severity Score (TRISS) and Revised Injury Severity Classification (RISC) scores. This study has been criticized because more patients in the whole-body CT group were treated at trauma centers, and it was acknowledged that the predicted mortality rate for the whole-body CT group was likely increased due to clinically insignificant findings that would in turn elevate the ISS.³⁸ Van Vugt et al³⁹ demonstrated that clinically insignificant findings found on CT do indeed elevate the ISS, artificially inflating mortality rate estimates beyond the true mortality rate, potentially altering the statistical importance of the pan-scan on survival. As overall survival in trauma generally continues to trend upward,³⁷ it is important to keep in mind that these observed effects are likely caused by complex systemic changes and that assigning a causal relationship to any one intervention is probably not appropriate.

Potential Pitfalls of Pan-Scan

Because the rates of CT use in EDs have skyrocketed in recent years, more attention is being directed toward the weaknesses of this diagnostic modality.⁶

Contrast-Induced Nephropathy (CIN). Published reports contain significant variability regarding the effect of intravenous (IV) contrast for CT on renal function. The incidence of CIN, most commonly defined as an increase in creatinine of 0.5 mg/dL or 25% from the baseline creatinine level, has been

Table 1. Average Adult Effective Doses of Various Radiologic Studies

Radiograph	Average Effective Dose (mSv)
Single chest	0.02
Cervical spine	0.1
Thoracic spine	1.0
Lumbar spine	1.5
Pelvis	0.6
CT	Average Effective Dose (mSv)
Head	2
Neck	3
Chest	7
Abdomen	8
Pelvis	6
Spine	6

Adapted from Mettler FA Jr, Huda W, Yoshizumi TT, Mahesh M. Effective doses in radiology and diagnostic nuclear medicine: A catalog. *Radiology* 2008;248:254-263.

estimated to be anywhere from 0% to 12%, depending on the study and the underlying risk factors of the patients.⁴⁰⁻⁴⁶ Importantly, it remains unclear whether IV contrast is the actual cause of the rise in creatinine. One literature review noted that in two studies in which control groups of patients did not receive IV contrast, no association was found between IV contrast and a rise in the serum creatinine level.⁴⁷⁻⁴⁹

Research into the true risks of IV contrast is ongoing. Mitchell and Kline found an incidence of CIN of 11% in a population of 633 general ED patients who underwent contrast-enhanced CT.⁴⁴ Of the 70 patients in whom CIN developed, renal failure developed in 7 (1% of the overall population), defined as an increase in serum creatinine of 3 mg/dL or more. Six of those seven patients died, and in four cases it was believed that renal failure significantly contributed to death.

Unfortunately, there is not a large body of literature investigating renal failure after contrast CT in the trauma patient. However, Matsushima and colleagues⁵⁰ did find an ISS of 16 or greater to be a risk factor for contrast-induced acute

kidney injury, although the dose of contrast received was not associated with an increased risk, suggesting the possibility of an association rather than causality.

Overall, although there is still controversy about CIN and the exact risks associated with acute renal failure, especially in regard to the trauma population, the consensus seems to be that there is a small but very real risk of CIN following CT with IV contrast, a point all providers should consider when ordering CT scans.

Cancer Risk. CT is responsible for more than 70% of medical radiation exposure; 16.2 million scans were ordered for ED patients in 2007.^{51,52} Brenner and Hall estimated that 1.5% to 2.0% of all cancers in the United States might be attributable to ionizing radiation from CT, including many types of scans other than those done during trauma assessments. The probable death rate is much lower, at about 0.1 to 0.35%.^{38,53,54} These and other estimates are not universally accepted because they are based on a linear no-threshold relationship that assumes the incidence of cancer induction is proportional to

Table 2. Intermediate Level Trauma Patients (per MIEMSS* protocols)

<p>Category B</p> <ul style="list-style-type: none"> • Glasgow Coma Scale (GCS) score 9-14 • Paralysis or vascular compromise of limb • Amputation proximal to wrist or ankle • Crushed, degloved, or mangled extremity • Penetrating injuries to extremities proximal to elbow or knee • Combination trauma with burns
<p>Category C</p> <ul style="list-style-type: none"> • Age < 5 years or > 55 years • Patient with bleeding disorder or patient on anticoagulants • Dialysis patient • Pregnancy > 20 weeks • EMS provider judgment • High-risk auto crash <ul style="list-style-type: none"> • Intrusion > 12 in. occupant site; > 18 in. any site • Ejection (partial or complete) from vehicle • Death in same passenger compartment • Vehicle telemetry data consistent with high risk of injury • Exposure to blast or explosion • Falls greater than 3 times patient's height <p>Intermediate trauma classifications based on the trauma decision tree protocols promulgated by the Maryland Institute for Emergency Medical Services Systems. Please refer to local trauma classifications and regulations for management in other jurisdictions. Adapted from <i>The Maryland Medical Protocols for EMS Providers</i>. Baltimore, Maryland: MIEMSS, 2011.</p>

exposure, which some argue is not congruent with biological and animal data.⁵⁵ Most models are based on data extrapolated from atomic bomb survivors, but there has been debate about the level of radiation that leads to an increased cancer risk. A full explanation of this debate is beyond the scope of this article, but some have concluded that there is no significant carcinogenic risk with a dose up to 150 to 200 millisieverts (mSv) to normal tissues,^{55,56} while others estimate that the safe dose is less than 100 mSv.⁵³ The sievert (Sv) is a measure of the effective dose of radiation on biological tissues based on the stochastic effect of ionizing radiation. The average underlying exposure from everyday background radiation is about 3 mSv per year.⁵⁴ Most patients receive an effective dose of about 20 to 50 mSv

from a single pan-scan,^{54,57} depending on the scanner power and scan technique. Table 1 includes a list of the average effective doses from individual radiologic studies.⁵⁸

The risk of death from severe trauma has been estimated to be 50 to 100 times higher than the risk of a cancer death from CT-related radiation exposure.^{59,60} In the trauma population, use of whole-body CT is usually not questioned because of the significant risk-benefit ratio. The population of particular interest and debate, however, has intermediate-level trauma, such as category B or C patients (*see Table 2*) or priority II patients; in other words, those who have potentially life-threatening, but not immediately life-endangering injuries. In this subset of patients, which has not been well-studied, trauma-related mortality is still a

real concern, estimated between 0.6% and 2%,^{38,57} but there is a larger proportion without serious injuries. In a recent study of 642 adult intermediate-level trauma patients, Laack and colleagues estimated that the risk of trauma-related mortality was six times greater than the cancer risk.³⁸ Trauma-related mortality was highest in older patients, and the risk of cancer death was inversely proportional to age; therefore, the youngest patients have the most potential danger from radiation — patients younger than 20 years have four times the estimated risk of those older than 60 years. (*See Figure 4.*) It is notable that no one younger than 80 died in Laack's study, and all deaths were caused by head injuries. The mortality rate and median ISS were relatively low compared with the findings in a smaller study by Winslow and associates, who examined the amount of radiation to which intermediate trauma patients were exposed.⁵⁷

Pediatrics. Because children are more susceptible to the carcinogenic effects of ionizing radiation, efforts to reduce unnecessary radiation exposure are paramount. CT remains the diagnostic test of choice for evaluation of blunt trauma in children, but the risk of inducible cancer is much higher than in adults. Mueller et al⁶¹ showed that the effective radiation dose during CT is on par with adults, but doses to organs such as the thyroid gland fell within the range of radiation doses historically correlated with increased cancer risk. Based on a model by Berrington de Gonzalez and associates, the mean lifetime cancer risk after whole-body CT in 3-year-old boys and girls is 1 in 133 and 1 in 166, respectively.⁶² At 15 years of age, the risks were estimated at 1 in 250 for girls and 1 in 500 for boys.

Although the FAST exam is well-established in adult trauma, its utility as a screening exam in pediatric trauma is not universally supported.^{7,26,63} CT has been shown to be sensitive for identification of injuries in children, similar to adults. An important consideration for pediatric

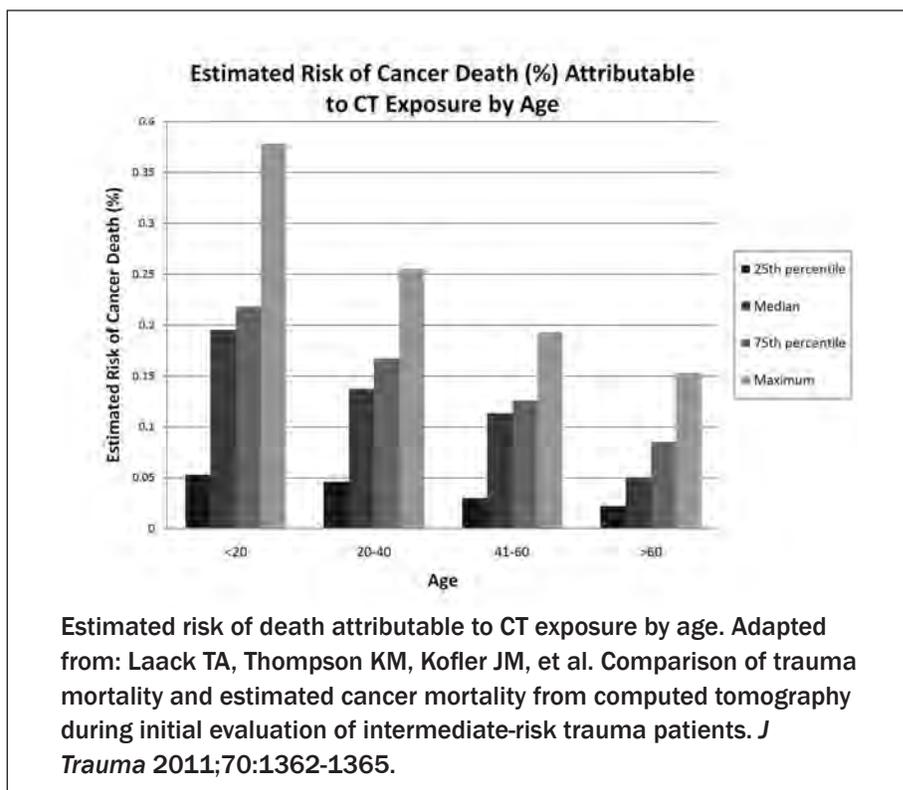
trauma, however, is that solid organ injuries in children are being treated increasingly non-operatively,^{64,65} so discovery of these injuries in hemodynamically stable children does not automatically lead to surgical intervention.⁶⁶ On the other hand, a review of three prospective studies looking at intra-abdominal injury determined that the negative predictive value (NPV) of abdominal CT was 99.8%, suggesting that routine admission and serial exams after a normal abdominal CT and a normal physical exam may not be necessary.⁶⁸

Clinical decision rules for obtaining a CT scan have been successful for pediatric head injury^{68,69} and C-spine injury,⁷⁰ and are being investigated for abdominal trauma.⁶⁶ Unfortunately, just as with adults, there is no clinical decision rule for using pan-scan in the pediatric trauma population. Use of a pan-scan is not routinely recommended in stable children without abnormal physical exam findings or a mechanism that induces concern for significant injury. Selective scanning of body areas, rather than whole-body scanning, results in a statistically significant decrease in all organ doses and total effective dose.⁶¹

Other Considerations

An Imperfect Test. While CT has been shown to have generally superior ability to identify injuries, it is not a perfect test. Alone, CT has demonstrated insufficient sensitivity to rule out diaphragm injury, although its positive predictive value appears to be very good.⁷¹ (See *Figure 5*.) Similar concern has been expressed for other radiographically occult injuries such as diffuse axonal injury, hollow viscus injuries, and mesenteric injuries, but Tan showed that patients with surgically confirmed hollow viscus and mesenteric injuries were very likely to have had an abnormal CT scan.⁷² Positive trauma scans are conclusive, but negative results require subsequent confirmation.¹⁷ Injuries initially missed on CT are uncommon; while these falsely negative scans can sometimes

Figure 4. Risk of Death and CT Exposure

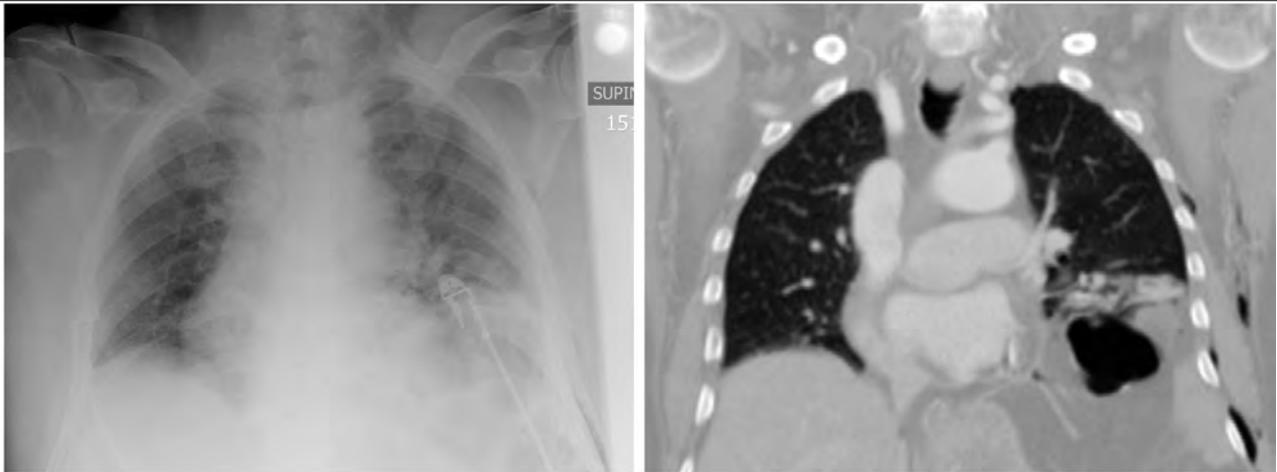


delay management, they have not yet been shown to affect the mortality rate.⁷³ In that vein, second readings are advocated to avoid missed injuries not seen on the initial preliminary “hot read,”⁷⁴ at least in patients with intermediate to high pretest probability for injury.

Imaging Prior to Transfer. Emergency medicine practitioners who do not work at designated trauma centers will certainly be responsible for the occasional trauma patient, and if the patient meets criteria for transfer to a trauma center, the question of whether to obtain imaging prior to transfer may arise. From a practical standpoint, the role of the practitioner is to stabilize the patient to the best of his or her abilities and to facilitate transport to the receiving facility for definitive management as soon as possible. As noted previously, CT is sensitive for diagnosing life-threatening injuries and can be used as a tool for determining disposition after trauma.¹⁸ In a stable trauma patient with

clinically suspected injuries based on mechanism or examination, CT might elucidate the need for further specialized management or it can obviate the need for admission or transfer. If a patient meets criteria for transfer prior to imaging, it is important to communicate with the receiving facility regarding the expectation of, or necessity for, imaging at the referring center. However, if a patient clinically requires transfer to a trauma center, sophisticated diagnostic studies may help with eventual management but should not delay transfer. Between 53% and 58% of transferred patients receive repeat imaging upon arrival at the receiving trauma center.^{35,75} The reasons for repeating studies are varied, including change of clinical status during transfer, inadequate original technique, software incompatibility, and even simple human error such as misplacing or forgetting to send the original scans. It is notable that patients who received repeat imaging tended to be more severely injured

Figure 5. Pan-scan Showing Rib Fractures, Left Hemidiaphragm Injury, and Large Flank Hematoma



Following a motor vehicle crash, a clinically stable 84-year-old man complained of mild left flank pain. His wife died as a result of the crash. Pan-scan revealed a number of rib fractures, a left hemidiaphragm injury, and large flank hematoma, which were not evident on plain radiograph and FAST examination. Images courtesy of University of Maryland School of Medicine Department of Emergency Medicine.

but also suffered longer delays⁷⁵ as well as additional radiation and financial charges. Lastly, incidental findings are found in up to one-third of trauma patients,³⁹ and trauma patients are notoriously often lost to follow-up,⁷⁶ which begs the question of who will arrange follow-up on these patients if incidental abnormalities are found during a formal over read after transfer.

Recommendations

Unfortunately, there is no universally accepted algorithm regarding routine whole-body CT compared with selective CT. A hospital could benefit from the development of a standardized CT protocol. Standardizing the radiologic workup of trauma patients needs to balance the risks of complications, cost, and incidental findings from CT versus the risk of missed injuries, repeat imaging, and delay in disposition or treatment.

Pan-scan is a noninvasive and effective method of injury determination in the hemodynamically stable trauma population, but it must be used with an awareness of its associated problems. Indiscriminate use

without proper clinical evaluation or concern is inappropriate. Efforts to reduce radiation exposure should be focused toward younger patients and those with obviously minor injuries or trauma mechanisms. Pan-scanning may reduce but does not eliminate the incidence of missed injuries and is not a substitute for a thorough clinical evaluation, appropriate repeat examinations, and follow-up. Single-pass pan-scans reduce scan time and radiation compared with conventional sequential imaging. Comprehensive secondary reading of a scan after a preliminary read may lower the rate of missed injuries.

Further research is needed. Currently in progress is the REACT-2 trial, a prospective, multi-center, multi-national study investigating the effects of immediate pan-scan CT during the primary survey on clinical outcomes compared with the use of conventional imaging and selective CT. The intervention group undergoes immediate whole-body CT, completely eliminating plain radiographs and the FAST exam. This is a novel approach and is focusing on the primary outcome of in-hospital mortality as well as

secondary endpoints such as effects on morbidity, radiation exposure, and cost-effectiveness. It is hoped that this study, and those to follow, will delineate an appropriately balanced diagnostic approach for trauma patients.

References

1. Niska R, Bhuiya F, Xu J. National Hospital Ambulatory Medical Care Survey: 2007 Emergency Department Summary. *Natl Health Stat Report* 2010;1-31.
2. CDC. Ten Leading Causes of Death and Injury. 2007; <http://www.cdc.gov/injury/wisqars/LeadingCauses.html>. Accessed May 5, 2012.
3. Fakhry SM, Brownstein M, Watts DD, et al. Relatively short diagnostic delays (< 8 hours) produce morbidity and mortality in blunt small bowel injury: An analysis of time to operative intervention in 198 patients from a multicenter experience. *J Trauma* 2000;48:408-415.
4. Malinoski DJ, Patel MS, Yakar DO, et al. A diagnostic delay of 5 hours increases the risk of death after blunt hollow viscus injury. *J Trauma* 2010;69:84-87.
5. Pfeifer R, Pape HC. Missed injuries in trauma patients: A literature review. *Patient Saf Surg* 2008;2:20.
6. Korley FK, Pham JC, Kirsch TD. Use of advanced radiology during visits to US emergency departments for injury-related conditions, 1998-2007. *JAMA* 2010;304:1465-1471.

7. *Advanced Trauma Life Support for Doctors ATLS: Manuals for Coordinators and Faculty*. Eighth ed. Chicago, IL: American College of Surgeons; 2008.
8. Rieger M, Czermak B, El Attal R, et al. Initial clinical experience with a 64-MDCT whole-body scanner in an emergency department: Better time management and diagnostic quality? *J Trauma* 2009;66:648-657.
9. Demetriades D, Gomez H, Velmahos GC, et al. Routine helical computed tomographic evaluation of the mediastinum in high-risk blunt trauma patients. *Arch Surg* 1998;133:1084-1088.
10. Exadaktylos AK, Sclabas G, Schmid SW, et al. Do we really need routine computed tomographic scanning in the primary evaluation of blunt chest trauma in patients with "normal" chest radiograph? *J Trauma* 2001;51:1173-1176.
11. Gestring ML, Gracias VH, Feliciano MA, et al. Evaluation of the lower spine after blunt trauma using abdominal computed tomographic scanning supplemented with lateral scanograms. *J Trauma* 2002;53:9-14.
12. Guillaumondegui OD, Pryor JP, Gracias VH, et al. Pelvic radiography in blunt trauma resuscitation: A diminishing role. *J Trauma* 2002;53:1043-1047.
13. Hauser CJ, Visvikis G, Hinrichs C, et al. Prospective validation of computed tomographic screening of the thoracolumbar spine in trauma. *J Trauma* 2003;55:228-235.
14. Griffen MM, Frykberg ER, Kerwin AJ, et al. Radiographic clearance of blunt cervical spine injury: Plain radiograph or computed tomography scan? *J Trauma* 2003;55:222-227.
15. Poletti PA, Mirvis SE, Shanmuganathan K, et al. Blunt abdominal trauma patients: Can organ injury be excluded without performing computed tomography? *J Trauma* 2004;57:1072-1081.
16. Brown CV, Antevil JL, Sise MJ, et al. Spiral computed tomography for the diagnosis of cervical, thoracic, and lumbar spine fractures: Its time has come. *J Trauma* 2005;58:890-896.
17. Stengel D, Ottersbach C, Matthes G, et al. Accuracy of single-pass whole-body computed tomography for detection of injuries in patients with major blunt trauma. *CMAJ* March 5, 2012 [Epub ahead of print].
18. Salim A, Sangthong B, Martin M, et al. Whole body imaging in blunt multisystem trauma patients without obvious signs of injury: Results of a prospective study. *Arch Surg* 2006;141:468-475.
19. Nguyen D, Platon A, Shanmuganathan K, et al. Evaluation of a single-pass continuous whole-body 16-MDCT protocol for patients with polytrauma. *AJR Am J Roentgenol* 2009;192:3-10.
20. Loupatatzis C, Schindera S, Gralla J, et al. Whole-body computed tomography for multiple traumas using a triphasic injection protocol. *Eur Radiol* 2008;18:1206-1214.
21. Gralla J, Spycher F, Pignolet C, et al. Evaluation of a 16-MDCT scanner in an emergency department: Initial clinical experience and workflow analysis. *AJR Am J Roentgenol* 2005;185:232-238.
22. Fanucci E, Fiaschetti V, Rotili A, et al. Whole body 16-row multislice CT in emergency room: effects of different protocols on scanning time, image quality and radiation exposure. *Emerg Radiol* 2007;13:251-257.
23. Livingston DH, Lavery RF, Passannante MR, et al. Admission or observation is not necessary after a negative abdominal computed tomographic scan in patients with suspected blunt abdominal trauma: Results of a prospective, multi-institutional trial. *J Trauma* 1998;44:273-282.
24. Livingston DH, Lavery RF, Passannante MR, et al. Emergency department discharge of patients with a negative cranial computed tomography scan after minimal head injury. *Ann Surg* 2000;232:126-132.
25. Richards JR, Derlet RW. Computed tomography for blunt abdominal trauma in the ED: A prospective study. *Am J Emerg Med* 1998;16:338-342.
26. Holmes JF, Gladman A, Chang CH. Performance of abdominal ultrasonography in pediatric blunt trauma patients: A meta-analysis. *J Pediatr Surg* 2007;42:1588-1594.
27. Pal JD, Victorino GP. Defining the role of computed tomography in blunt abdominal trauma: Use in the hemodynamically stable patient with a depressed level of consciousness. *Arch Surg* 2002;137:1029-1033.
28. Ferrera PC, Verdile VP, Bartfield JM, et al. Injuries distracting from intra-abdominal injuries after blunt trauma. *Am J Emerg Med* 1998;16:145-149.
29. Self ML, Blake AM, Whitley M, et al. The benefit of routine thoracic, abdominal, and pelvic computed tomography to evaluate trauma patients with closed head injuries. *Am J Surg* 2003;186:609-614.
30. Snyder GE. Whole-body imaging in blunt multisystem trauma patients who were never examined. *Ann Emerg Med* 2008;52:101-103.
31. Tillou A, Gupta M, Baraff LJ, et al. Is the use of pan-computed tomography for blunt trauma justified? A prospective evaluation. *J Trauma* 2009;67:779-787.
32. Deunk J, Brink M, Dekker HM, et al. Routine versus selective computed tomography of the abdomen, pelvis, and lumbar spine in blunt trauma: A prospective evaluation. *J Trauma* 2009;66:1108-1117.
33. Smith CB, Barrett TW, Berger CL, et al. Prediction of blunt traumatic injury in high-acuity patients: Bedside examination vs computed tomography. *Am J Emerg Med* 2011;29:1-10.
34. Richards JR, Derlet RW. Computed tomography and blunt abdominal injury: Patient selection based on examination, haematocrit and haematuria. *Injury* 1997;28:181-185.
35. Gupta R, Greer SE, Martin ED. Inefficiencies in a rural trauma system: The burden of repeat imaging in interfacility transfers. *J Trauma* 2010;69:253-255.
36. Hutter M, Woltmann A, Hierholzer C, et al. Association between a single-pass whole-body computed tomography policy and survival after blunt major trauma: A retrospective cohort study. *Scand J Trauma Resusc Emerg Med* 2011;19:73.
37. Huber-Wagner S, Lefering R, Qvick LM, et al. Effect of whole-body CT during trauma resuscitation on survival: A retrospective, multicentre study. *Lancet* 2009;373:1455-1461.
38. Laack TA, Thompson KM, Kofler JM, et al. Comparison of trauma mortality and estimated cancer mortality from computed tomography during initial evaluation of intermediate-risk trauma patients. *J Trauma* 2011;70:1362-1365.
39. van Vugt R, Deunk J, Brink M, et al. Influence of routine computed tomography on predicted survival from blunt thoracoabdominal trauma. *Eur J Trauma Emerg Surg* 2011;37:185-190.
40. Bell GW, Edwardes M, Dunning AM, et al. Periprocedural safety of 64-detector row coronary computed tomographic angiography: Results from the prospective multicenter ACCURACY trial. *J Cardiovasc Comput Tomogr* 2010;4:375-380.
41. Kim SM, Cha RH, Lee JP, et al. Incidence and outcomes of contrast-induced nephropathy after computed tomography in patients with CKD: A quality improvement report. *Am J Kidney Dis* 2010;55:1018-1025.
42. Lencioni R, Fattori R, Morana G, et al. Contrast-induced nephropathy in patients undergoing computed tomography (CONNECT) — A clinical problem in daily practice? A multicenter observational study. *Acta Radiol* 2010;51:741-750.
43. Mitchell AM, Jones AE, Tumlin JA, et al. Incidence of contrast-induced nephropathy after contrast-enhanced computed tomography in the outpatient setting. *Clin J Am Soc Nephrol* 2010;5:4-9.
44. Mitchell AM, Kline JA. Contrast nephropathy following computed tomography angiography of the chest for pulmonary embolism in the emergency department. *J Thromb Haemost* 2007;5:50-54.
45. Rashid AH, Brieve JL, Stokes B. Incidence of contrast-induced nephropathy in intensive care patients undergoing computerised tomography and prevalence of risk factors. *Anaesth Intensive Care* 2009;37:968-975.
46. Weisbord SD, Mor MK, Resnick AL, et al. Incidence and outcomes of contrast-induced AKI following computed tomography. *Clin J Am Soc Nephrol* 2008;3:1274-1281.
47. Rao QA, Newhouse JH. Risk of nephropathy after intravenous administration of contrast material: A critical literature analysis. *Radiology* 2006;239:392-397.

48. Cramer BC, Parfrey PS, Hutchinson TA, et al. Renal function following infusion of radiologic contrast material: A prospective controlled study. *Arch Intern Med* 1985;145:87-89.
49. Heller CA, Knapp J, Halliday J, et al. Failure to demonstrate contrast nephrotoxicity. *Med J Aust* 1991;155:329-332.
50. Matsushima K, Peng M, Schaefer EW, et al. Posttraumatic contrast-induced acute kidney injury: Minimal consequences or significant threat? *J Trauma* 2011;70:415-420.
51. Martin DR, Semelka RC. Health effects of ionising radiation from diagnostic CT. *Lancet* 2006;367:1712-1714.
52. Larson DB, Johnson LW, Schnell BM, et al. National trends in CT use in the emergency department: 1995-2007. *Radiology* 2011;258:164-173.
53. Brenner DJ, Hall EJ. Computed tomography — an increasing source of radiation exposure. *N Engl J Med* 2007;357:2277-2284.
54. Sharma OP, Oswanski MF, Sidhu R, et al. Analysis of radiation exposure in trauma patients at a level I trauma center. *J Emerg Med* 2011;41:640-648.
55. Tubiana M, Feinendegen LE, Yang C, et al. The linear no-threshold relationship is inconsistent with radiation biologic and experimental data. *Radiology* 2009;251:13-22.
56. Heidenreich WF, Paretzke HG, Jacob P. No evidence for increased tumor rates below 200 mSv in the atomic bomb survivors data. *Radiat Environ Biophys* 1997;36:205-207.
57. Winslow JE, Hinshaw JW, Hughes MJ, et al. Quantitative assessment of diagnostic radiation doses in adult blunt trauma patients. *Ann Emerg Med* 2008;52:93-97.
58. Mettler FA Jr, Huda W, Yoshizumi TT, et al. Effective doses in radiology and diagnostic nuclear medicine: A catalog. *Radiology* 2008;248:254-263.
59. Tien HC, Tremblay LN, Rizoli SB, et al. Radiation exposure from diagnostic imaging in severely injured trauma patients. *J Trauma* 2007;62:151-156.
60. Ott M, McAlister J, VanderKolk WE, et al. Radiation exposure in trauma patients. *J Trauma* 2006;61:607-610.
61. Mueller DL, Hatab M, Al-Senan R, et al. Pediatric radiation exposure during the initial evaluation for blunt trauma. *J Trauma* 2011;70:724-731.
62. Berrington de Gonzalez A, Mahesh M, Kim KP, et al. Projected cancer risks from computed tomographic scans performed in the United States in 2007. *Arch Intern Med* 2009;169:2071-2077.
63. Fox JC, Boysen M, Gharahbaghian L, et al. Test characteristics of focused assessment of sonography for trauma for clinically significant abdominal free fluid in pediatric blunt abdominal trauma. *Acad Emerg Med* 2011;18:477-482.
64. Feigin E, Aharonson-Daniel L, Savitsky B, et al. Conservative approach to the treatment of injured liver and spleen in children: Association with reduced mortality. *Pediatr Surg Int* 2009;25:583-586.
65. Davies DA, Pearl RH, Ein SH, et al. Management of blunt splenic injury in children: Evolution of the nonoperative approach. *J Pediatr Surg* 2009;44:1005-1008.
66. Schonfeld D, Lee LK. Blunt abdominal trauma in children. *Curr Opin Pediatr* 2012;24:314-318.
67. Hom J. The risk of intra-abdominal injuries in pediatric patients with stable blunt abdominal trauma and negative abdominal computed tomography. *Acad Emerg Med* 2010;17:469-475.
68. Kuppermann N, Holmes JF, Dayan PS, et al. Identification of children at very low risk of clinically-important brain injuries after head trauma: A prospective cohort study. *Lancet* 2009;374:1160-1170.
69. Osmond MH, Klassen TP, Wells GA, et al. CATCH: A clinical decision rule for the use of computed tomography in children with minor head injury. *CMAJ* 2010;182:341-348.
70. Viccellio P, Simon H, Pressman BD, et al. A prospective multicenter study of cervical spine injury in children. *Pediatrics* 2001;108:E20.
71. Allen TL, Cummins BF, Bonk RT, et al. Computed tomography without oral contrast solution for blunt diaphragmatic injuries in abdominal trauma. *Am J Emerg Med* 2005;23:253-258.
72. Tan KK, Liu JZ, Go TS, et al. Computed tomography has an important role in hollow viscus and mesenteric injuries after blunt abdominal trauma. *Injury* 2010;41:475-478.
73. Agostini C, Durieux M, Milot L, et al. Value of double reading of whole body CT in polytrauma patients [article in French]. *J Radiol* 2008;89:325-330.
74. Eurin M, Haddad N, Zappa M, et al. Incidence and predictors of missed injuries in trauma patients in the initial hot report of whole-body CT scan. *Injury* 2012;43:73-77.
75. Haley T, Ghaemmaghami V, Loftus T, et al. Trauma: The impact of repeat imaging. *Am J Surg* 2009;198:858-862.
76. Malhotra AK, Martin N, Jacoby M, et al. What are we missing: Results of a 13-month active follow-up program at a level I trauma center. *J Trauma* 2009;66:1696-1703.
- C. single-pass pan-scan
D. no imaging; observation only
2. Which of these patients would most benefit from a pan-scan?
A. any blunt trauma patient, regardless of mechanism
B. a hemodynamically stable patient injured in a motor vehicle crash, with no obvious physical findings, whose passenger died on scene
C. a hemodynamically stable 8-year-old boy with no physical findings after falling from a 5-foot ladder
D. a hemodynamically stable pedestrian struck at 10 mph with right knee and hip pain
3. Which of the following statements regarding contrast-induced nephropathy (CIN) in patients undergoing enhanced CT after trauma is correct?
A. The incidence may be as high as 12%, although this may be an associative rather than causative effect.
B. CIN is defined as an increase of 25% or 0.5 mg/dL in creatinine from baseline.
C. An Injury Severity Score of 16 or higher may be a risk factor for CIN.
D. The higher the dose of contrast administered, the higher the risk of CIN.
E. All of the above
F. Only A, B, and C are correct.
4. You are a physician at a community hospital and have just placed a chest tube to stabilize a woman who was injured in a motor vehicle collision. She arrived with a pneumothorax caused by multiple rib fractures. The FAST exam is positive, and you have to transfer her to the nearest trauma center for surgery. What is the most appropriate approach to imaging?
A. The patient is stable, so send her to CT for pan-scan.
B. The patient is stable, but imaging should not delay definitive management; transfer her immediately.
C. Scan the chest and abdomen, then transfer with accompanying images.
D. Send the patient to CT, because the receiving facility will probably want a scan.
5. Regarding pediatric blunt trauma patients, which of the following is true?
A. Children can be unreliable during history and physical, so they should be pan-scanned.
B. The FAST exam is a reliable way to rule out surgical abdominal injuries in stable children without abdominal pain.
C. Children receive the same effective radiation doses from CT as adults, but these doses carry a higher risk of secondary cancer.
D. Pan-scan is a good way to rule out surgical injuries for all pediatric trauma patients.
6. Which of the following statements about lifetime cancer risk associated with pan-scan is true?
A. The risk of trauma-related mortality is approximately six times that

CME/CNE Questions

1. Of the following, what is the most appropriate choice for a 48-year-old pedestrian who was struck by a motor vehicle with a moderate impact, is hemodynamically stable, and has no major physical exam findings except chest wall tenderness and a Glasgow Coma Scale (GCS) score of 13?
A. separate head and chest CT scans
B. head CT scan and chest radiograph only

- of CT-related cancer mortality in all-comer intermediate-level trauma patients.
- B. Age is a factor in CT-related cancer risk, and the risk is proportional to age.
 - C. The mean lifetime cancer risk after whole body CT in 3-year-old boys is 1 in 133 (0.75%) and 1 in 166 (0.60%) in girls.
 - D. The background radiation received on a yearly basis in an urban environment is almost as much as a single pan-scan.
 - E. B and D are correct.
 - F. A and C are correct.
7. Incidental findings are detected in approximately what percentage of trauma patients undergoing CT?
 - A. 1%
 - B. 10%
 - C. 33%
 - D. 50%
 8. In a recent poll of emergency physicians and trauma surgeons about the components of the pan-scan obtained for individual trauma patients the physicians thought were necessary, which of the following is true?
 - A. The ED physicians and trauma surgeons always agreed on which scans were necessary.
 - B. ED physicians desired 35% fewer scans, and < 1% of these undesired scans would have led to a predefined "critical action."
 - C. The trauma surgeons desired fewer scans than the ED physicians.
 - D. All scans were ordered at the discretion of the ED physician.
 9. Which of the following statements regarding clinician ability to rule out injury based on physical exam is true?
 - A. The sensitivity of ruling out injuries in specific body regions steadily decreases with higher pretest probabilities of injury.
 - B. Multiple studies have shown that physical exam is superior to CT scan for ruling out injuries.
 - C. All patients with abdominal injuries found on CT will have abdominal tenderness.
 - D. Ruling out injury based on physical exam findings is reliable in patients with altered mental status.
 10. Which of the follow statements about the use of CT is true?
 - A. The use of CT in EDs declined between 1998 and 2007.
 - B. A typical "pan-scan" is a non-contrast CT of the head, followed by contrast-enhanced scans of the neck, chest, abdomen, and pelvis.
 - C. A typical "pan-scan" is a non-contrast CT of the head, followed by contrast-enhanced scans of the neck, chest, abdomen, and pelvis and CT angiography of the extremities.
 - D. CT accounts for approximately 70% of radiation exposure in medical populations.
 - E. B and D are true.

CNE/CME Objectives

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

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

CNE/CME Instructions

HERE ARE THE STEPS YOU NEED TO TAKE TO EARN CREDIT FOR THIS ACTIVITY:

1. Read and study the activity, using the provided references for further research.
2. Log on to www.cmecity.com to take a post-test; tests can be taken after each issue or collectively at the end of the semester. *First-time users will have to register on the site using the 8-digit subscriber number printed on their mailing label, invoice, or renewal notice.*
3. Pass the online tests with a score of 100%; you will be allowed to answer the questions as many times as needed to achieve a score of 100%.
4. After successfully completing the last test of the semester, your browser will be automatically directed to the activity evaluation form, which you will submit online.
5. **Once the completed evaluation is received, a credit letter will be e-mailed to you instantly.** You will no longer have to wait to receive your credit letter.

To reproduce any part of this newsletter for promotional purposes, please contact:

Stephen Vance

Phone: (800) 688-2421, ext. 5511

Fax: (800) 284-3291

Email: stephen.vance@ahcmedia.com

To obtain information and pricing on group discounts, multiple copies, site-licenses, or electronic distribution please contact:

Tria Kreutzer

Phone: (800) 688-2421, ext. 5482

Fax: (800) 284-3291

Email: tria.kreutzer@ahcmedia.com

To reproduce any part of AHC newsletters for educational purposes, please contact:

The Copyright Clearance Center for permission

Email: info@copyright.com

Website: www.copyright.com

Phone: (978) 750-8400

Editor in Chief

Ann Dietrich, MD, FAAP, FACEP

Professor of Pediatrics
Ohio State University
Attending Physician
Nationwide Children's Hospital
Associate Pediatric Medical Director
MedFlight
Columbus, Ohio

Editorial Board

Mary Jo Bowman, MD, FAAP, FCP

Associate Professor of Clinical Pediatrics
Ohio State University College of Medicine
PEM Fellowship Director, Attending Physician
Children's Hospital of Columbus
Columbus, Ohio

Lawrence N. Diebel, MD

Professor of Surgery
Wayne State University
Detroit, Michigan

Robert Falcone, MD, FACS

Clinical Professor of Surgery
The Ohio State University
College of Medicine
Columbus, Ohio

Theresa Rodier Finerty, RN, MS, CNA, BC

Director, Emergency and Trauma

Services,
OSF Saint Francis Medical Center
Peoria, Illinois

Dennis Hanlon, MD, FAAEM

Vice Chairman, Academics
Department of Emergency Medicine
Allegheny General Hospital
Pittsburgh, Pennsylvania

Jeffrey Linzer Sr., MD, FAAP, FACEP

Assistant Professor of Pediatrics and Emergency Medicine
Emory University School of Medicine
Associate Medical Director for Compliance
Emergency Pediatric Group
Children's Healthcare of Atlanta at Egleston and Hughes Spalding
Atlanta, Georgia

S.V. Mahadevan, MD, FACEP, FAAEM

Associate Professor of Surgery/
Emergency Medicine
Stanford University School of Medicine
Associate Chief, Division of
Emergency Medicine
Medical Director, Stanford University
Emergency Department
Stanford, California

Janet A. Neff, RN, MN, CEN

Trauma Program Manager

Stanford University Medical Center
Stanford, California

Ronald M. Perkin, MD, MA, FAAP, FCCM

Professor and Chairman
Department of Pediatrics
The Brody School of Medicine at East Carolina University
Medical Director, Children's Hospital
University Health Systems of Eastern Carolina
Greenville, North Carolina

Andrew D. Perron, MD, FACEP, FACS

Professor and Residency Program Director,
Department of Emergency Medicine,
Maine Medical Center
Portland, Maine

Steven A. Santanello, DO

Medical Director, Trauma Services
Grant Medical Center
Columbus, Ohio

Eric Savitsky, MD

Associate Professor Emergency Medicine
Director, UCLA EMC Trauma Services and Education
UCLA Emergency Medicine
Residency Program
Los Angeles, California

Thomas M. Scalea, MD

Physician-in-Chief
R Adams Cowley Shock Trauma Center
Francis X. Kelly Professor of Trauma Surgery
Director, Program in Trauma
University of Maryland School of Medicine

Perry W. Stafford, MD, FACS, FAAP, FCCM

Professor of Surgery
UMDNJ Robert Wood Johnson Medical School
New Brunswick, New Jersey

Steven M. Winograd, MD, FACEP

St. Barnabus Hospital, Core Faculty
Emergency Medicine Residency Program
Albert Einstein Medical School,
Bronx, New York

CNE Nurse Reviewer

Sue A. Behrens, APRN, BC

Director of Emergency/ECU/Trauma Services
OSF Saint Francis Medical Center
Peoria, IL

© 2012 AHC Media. All rights reserved.

Trauma Reports™ (ISSN 1531-1082) is published bimonthly by AHC Media, a division of Thompson Media Group, LLC, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Senior Vice President / Group Publisher: Donald R. Johnston

Executive Editor: Shelly Morrow Mark

Managing Editor: Leslie Hamlin

POSTMASTER: Send address changes to Trauma Reports, P.O. Box 105109, Atlanta, GA 30348.

Copyright © 2012 by AHC Media, Atlanta, GA, a division of Thompson Media Group LLC. All rights reserved. Reproduction, distribution, or translation without express written permission is strictly prohibited.

Subscriber Information

Customer Service: 1-800-688-2421

Customer Service E-Mail:
customerservice@ahcmedia.com

Editorial E-Mail:
shelly.mark@ahcmedia.com

World Wide Web page:
<http://www.ahcmedia.com>

FREE to subscribers of *Emergency Medicine Reports* and *Pediatric Emergency Medicine Reports*

Subscription Prices

United States

\$249 per year. Add \$17.95 for shipping & handling

Multiple Copies

Discounts are available for group subscriptions, multiple copies, site-licenses or electronic distribution. For pricing information, call Tria Kreutzer at 404-262-5482.

All prices U.S. only. U.S. possessions and Canada, add \$30 postage plus applicable GST.

Other international orders, add \$30.

Accreditation

AHC Media is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

AHC Media designates this enduring material for a maximum of 2.5 *AMA PRA Category 1 Credits™*. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Approved by the American College of Emergency Physicians for a maximum of 2.5 hour(s) of ACEP Category 1 credit.

AHC Media is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.

This activity has been approved for 1.5 nursing contact hours using a 60-minute contact hour.

Provider approved by the California Board of Registered Nursing, Provider # 14749, for 1.5 Contact Hours.

This is an educational publication designed to present scientific information and opinion to health professionals, to stimulate thought, and further investigation. It does not provide advice regarding medical diagnosis or treatment for any individual case. It is not intended for use by the layman. Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. Clinical, legal, tax, and other comments are offered for general guidance only; professional counsel should be sought for specific situations.

This CME/CNE activity is intended for emergency, family, osteopathic, trauma, surgical, and general practice physicians and nurses who have contact with trauma patients.

It is in effect for 24 months from the date of publication.

© 2012 AHC Media. All rights reserved.

AHC Media

**Rhabdomyolysis:
 Review and
 Update**

Major Causes of Rhabdomyolysis

- Exertional
 - Genetic predisposition
- Trauma
- Drugs
- Toxins/venoms
- Infection
- Environmental
 - Heat or cold
- Electrolyte abnormalities
- Food

Hereditary Conditions Associated with Rhabdomyolysis

- Carnitine Metabolism Disorders
 - Carnitine palmitoyl transferase (CPT2) deficiency
 - VLCAD (very long chain acyl CoA deficiency)
- Adenosine monophosphate deaminase deficiency (AMPD)
- McArdle's disease (glycogen storage disease type 5)
- Malignant hyperthermia
- Phosphorylase kinase deficiency
- Duchenne muscular dystrophy
- 11-hydroxylase deficiency
- Phosphofructokinase deficiency

Trauma Conditions Associated with Rhabdomyolysis

- Crush injury
- Compartment syndrome
- Physical torture and abuse
- Exercise
- Heat stroke
- High voltage electrical injury
- Lightning
- Elevated ambient temperature (heat exposure)
- Low ambient temperature (cold exposure)

Medication/Drug Causes of Rhabdomyolysis

- Ethanol
- Cocaine and other sympathomimetics
- Anticholinergic agents
- Sedatives and hypnotics
- Metabolic poisons (cyanide, carbon monoxide)
- Colchicine
- Steroids
- Zidovudine
- Statins
- Propofol (infusion)
- Daptomycin
- Sunitinib and Imatinib
- Leflunomide
- Serotonin syndrome
- Neuroleptic malignant syndrome

Envenomations Associated with Rhabdomyolysis

- Ants (fire ants in particular)
- Bees
- Centipedes
- Wasps
- Scorpions
- Snakes (hemotoxic and myotoxic snakes)

Infections Associated with Rhabdomyolysis

Viral

- Influenza (H1N1; A; B)
- Coronavirus
- Herpesvirus
- HIV
- Dengue
- Parainfluenza
- Varicella
- West Nile encephalitis
- Mononucleosis (Epstein-Barr)
- Cytomegalovirus (CMV)
- Coxsackie

Bacterial

- *Staphylococcus aureus*
- *Salmonella typhi*
- *Pseudomonas aeruginosa*
- *Mycoplasma pneumoniae*
- *Bacillus cereus*
- *Clostridium tetani*
- *E. coli*
- *Listeria monocytogenes*
- *Legionella pneumophila*
- Tularemia
- Tetanus

Protozoan

- *P. vivax* (malaria)

Miscellaneous Causes of Rhabdomyolysis

Electrolytes

- Hypokalemia
- Hypernatremia
- Hypocalcemia
- Hypophosphatemia

Food

- Quail ingestion (coturnism)
- Mushrooms
- Licorice
- Red yeast rice (*Monascus purpureus*)

Endocrine

- Thyrotoxicosis
- Hyperaldosteronism
- DKA

Other

- Status asthmaticus
- Massage
- Polymyositis
- Dermatomyositis
- Neurosarcoidosis
- Sjögren's syndrome

Supplement to *Emergency Medicine Reports*, September 10, 2012: "Rhabdomyolysis: Review and Update." Authors: Larissa I. Velez, MD, Assistant Professor of Emergency Medicine, University of Texas Southwestern, Dallas; Melanie J. Lippmann, MD, Department of Emergency Medicine, University of Texas Southwestern, Dallas; Janna Welch, MD, Assistant Residency Director, University of Texas Southwestern, Dallas; and Gilberto A. Salazar, MD, Department of Emergency Medicine, University of Texas Southwestern, Dallas.

Emergency Medicine Reports' "Rapid Access Guidelines." Copyright © 2012 AHC Media, a division of Thompson Media Group LLC, Atlanta, GA. Editors: Sandra M. Schneider, MD, FACEP, and J. Stephan Stacpzyński, MD. Senior Vice President/Group Publisher: Donald R. Johnston. Executive Editor: Shelly Morrow Mark. Managing Editor: Leslie Hamlin. For customer service, call: 1-800-688-2421. This is an educational publication designed to present scientific information and opinion to health care professionals. It does not provide advice regarding medical diagnosis or treatment for any individual case. Not intended for use by the layman.

Trauma Reports

EVIDENCE-BASED MEDICINE FOR THE ED

Volume 13, Number 5

Sept/Oct 2012

Authors:

Michael C. Bond, MD, FACEP, FAAEM, Assistant Professor, Residency Program Director, Department of Emergency Medicine, University of Maryland School of Medicine, Baltimore.

Michael Scott, MD, Departments of Emergency Medicine and Internal Medicine, University of Maryland Medical Center, Baltimore.

T. Andrew Windsor, MD, Department of Emergency Medicine, University of Maryland Medical Center, Baltimore.

Peer Reviewer:

Robert E. Falcone, MD, FACS, Clinical Professor of Surgery, The Ohio State University College of Medicine, Columbus.

**Winner
Best Instructional Reporting**

March/Apr 2011 Issue

Specialized Information
Publishers Association

Statement of Financial Disclosure

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Dietrich (editor in chief), Drs. Bond, Scott, and Windsor (authors), Dr. Falcone (peer reviewer), and Ms. Behrens (nurse reviewer) report no relationships with companies related to this field of study. Ms. Mark (executive editor), and Ms. Hamlin (managing editor) report no relationships with companies related to the field of study covered by this CME activity.

The Roles and Risks of Whole-Body Computed Tomography Scans in the Trauma Patient

Emergency departments in the United States are frequently confronted with trauma patients with varying degrees of injury. Clinically significant injuries may be missed, with devastating consequences for the patient. Concern for not missing any potentially serious injuries has led to an aggressive diagnostic approach with a goal of not missing any injuries. The CT scan has facilitated this approach, providing substantial information guiding management. However, CT scans have risks, especially when the pan-scan approach is used. The authors review the uses, advantages, and disadvantages of the pan-scan.

— Ann M. Dietrich, MD, Editor

Background

Emergency departments (EDs) in the United States received approximately 35 million trauma-related visits in 2007.¹ According to the Centers for Disease Control (CDC), unintentional injury accounted for 123,706 deaths in the same year, making it the fourth leading cause of death overall and the primary cause of death among people between 1 and 44 years of age.²

The trauma population constitutes a remarkably high-risk cohort for emergency care providers and trauma surgeons. The principles of Advanced Trauma Life Support (ATLS) aim to provide a simple and effective standardized approach for the assessment and care of injured patients. Between the patient with minor isolated trauma and the unstable patient with multi-trauma requiring immediate surgical intervention lies a complex group of various injury patterns that represents a significant gray area for any trauma care provider. Patients who are stable after initial clinical evaluation still might have serious injuries that require an expedient evaluation performed in an organized manner to avoid the significant morbidity and mortality associated with delays in localization and intervention.^{3,4} Approximately 15% to 22.3% of trauma patients in whom injuries are missed have injuries that are clinically significant.⁵ This prevalence of missed serious injury has led to an aggressive diagnostic approach with an emphasis on high sensitivity and early identification of all injuries. Initially, this aggressiveness was illustrated by the use of diagnostic peritoneal lavage (DPL), an invasive but highly sensitive diagnostic screening tool that decreased the number of missed intra-abdominal injuries. With the advent of computed tomography (CT), emergency care providers and trauma surgeons have increasingly relied upon this technology as an integral part of trauma evaluation and resuscitation.⁶ The “traditional” imaging strategy includes plain radiographs of the chest, pelvis, and lateral cervical spine (C-spine), in accordance with ATLS guidelines; a Focused Assessment with Sonography for Trauma (FAST) exam; and selected CT scans as deemed necessary based on the physical examination, radiographs, or ultrasound assessment.⁷

The first generations of CT scanners suffered from a significant lack of

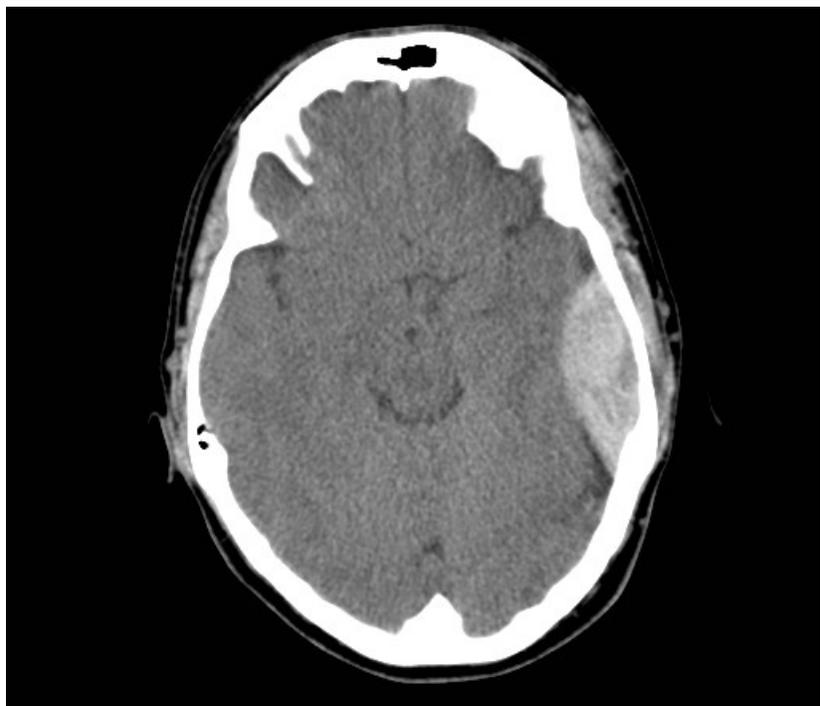
Executive Summary

- Approximately 15% to 22.3% of trauma patients in whom injuries are missed have injuries that are clinically significant. This prevalence of missed serious injury has led to an aggressive diagnostic approach with an emphasis on high sensitivity and early identification of all injuries.
- Multiple studies have validated the hypothesis that CT is superior in accuracy and reliability compared with physical examination, laboratory screening, plain radiographs, and sonography alone in the evaluation of most serious traumatic injuries.
- A typical pan-scan involves a non-contrast CT scan of the head and contrast-enhanced scans of the neck, chest, abdomen, and pelvis.
- Recent studies have shown the single-pass pan-scan to be a viable, if not superior, alternative to conventional segmental whole-body protocols. The single-pass technique has been found to be accurate and timesaving, reducing acquisition time by as much as 42.5% and decreasing radiation dose.

sensitivity and specificity and were limited by the amount of time required for the scan (separating the patient from the monitoring of the trauma resuscitation bay). The introduction of multi-slice CT scanners has both improved diagnostic accuracy and reduced the time of scanning significantly.⁸ Multiple studies have validated the hypothesis that CT is superior in accuracy and reliability compared with physical examination, laboratory screening, plain radiographs, and sonography alone in the evaluation of most serious traumatic injuries.⁹⁻¹⁶

The number of CTs performed in EDs for injury-related conditions nearly doubled between 1998 and 2007.⁶ CT now has a well-established role in the secondary evaluation of trauma, and the concept of a whole-body CT scan, or “pan-scan,” has become more accepted as an adjunct for definitive assessment of injuries during the early stages of trauma management.^{6,17,18} A typical pan-scan involves a non-contrast CT scan of the head (see Figure 1) and contrast-enhanced scans of the neck, chest, abdomen, and pelvis. Some protocols also include dedicated reconstructed views of the rest of the spine or other osseous structures. (See Figure 2.) Modern multi-detector CT scanners have the ability to produce specialized studies such as fine cuts through the facial, orbital, and temporal bones, as well as CT angiography of the body and extremities (these are not included in most

Figure 1. Epidural Hematoma



CT of a 26-year-old male on warfarin for a mechanical heart valve replacement. He fell from a ladder, sustaining an epidural hematoma. Image courtesy of University of Maryland School of Medicine Department of Emergency Medicine.

“standard” pan-scans).¹⁹ Naturally, since departmental policies and CT manufacturers differ, institutions tend to have slightly different whole-body CT protocols. Strategies for reducing scan time, improving image quality, and decreasing radiation exposure are being investigated.^{17,19,20}

Recent studies have shown the

single-pass pan-scan to be a viable, if not superior, alternative to conventional segmental whole-body protocols. A single-pass CT scan captures the neck and body portions in a single scan, usually with multi-phased contrast injection. Conventional pan-scans usually incorporate pauses and multiple overlapping scans to

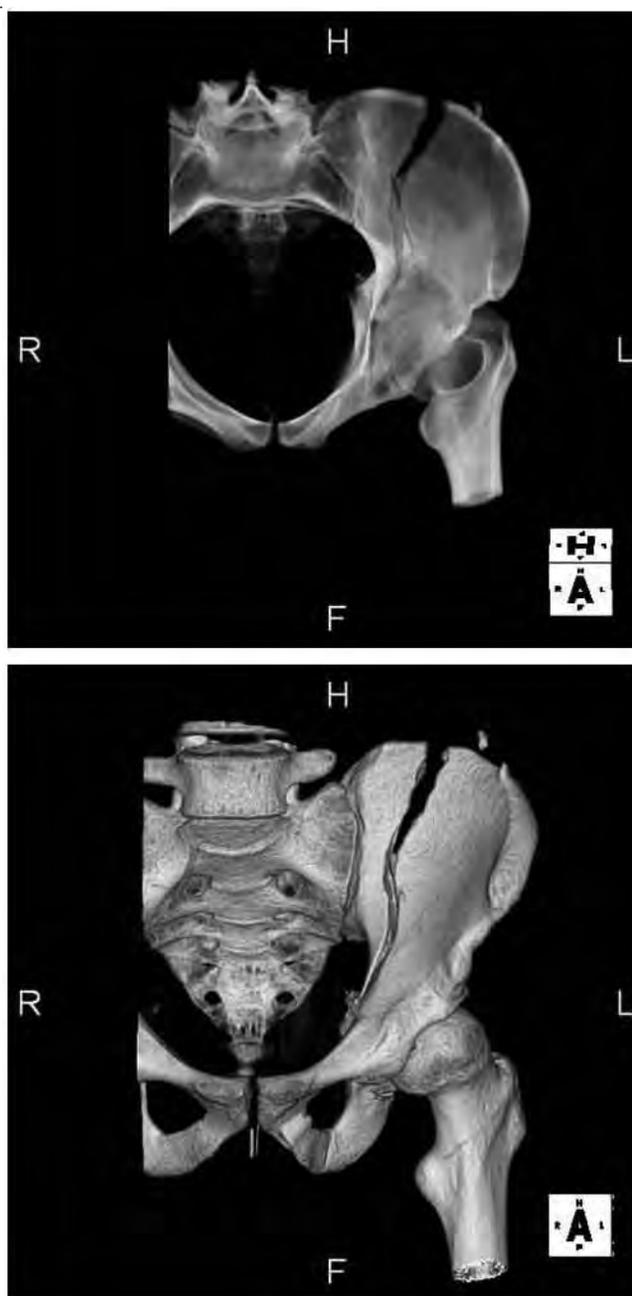
achieve separate portal and arterial phases. The single-pass technique has been found to be accurate and time-saving,^{17,21} reducing acquisition time by as much as 42.5%¹⁹ and decreasing radiation dose.²²

The immediate benefits of CT are easy to recognize: A significant amount of clinical information can be gained in a short period of time in a noninvasive manner, which aids in triaging, surgical planning, and disposition. Multiple studies have suggested that the sensitivity of CT has progressed to the point that a negative study can effectively eliminate the possibility of significant traumatic injuries, allowing patients who otherwise might have required observation for hours or days to be discharged home earlier.^{8,17,23-26} Few experts dispute the necessity of pan-scanning patients with significant physical evidence of multi-trauma (see Figure 3), those with massive blunt or penetrating injuries, and those in whom the physical examination is unreliable because of altered mental status, depressed level of consciousness, or significant distracting injuries.²⁷⁻²⁹ However, an ongoing debate focuses on the utility of the pan-scan as a standard part of the evaluation of patients with moderate trauma and of those without clinically evident injuries who have normal laboratory values and plain radiographs.^{30,31} The concerns are not without merit, as the pan-scan protocol poses several risks for the patient: radiation exposure, allergic reaction to contrast, contrast-induced nephropathy, and contrast extravasation.

Potential Benefits of Pan-Scan

Diagnostic Yield. As discussed above, the initial management of trauma patients involves an aggressive attempt to identify all injuries early. The use of whole-body CT for this purpose has been supported by studies that suggest that a pan-scan identifies more injuries and leads to a change in management more often than following selective CT protocols.^{18,32} In the study by Salim and

Figure 2. Iliac Wing Fracture

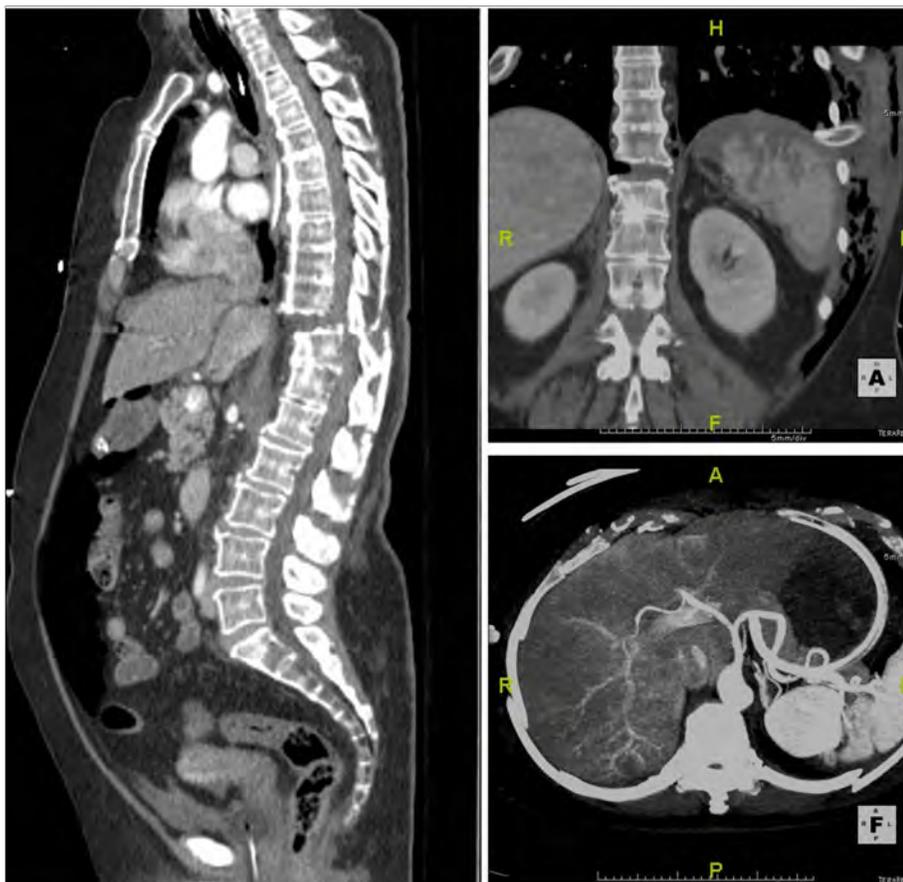


Two versions of 3D reconstructions of an iliac wing fracture in a 24-year-old male who was injured in a high-speed motor vehicle crash. Images courtesy of University of Maryland School of Medicine Department of Emergency Medicine.

colleagues,¹⁸ 18.9% of patients had a change in management as a result of a finding on whole-body CT, including earlier discharge and procedural or surgical intervention. The study's data analysis focused on the

abdominal portion of the pan-scan and found that 20.3% of patients who had a normal abdominal examination had a change in management after abdominal CT. Six of these patients required laparotomy. These

Figure 3. Pan-scan of Serious Injuries



In this 64-year-old woman who was involved in a high-speed motor vehicle crash, pan-scan identified a number of serious injuries, including spinal fracture/dislocation, grade 3 splenic laceration, and grade 5 liver injury. Image courtesy of University of Maryland School of Medicine Department of Emergency Medicine.

results suggest that CT can identify injuries in patients with normal results on the physical examination. In another study, 18 emergency physicians' clinical judgment showed relatively high sensitivity (69.9% to 100%) in excluding injuries without a pan-scan if a patient's pretest probability of injury was "very low."³³ The sensitivity of excluding injuries in specific body regions steadily decreased with higher pretest probabilities of injury, supporting the idea that the accuracy of clinician judgment worsens in the assessment of severely injured patients.

Multiple studies have failed to develop a clinical decision rule that

could completely exclude all types of intra-abdominal injuries after blunt trauma without performing CT.^{15,25,34} In two of these studies, the presence of abdominal pain or tenderness achieved 100% sensitivity in detecting intra-abdominal injuries requiring surgical intervention,^{25,30,34} even though it missed non-surgical injuries. Because the primary outcome of these studies was identifying all injuries, the clinical decision rules were viewed as failures. This illustrates two viewpoints as to what is the most important endpoint when evaluating the use of CT in trauma. Should the endpoint be finding any injury, or should it be finding only injuries that

require surgical or medical intervention? Do clinicians really need to know about an injury that does not require treatment?

Gupta and colleagues presented a study that further illustrates this debate.³⁵ They polled emergency physicians and trauma surgeons about which components of the pan-scan obtained for individual trauma patients they thought were necessary. All scans were ordered at the discretion of the trauma surgeon. The ED physicians would have ordered 35% fewer scans, but in doing so would have missed 10% of injuries. However, only 0.3% of these injuries would have led to a predefined critical action. This suggests that although CT is superior in identifying objective injuries that the physical examination and clinical suspicion might miss, very few of these injuries prove to be emergently dangerous. Of note, the authors had difficulty agreeing on the true importance of the abnormal findings. The emergency medicine authors thought the projected miss rate of 0.3% (the number of missed injuries that would have required predefined critical actions) supported a more selective use of CT based on physician judgment. The trauma surgeon authors pointed to the projected missed injury rate of 10% (the number of all missed injuries, regardless of requiring a critical action or not) as justification for more liberal use of CT.

Survival/Mortality. Although most of these studies have used injuries identified on CT or change in management based on CT as the primary outcome, some studies have indicated a possible decrease in the mortality rate with a liberal CT approach. Hutter et al³⁶ studied the effect on survival before and after the institution of a liberal pan-scan policy at a major high-volume trauma center in Germany. The study included patients who did not undergo a pan-scan due to the unavailability of the method, patients who were eligible but were not pan-scanned due to physician discretion, and eligible patients who underwent a pan-scan. Patients who actually underwent a

pan-scan had a statistically significant reduction in overall mortality, with an odds ratio (OR) of 0.17 (95% CI, 0.1–0.28) and a total risk difference of 7%. The relative impact of a patient receiving a pan-scan on the mortality rate was small compared with the effect of Injury Severity Score (ISS) or neurologic function.

Huber-Wagner and associates³⁷ suggested that the use of whole-body CT in the management of trauma patients increased the probability of survival compared with the predicted mortality rate based on the Trauma Injury Severity Score (TRISS) and Revised Injury Severity Classification (RISC) scores. This study has been criticized because more patients in the whole-body CT group were treated at trauma centers, and it was acknowledged that the predicted mortality rate for the whole-body CT group was likely increased due to clinically insignificant findings that would in turn elevate the ISS.³⁸ Van Vugt et al³⁹ demonstrated that clinically insignificant findings found on CT do indeed elevate the ISS, artificially inflating mortality rate estimates beyond the true mortality rate, potentially altering the statistical importance of the pan-scan on survival. As overall survival in trauma generally continues to trend upward,³⁷ it is important to keep in mind that these observed effects are likely caused by complex systemic changes and that assigning a causal relationship to any one intervention is probably not appropriate.

Potential Pitfalls of Pan-Scan

Because the rates of CT use in EDs have skyrocketed in recent years, more attention is being directed toward the weaknesses of this diagnostic modality.⁶

Contrast-Induced Nephropathy (CIN). Published reports contain significant variability regarding the effect of intravenous (IV) contrast for CT on renal function. The incidence of CIN, most commonly defined as an increase in creatinine of 0.5 mg/dL or 25% from the baseline creatinine level, has been

Table 1. Average Adult Effective Doses of Various Radiologic Studies

Radiograph	Average Effective Dose (mSv)
Single chest	0.02
Cervical spine	0.1
Thoracic spine	1.0
Lumbar spine	1.5
Pelvis	0.6
CT	Average Effective Dose (mSv)
Head	2
Neck	3
Chest	7
Abdomen	8
Pelvis	6
Spine	6

Adapted from Mettler FA Jr, Huda W, Yoshizumi TT, Mahesh M. Effective doses in radiology and diagnostic nuclear medicine: A catalog. *Radiology* 2008;248:254-263.

estimated to be anywhere from 0% to 12%, depending on the study and the underlying risk factors of the patients.⁴⁰⁻⁴⁶ Importantly, it remains unclear whether IV contrast is the actual cause of the rise in creatinine. One literature review noted that in two studies in which control groups of patients did not receive IV contrast, no association was found between IV contrast and a rise in the serum creatinine level.⁴⁷⁻⁴⁹

Research into the true risks of IV contrast is ongoing. Mitchell and Kline found an incidence of CIN of 11% in a population of 633 general ED patients who underwent contrast-enhanced CT.⁴⁴ Of the 70 patients in whom CIN developed, renal failure developed in 7 (1% of the overall population), defined as an increase in serum creatinine of 3 mg/dL or more. Six of those seven patients died, and in four cases it was believed that renal failure significantly contributed to death.

Unfortunately, there is not a large body of literature investigating renal failure after contrast CT in the trauma patient. However, Matsushima and colleagues⁵⁰ did find an ISS of 16 or greater to be a risk factor for contrast-induced acute

kidney injury, although the dose of contrast received was not associated with an increased risk, suggesting the possibility of an association rather than causality.

Overall, although there is still controversy about CIN and the exact risks associated with acute renal failure, especially in regard to the trauma population, the consensus seems to be that there is a small but very real risk of CIN following CT with IV contrast, a point all providers should consider when ordering CT scans.

Cancer Risk. CT is responsible for more than 70% of medical radiation exposure; 16.2 million scans were ordered for ED patients in 2007.^{51,52} Brenner and Hall estimated that 1.5% to 2.0% of all cancers in the United States might be attributable to ionizing radiation from CT, including many types of scans other than those done during trauma assessments. The probable death rate is much lower, at about 0.1 to 0.35%.^{38,53,54} These and other estimates are not universally accepted because they are based on a linear no-threshold relationship that assumes the incidence of cancer induction is proportional to

Table 2. Intermediate Level Trauma Patients (per MIEMSS* protocols)

<p>Category B</p> <ul style="list-style-type: none"> • Glasgow Coma Scale (GCS) score 9-14 • Paralysis or vascular compromise of limb • Amputation proximal to wrist or ankle • Crushed, degloved, or mangled extremity • Penetrating injuries to extremities proximal to elbow or knee • Combination trauma with burns
<p>Category C</p> <ul style="list-style-type: none"> • Age < 5 years or > 55 years • Patient with bleeding disorder or patient on anticoagulants • Dialysis patient • Pregnancy > 20 weeks • EMS provider judgment • High-risk auto crash <ul style="list-style-type: none"> • Intrusion > 12 in. occupant site; > 18 in. any site • Ejection (partial or complete) from vehicle • Death in same passenger compartment • Vehicle telemetry data consistent with high risk of injury • Exposure to blast or explosion • Falls greater than 3 times patient's height <p>Intermediate trauma classifications based on the trauma decision tree protocols promulgated by the Maryland Institute for Emergency Medical Services Systems. Please refer to local trauma classifications and regulations for management in other jurisdictions. Adapted from <i>The Maryland Medical Protocols for EMS Providers</i>. Baltimore, Maryland: MIEMSS, 2011.</p>

exposure, which some argue is not congruent with biological and animal data.⁵⁵ Most models are based on data extrapolated from atomic bomb survivors, but there has been debate about the level of radiation that leads to an increased cancer risk. A full explanation of this debate is beyond the scope of this article, but some have concluded that there is no significant carcinogenic risk with a dose up to 150 to 200 millisieverts (mSv) to normal tissues,^{55,56} while others estimate that the safe dose is less than 100 mSv.⁵³ The sievert (Sv) is a measure of the effective dose of radiation on biological tissues based on the stochastic effect of ionizing radiation. The average underlying exposure from everyday background radiation is about 3 mSv per year.⁵⁴ Most patients receive an effective dose of about 20 to 50 mSv

from a single pan-scan,^{54,57} depending on the scanner power and scan technique. Table 1 includes a list of the average effective doses from individual radiologic studies.⁵⁸

The risk of death from severe trauma has been estimated to be 50 to 100 times higher than the risk of a cancer death from CT-related radiation exposure.^{59,60} In the trauma population, use of whole-body CT is usually not questioned because of the significant risk-benefit ratio. The population of particular interest and debate, however, has intermediate-level trauma, such as category B or C patients (*see Table 2*) or priority II patients; in other words, those who have potentially life-threatening, but not immediately life-endangering injuries. In this subset of patients, which has not been well-studied, trauma-related mortality is still a

real concern, estimated between 0.6% and 2%,^{38,57} but there is a larger proportion without serious injuries. In a recent study of 642 adult intermediate-level trauma patients, Laack and colleagues estimated that the risk of trauma-related mortality was six times greater than the cancer risk.³⁸ Trauma-related mortality was highest in older patients, and the risk of cancer death was inversely proportional to age; therefore, the youngest patients have the most potential danger from radiation — patients younger than 20 years have four times the estimated risk of those older than 60 years. (*See Figure 4.*) It is notable that no one younger than 80 died in Laack's study, and all deaths were caused by head injuries. The mortality rate and median ISS were relatively low compared with the findings in a smaller study by Winslow and associates, who examined the amount of radiation to which intermediate trauma patients were exposed.⁵⁷

Pediatrics. Because children are more susceptible to the carcinogenic effects of ionizing radiation, efforts to reduce unnecessary radiation exposure are paramount. CT remains the diagnostic test of choice for evaluation of blunt trauma in children, but the risk of inducible cancer is much higher than in adults. Mueller et al⁶¹ showed that the effective radiation dose during CT is on par with adults, but doses to organs such as the thyroid gland fell within the range of radiation doses historically correlated with increased cancer risk. Based on a model by Berrington de Gonzalez and associates, the mean lifetime cancer risk after whole-body CT in 3-year-old boys and girls is 1 in 133 and 1 in 166, respectively.⁶² At 15 years of age, the risks were estimated at 1 in 250 for girls and 1 in 500 for boys.

Although the FAST exam is well-established in adult trauma, its utility as a screening exam in pediatric trauma is not universally supported.^{7,26,63} CT has been shown to be sensitive for identification of injuries in children, similar to adults. An important consideration for pediatric

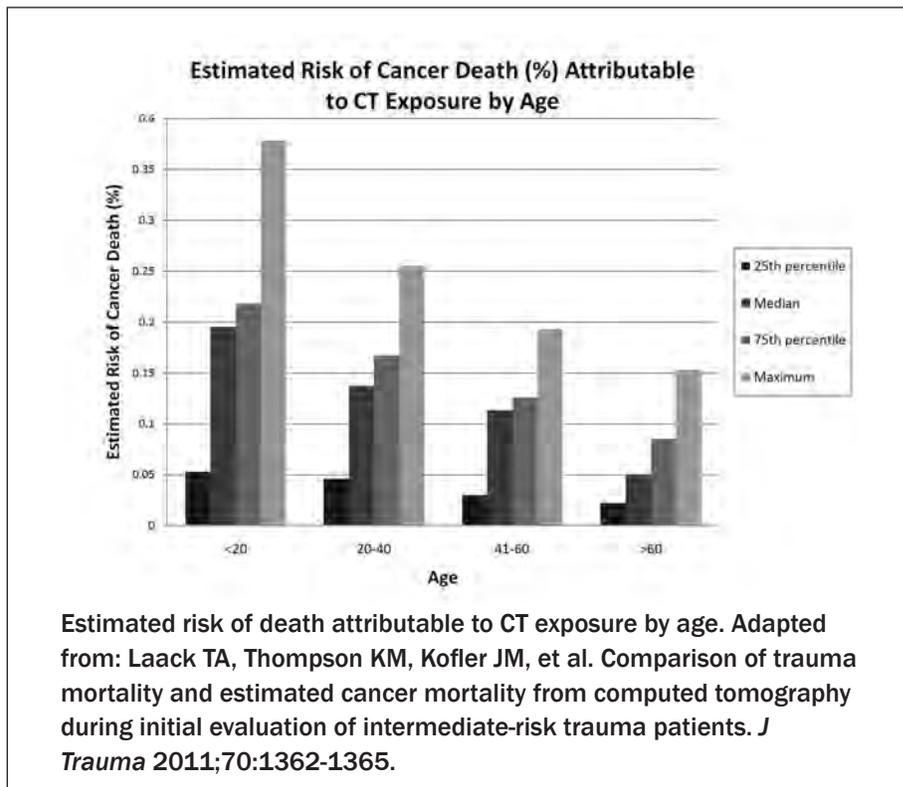
trauma, however, is that solid organ injuries in children are being treated increasingly non-operatively,^{64,65} so discovery of these injuries in hemodynamically stable children does not automatically lead to surgical intervention.⁶⁶ On the other hand, a review of three prospective studies looking at intra-abdominal injury determined that the negative predictive value (NPV) of abdominal CT was 99.8%, suggesting that routine admission and serial exams after a normal abdominal CT and a normal physical exam may not be necessary.⁶⁸

Clinical decision rules for obtaining a CT scan have been successful for pediatric head injury^{68,69} and C-spine injury,⁷⁰ and are being investigated for abdominal trauma.⁶⁶ Unfortunately, just as with adults, there is no clinical decision rule for using pan-scan in the pediatric trauma population. Use of a pan-scan is not routinely recommended in stable children without abnormal physical exam findings or a mechanism that induces concern for significant injury. Selective scanning of body areas, rather than whole-body scanning, results in a statistically significant decrease in all organ doses and total effective dose.⁶¹

Other Considerations

An Imperfect Test. While CT has been shown to have generally superior ability to identify injuries, it is not a perfect test. Alone, CT has demonstrated insufficient sensitivity to rule out diaphragm injury, although its positive predictive value appears to be very good.⁷¹ (See *Figure 5*.) Similar concern has been expressed for other radiographically occult injuries such as diffuse axonal injury, hollow viscus injuries, and mesenteric injuries, but Tan showed that patients with surgically confirmed hollow viscus and mesenteric injuries were very likely to have had an abnormal CT scan.⁷² Positive trauma scans are conclusive, but negative results require subsequent confirmation.¹⁷ Injuries initially missed on CT are uncommon; while these falsely negative scans can sometimes

Figure 4. Risk of Death and CT Exposure

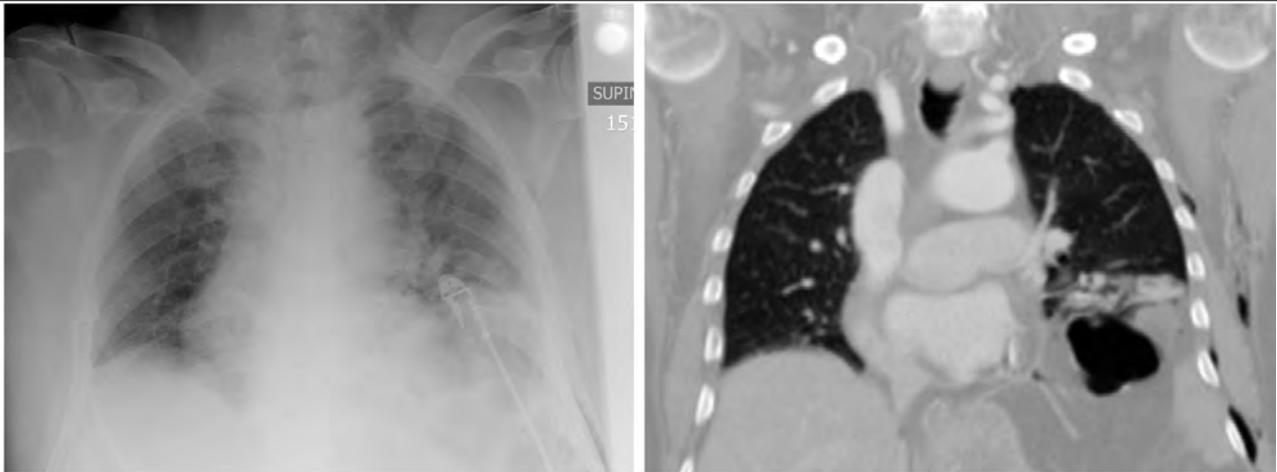


delay management, they have not yet been shown to affect the mortality rate.⁷³ In that vein, second readings are advocated to avoid missed injuries not seen on the initial preliminary “hot read,”⁷⁴ at least in patients with intermediate to high pretest probability for injury.

Imaging Prior to Transfer. Emergency medicine practitioners who do not work at designated trauma centers will certainly be responsible for the occasional trauma patient, and if the patient meets criteria for transfer to a trauma center, the question of whether to obtain imaging prior to transfer may arise. From a practical standpoint, the role of the practitioner is to stabilize the patient to the best of his or her abilities and to facilitate transport to the receiving facility for definitive management as soon as possible. As noted previously, CT is sensitive for diagnosing life-threatening injuries and can be used as a tool for determining disposition after trauma.¹⁸ In a stable trauma patient with

clinically suspected injuries based on mechanism or examination, CT might elucidate the need for further specialized management or it can obviate the need for admission or transfer. If a patient meets criteria for transfer prior to imaging, it is important to communicate with the receiving facility regarding the expectation of, or necessity for, imaging at the referring center. However, if a patient clinically requires transfer to a trauma center, sophisticated diagnostic studies may help with eventual management but should not delay transfer. Between 53% and 58% of transferred patients receive repeat imaging upon arrival at the receiving trauma center.^{35,75} The reasons for repeating studies are varied, including change of clinical status during transfer, inadequate original technique, software incompatibility, and even simple human error such as misplacing or forgetting to send the original scans. It is notable that patients who received repeat imaging tended to be more severely injured

Figure 5. Pan-scan Showing Rib Fractures, Left Hemidiaphragm Injury, and Large Flank Hematoma



Following a motor vehicle crash, a clinically stable 84-year-old man complained of mild left flank pain. His wife died as a result of the crash. Pan-scan revealed a number of rib fractures, a left hemidiaphragm injury, and large flank hematoma, which were not evident on plain radiograph and FAST examination. Images courtesy of University of Maryland School of Medicine Department of Emergency Medicine.

but also suffered longer delays⁷⁵ as well as additional radiation and financial charges. Lastly, incidental findings are found in up to one-third of trauma patients,³⁹ and trauma patients are notoriously often lost to follow-up,⁷⁶ which begs the question of who will arrange follow-up on these patients if incidental abnormalities are found during a formal over read after transfer.

Recommendations

Unfortunately, there is no universally accepted algorithm regarding routine whole-body CT compared with selective CT. A hospital could benefit from the development of a standardized CT protocol. Standardizing the radiologic workup of trauma patients needs to balance the risks of complications, cost, and incidental findings from CT versus the risk of missed injuries, repeat imaging, and delay in disposition or treatment.

Pan-scan is a noninvasive and effective method of injury determination in the hemodynamically stable trauma population, but it must be used with an awareness of its associated problems. Indiscriminate use

without proper clinical evaluation or concern is inappropriate. Efforts to reduce radiation exposure should be focused toward younger patients and those with obviously minor injuries or trauma mechanisms. Pan-scanning may reduce but does not eliminate the incidence of missed injuries and is not a substitute for a thorough clinical evaluation, appropriate repeat examinations, and follow-up. Single-pass pan-scans reduce scan time and radiation compared with conventional sequential imaging. Comprehensive secondary reading of a scan after a preliminary read may lower the rate of missed injuries.

Further research is needed. Currently in progress is the REACT-2 trial, a prospective, multi-center, multi-national study investigating the effects of immediate pan-scan CT during the primary survey on clinical outcomes compared with the use of conventional imaging and selective CT. The intervention group undergoes immediate whole-body CT, completely eliminating plain radiographs and the FAST exam. This is a novel approach and is focusing on the primary outcome of in-hospital mortality as well as

secondary endpoints such as effects on morbidity, radiation exposure, and cost-effectiveness. It is hoped that this study, and those to follow, will delineate an appropriately balanced diagnostic approach for trauma patients.

References

1. Niska R, Bhuiya F, Xu J. National Hospital Ambulatory Medical Care Survey: 2007 Emergency Department Summary. *Natl Health Stat Report* 2010;1-31.
2. CDC. Ten Leading Causes of Death and Injury. 2007; <http://www.cdc.gov/injury/wisqars/LeadingCauses.html>. Accessed May 5, 2012.
3. Fakhry SM, Brownstein M, Watts DD, et al. Relatively short diagnostic delays (< 8 hours) produce morbidity and mortality in blunt small bowel injury: An analysis of time to operative intervention in 198 patients from a multicenter experience. *J Trauma* 2000;48:408-415.
4. Malinoski DJ, Patel MS, Yakar DO, et al. A diagnostic delay of 5 hours increases the risk of death after blunt hollow viscus injury. *J Trauma* 2010;69:84-87.
5. Pfeifer R, Pape HC. Missed injuries in trauma patients: A literature review. *Patient Saf Surg* 2008;2:20.
6. Korley FK, Pham JC, Kirsch TD. Use of advanced radiology during visits to US emergency departments for injury-related conditions, 1998-2007. *JAMA* 2010;304:1465-1471.

7. *Advanced Trauma Life Support for Doctors ATLS: Manuals for Coordinators and Faculty*. Eighth ed. Chicago, IL: American College of Surgeons; 2008.
8. Rieger M, Czermak B, El Attal R, et al. Initial clinical experience with a 64-MDCT whole-body scanner in an emergency department: Better time management and diagnostic quality? *J Trauma* 2009;66:648-657.
9. Demetriades D, Gomez H, Velmahos GC, et al. Routine helical computed tomographic evaluation of the mediastinum in high-risk blunt trauma patients. *Arch Surg* 1998;133:1084-1088.
10. Exadaktylos AK, Sclabas G, Schmid SW, et al. Do we really need routine computed tomographic scanning in the primary evaluation of blunt chest trauma in patients with "normal" chest radiograph? *J Trauma* 2001;51:1173-1176.
11. Gestring ML, Gracias VH, Feliciano MA, et al. Evaluation of the lower spine after blunt trauma using abdominal computed tomographic scanning supplemented with lateral scanograms. *J Trauma* 2002;53:9-14.
12. Guillaumondegui OD, Pryor JP, Gracias VH, et al. Pelvic radiography in blunt trauma resuscitation: A diminishing role. *J Trauma* 2002;53:1043-1047.
13. Hauser CJ, Visvikis G, Hinrichs C, et al. Prospective validation of computed tomographic screening of the thoracolumbar spine in trauma. *J Trauma* 2003;55:228-235.
14. Griffen MM, Frykberg ER, Kerwin AJ, et al. Radiographic clearance of blunt cervical spine injury: Plain radiograph or computed tomography scan? *J Trauma* 2003;55:222-227.
15. Poletti PA, Mirvis SE, Shanmuganathan K, et al. Blunt abdominal trauma patients: Can organ injury be excluded without performing computed tomography? *J Trauma* 2004;57:1072-1081.
16. Brown CV, Antevil JL, Sise MJ, et al. Spiral computed tomography for the diagnosis of cervical, thoracic, and lumbar spine fractures: Its time has come. *J Trauma* 2005;58:890-896.
17. Stengel D, Ottersbach C, Matthes G, et al. Accuracy of single-pass whole-body computed tomography for detection of injuries in patients with major blunt trauma. *CMAJ* March 5, 2012 [Epub ahead of print].
18. Salim A, Sangthong B, Martin M, et al. Whole body imaging in blunt multisystem trauma patients without obvious signs of injury: Results of a prospective study. *Arch Surg* 2006;141:468-475.
19. Nguyen D, Platon A, Shanmuganathan K, et al. Evaluation of a single-pass continuous whole-body 16-MDCT protocol for patients with polytrauma. *AJR Am J Roentgenol* 2009;192:3-10.
20. Loupatatzis C, Schindera S, Gralla J, et al. Whole-body computed tomography for multiple traumas using a triphasic injection protocol. *Eur Radiol* 2008;18:1206-1214.
21. Gralla J, Spycher F, Pignolet C, et al. Evaluation of a 16-MDCT scanner in an emergency department: Initial clinical experience and workflow analysis. *AJR Am J Roentgenol* 2005;185:232-238.
22. Fanucci E, Fiaschetti V, Rotili A, et al. Whole body 16-row multislice CT in emergency room: effects of different protocols on scanning time, image quality and radiation exposure. *Emerg Radiol* 2007;13:251-257.
23. Livingston DH, Lavery RF, Passannante MR, et al. Admission or observation is not necessary after a negative abdominal computed tomographic scan in patients with suspected blunt abdominal trauma: Results of a prospective, multi-institutional trial. *J Trauma* 1998;44:273-282.
24. Livingston DH, Lavery RF, Passannante MR, et al. Emergency department discharge of patients with a negative cranial computed tomography scan after minimal head injury. *Ann Surg* 2000;232:126-132.
25. Richards JR, Derlet RW. Computed tomography for blunt abdominal trauma in the ED: A prospective study. *Am J Emerg Med* 1998;16:338-342.
26. Holmes JF, Gladman A, Chang CH. Performance of abdominal ultrasonography in pediatric blunt trauma patients: A meta-analysis. *J Pediatr Surg* 2007;42:1588-1594.
27. Pal JD, Victorino GP. Defining the role of computed tomography in blunt abdominal trauma: Use in the hemodynamically stable patient with a depressed level of consciousness. *Arch Surg* 2002;137:1029-1033.
28. Ferrera PC, Verdile VP, Bartfield JM, et al. Injuries distracting from intra-abdominal injuries after blunt trauma. *Am J Emerg Med* 1998;16:145-149.
29. Self ML, Blake AM, Whitley M, et al. The benefit of routine thoracic, abdominal, and pelvic computed tomography to evaluate trauma patients with closed head injuries. *Am J Surg* 2003;186:609-614.
30. Snyder GE. Whole-body imaging in blunt multisystem trauma patients who were never examined. *Ann Emerg Med* 2008;52:101-103.
31. Tillou A, Gupta M, Baraff LJ, et al. Is the use of pan-computed tomography for blunt trauma justified? A prospective evaluation. *J Trauma* 2009;67:779-787.
32. Deunk J, Brink M, Dekker HM, et al. Routine versus selective computed tomography of the abdomen, pelvis, and lumbar spine in blunt trauma: A prospective evaluation. *J Trauma* 2009;66:1108-1117.
33. Smith CB, Barrett TW, Berger CL, et al. Prediction of blunt traumatic injury in high-acuity patients: Bedside examination vs computed tomography. *Am J Emerg Med* 2011;29:1-10.
34. Richards JR, Derlet RW. Computed tomography and blunt abdominal injury: Patient selection based on examination, haematocrit and haematuria. *Injury* 1997;28:181-185.
35. Gupta R, Greer SE, Martin ED. Inefficiencies in a rural trauma system: The burden of repeat imaging in interfacility transfers. *J Trauma* 2010;69:253-255.
36. Hutter M, Woltmann A, Hierholzer C, et al. Association between a single-pass whole-body computed tomography policy and survival after blunt major trauma: A retrospective cohort study. *Scand J Trauma Resusc Emerg Med* 2011;19:73.
37. Huber-Wagner S, Lefering R, Qvick LM, et al. Effect of whole-body CT during trauma resuscitation on survival: A retrospective, multicentre study. *Lancet* 2009;373:1455-1461.
38. Laack TA, Thompson KM, Kofler JM, et al. Comparison of trauma mortality and estimated cancer mortality from computed tomography during initial evaluation of intermediate-risk trauma patients. *J Trauma* 2011;70:1362-1365.
39. van Vugt R, Deunk J, Brink M, et al. Influence of routine computed tomography on predicted survival from blunt thoracoabdominal trauma. *Eur J Trauma Emerg Surg* 2011;37:185-190.
40. Bell GW, Edwardes M, Dunning AM, et al. Periprocedural safety of 64-detector row coronary computed tomographic angiography: Results from the prospective multicenter ACCURACY trial. *J Cardiovasc Comput Tomogr* 2010;4:375-380.
41. Kim SM, Cha RH, Lee JP, et al. Incidence and outcomes of contrast-induced nephropathy after computed tomography in patients with CKD: A quality improvement report. *Am J Kidney Dis* 2010;55:1018-1025.
42. Lencioni R, Fattori R, Morana G, et al. Contrast-induced nephropathy in patients undergoing computed tomography (CONNECT) — A clinical problem in daily practice? A multicenter observational study. *Acta Radiol* 2010;51:741-750.
43. Mitchell AM, Jones AE, Tumlin JA, et al. Incidence of contrast-induced nephropathy after contrast-enhanced computed tomography in the outpatient setting. *Clin J Am Soc Nephrol* 2010;5:4-9.
44. Mitchell AM, Kline JA. Contrast nephropathy following computed tomography angiography of the chest for pulmonary embolism in the emergency department. *J Thromb Haemost* 2007;5:50-54.
45. Rashid AH, Brieve JL, Stokes B. Incidence of contrast-induced nephropathy in intensive care patients undergoing computerised tomography and prevalence of risk factors. *Anaesth Intensive Care* 2009;37:968-975.
46. Weisbord SD, Mor MK, Resnick AL, et al. Incidence and outcomes of contrast-induced AKI following computed tomography. *Clin J Am Soc Nephrol* 2008;3:1274-1281.
47. Rao QA, Newhouse JH. Risk of nephropathy after intravenous administration of contrast material: A critical literature analysis. *Radiology* 2006;239:392-397.

48. Cramer BC, Parfrey PS, Hutchinson TA, et al. Renal function following infusion of radiologic contrast material: A prospective controlled study. *Arch Intern Med* 1985;145:87-89.
49. Heller CA, Knapp J, Halliday J, et al. Failure to demonstrate contrast nephrotoxicity. *Med J Aust* 1991;155:329-332.
50. Matsushima K, Peng M, Schaefer EW, et al. Posttraumatic contrast-induced acute kidney injury: Minimal consequences or significant threat? *J Trauma* 2011;70:415-420.
51. Martin DR, Semelka RC. Health effects of ionising radiation from diagnostic CT. *Lancet* 2006;367:1712-1714.
52. Larson DB, Johnson LW, Schnell BM, et al. National trends in CT use in the emergency department: 1995-2007. *Radiology* 2011;258:164-173.
53. Brenner DJ, Hall EJ. Computed tomography — an increasing source of radiation exposure. *N Engl J Med* 2007;357:2277-2284.
54. Sharma OP, Oswanski MF, Sidhu R, et al. Analysis of radiation exposure in trauma patients at a level I trauma center. *J Emerg Med* 2011;41:640-648.
55. Tubiana M, Feinendegen LE, Yang C, et al. The linear no-threshold relationship is inconsistent with radiation biologic and experimental data. *Radiology* 2009;251:13-22.
56. Heidenreich WF, Paretzke HG, Jacob P. No evidence for increased tumor rates below 200 mSv in the atomic bomb survivors data. *Radiat Environ Biophys* 1997;36:205-207.
57. Winslow JE, Hinshaw JW, Hughes MJ, et al. Quantitative assessment of diagnostic radiation doses in adult blunt trauma patients. *Ann Emerg Med* 2008;52:93-97.
58. Mettler FA Jr, Huda W, Yoshizumi TT, et al. Effective doses in radiology and diagnostic nuclear medicine: A catalog. *Radiology* 2008;248:254-263.
59. Tien HC, Tremblay LN, Rizoli SB, et al. Radiation exposure from diagnostic imaging in severely injured trauma patients. *J Trauma* 2007;62:151-156.
60. Ott M, McAlister J, VanderKolk WE, et al. Radiation exposure in trauma patients. *J Trauma* 2006;61:607-610.
61. Mueller DL, Hatab M, Al-Senan R, et al. Pediatric radiation exposure during the initial evaluation for blunt trauma. *J Trauma* 2011;70:724-731.
62. Berrington de Gonzalez A, Mahesh M, Kim KP, et al. Projected cancer risks from computed tomographic scans performed in the United States in 2007. *Arch Intern Med* 2009;169:2071-2077.
63. Fox JC, Boysen M, Gharahbaghian L, et al. Test characteristics of focused assessment of sonography for trauma for clinically significant abdominal free fluid in pediatric blunt abdominal trauma. *Acad Emerg Med* 2011;18:477-482.
64. Feigin E, Aharonson-Daniel L, Savitsky B, et al. Conservative approach to the treatment of injured liver and spleen in children: Association with reduced mortality. *Pediatr Surg Int* 2009;25:583-586.
65. Davies DA, Pearl RH, Ein SH, et al. Management of blunt splenic injury in children: Evolution of the nonoperative approach. *J Pediatr Surg* 2009;44:1005-1008.
66. Schonfeld D, Lee LK. Blunt abdominal trauma in children. *Curr Opin Pediatr* 2012;24:314-318.
67. Hom J. The risk of intra-abdominal injuries in pediatric patients with stable blunt abdominal trauma and negative abdominal computed tomography. *Acad Emerg Med* 2010;17:469-475.
68. Kuppermann N, Holmes JF, Dayan PS, et al. Identification of children at very low risk of clinically-important brain injuries after head trauma: A prospective cohort study. *Lancet* 2009;374:1160-1170.
69. Osmond MH, Klassen TP, Wells GA, et al. CATCH: A clinical decision rule for the use of computed tomography in children with minor head injury. *CMAJ* 2010;182:341-348.
70. Viccellio P, Simon H, Pressman BD, et al. A prospective multicenter study of cervical spine injury in children. *Pediatrics* 2001;108:E20.
71. Allen TL, Cummins BF, Bonk RT, et al. Computed tomography without oral contrast solution for blunt diaphragmatic injuries in abdominal trauma. *Am J Emerg Med* 2005;23:253-258.
72. Tan KK, Liu JZ, Go TS, et al. Computed tomography has an important role in hollow viscus and mesenteric injuries after blunt abdominal trauma. *Injury* 2010;41:475-478.
73. Agostini C, Durieux M, Milot L, et al. Value of double reading of whole body CT in polytrauma patients [article in French]. *J Radiol* 2008;89:325-330.
74. Eurin M, Haddad N, Zappa M, et al. Incidence and predictors of missed injuries in trauma patients in the initial hot report of whole-body CT scan. *Injury* 2012;43:73-77.
75. Haley T, Ghaemmaghami V, Loftus T, et al. Trauma: The impact of repeat imaging. *Am J Surg* 2009;198:858-862.
76. Malhotra AK, Martin N, Jacoby M, et al. What are we missing: Results of a 13-month active follow-up program at a level I trauma center. *J Trauma* 2009;66:1696-1703.
- C. single-pass pan-scan
D. no imaging; observation only
2. Which of these patients would most benefit from a pan-scan?
A. any blunt trauma patient, regardless of mechanism
B. a hemodynamically stable patient injured in a motor vehicle crash, with no obvious physical findings, whose passenger died on scene
C. a hemodynamically stable 8-year-old boy with no physical findings after falling from a 5-foot ladder
D. a hemodynamically stable pedestrian struck at 10 mph with right knee and hip pain
3. Which of the following statements regarding contrast-induced nephropathy (CIN) in patients undergoing enhanced CT after trauma is correct?
A. The incidence may be as high as 12%, although this may be an associative rather than causative effect.
B. CIN is defined as an increase of 25% or 0.5 mg/dL in creatinine from baseline.
C. An Injury Severity Score of 16 or higher may be a risk factor for CIN.
D. The higher the dose of contrast administered, the higher the risk of CIN.
E. All of the above
F. Only A, B, and C are correct.
4. You are a physician at a community hospital and have just placed a chest tube to stabilize a woman who was injured in a motor vehicle collision. She arrived with a pneumothorax caused by multiple rib fractures. The FAST exam is positive, and you have to transfer her to the nearest trauma center for surgery. What is the most appropriate approach to imaging?
A. The patient is stable, so send her to CT for pan-scan.
B. The patient is stable, but imaging should not delay definitive management; transfer her immediately.
C. Scan the chest and abdomen, then transfer with accompanying images.
D. Send the patient to CT, because the receiving facility will probably want a scan.
5. Regarding pediatric blunt trauma patients, which of the following is true?
A. Children can be unreliable during history and physical, so they should be pan-scanned.
B. The FAST exam is a reliable way to rule out surgical abdominal injuries in stable children without abdominal pain.
C. Children receive the same effective radiation doses from CT as adults, but these doses carry a higher risk of secondary cancer.
D. Pan-scan is a good way to rule out surgical injuries for all pediatric trauma patients.
6. Which of the following statements about lifetime cancer risk associated with pan-scan is true?
A. The risk of trauma-related mortality is approximately six times that

CME/CNE Questions

1. Of the following, what is the most appropriate choice for a 48-year-old pedestrian who was struck by a motor vehicle with a moderate impact, is hemodynamically stable, and has no major physical exam findings except chest wall tenderness and a Glasgow Coma Scale (GCS) score of 13?
A. separate head and chest CT scans
B. head CT scan and chest radiograph only

- of CT-related cancer mortality in all-comer intermediate-level trauma patients.
- B. Age is a factor in CT-related cancer risk, and the risk is proportional to age.
 - C. The mean lifetime cancer risk after whole body CT in 3-year-old boys is 1 in 133 (0.75%) and 1 in 166 (0.60%) in girls.
 - D. The background radiation received on a yearly basis in an urban environment is almost as much as a single pan-scan.
 - E. B and D are correct.
 - F. A and C are correct.
7. Incidental findings are detected in approximately what percentage of trauma patients undergoing CT?
 - A. 1%
 - B. 10%
 - C. 33%
 - D. 50%
 8. In a recent poll of emergency physicians and trauma surgeons about the components of the pan-scan obtained for individual trauma patients the physicians thought were necessary, which of the following is true?
 - A. The ED physicians and trauma surgeons always agreed on which scans were necessary.
 - B. ED physicians desired 35% fewer scans, and < 1% of these undesired scans would have led to a predefined "critical action."
 - C. The trauma surgeons desired fewer scans than the ED physicians.
 - D. All scans were ordered at the discretion of the ED physician.
 9. Which of the following statements regarding clinician ability to rule out injury based on physical exam is true?
 - A. The sensitivity of ruling out injuries in specific body regions steadily decreases with higher pretest probabilities of injury.
 - B. Multiple studies have shown that physical exam is superior to CT scan for ruling out injuries.
 - C. All patients with abdominal injuries found on CT will have abdominal tenderness.
 - D. Ruling out injury based on physical exam findings is reliable in patients with altered mental status.
 10. Which of the follow statements about the use of CT is true?
 - A. The use of CT in EDs declined between 1998 and 2007.
 - B. A typical "pan-scan" is a non-contrast CT of the head, followed by contrast-enhanced scans of the neck, chest, abdomen, and pelvis.
 - C. A typical "pan-scan" is a non-contrast CT of the head, followed by contrast-enhanced scans of the neck, chest, abdomen, and pelvis and CT angiography of the extremities.
 - D. CT accounts for approximately 70% of radiation exposure in medical populations.
 - E. B and D are true.

CNE/CME Objectives

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

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

CNE/CME Instructions

HERE ARE THE STEPS YOU NEED TO TAKE TO EARN CREDIT FOR THIS ACTIVITY:

1. Read and study the activity, using the provided references for further research.
2. Log on to www.cmecity.com to take a post-test; tests can be taken after each issue or collectively at the end of the semester. *First-time users will have to register on the site using the 8-digit subscriber number printed on their mailing label, invoice, or renewal notice.*
3. Pass the online tests with a score of 100%; you will be allowed to answer the questions as many times as needed to achieve a score of 100%.
4. After successfully completing the last test of the semester, your browser will be automatically directed to the activity evaluation form, which you will submit online.
5. **Once the completed evaluation is received, a credit letter will be e-mailed to you instantly.** You will no longer have to wait to receive your credit letter.

To reproduce any part of this newsletter for promotional purposes, please contact:

Stephen Vance

Phone: (800) 688-2421, ext. 5511

Fax: (800) 284-3291

Email: stephen.vance@ahcmedia.com

To obtain information and pricing on group discounts, multiple copies, site-licenses, or electronic distribution please contact:

Tria Kreutzer

Phone: (800) 688-2421, ext. 5482

Fax: (800) 284-3291

Email: tria.kreutzer@ahcmedia.com

To reproduce any part of AHC newsletters for educational purposes, please contact:

The Copyright Clearance Center for permission

Email: info@copyright.com

Website: www.copyright.com

Phone: (978) 750-8400

Editor in Chief

Ann Dietrich, MD, FAAP, FACEP

Professor of Pediatrics
Ohio State University
Attending Physician
Nationwide Children's Hospital
Associate Pediatric Medical Director
MedFlight
Columbus, Ohio

Editorial Board

Mary Jo Bowman, MD, FAAP, FCP

Associate Professor of Clinical Pediatrics
Ohio State University College of Medicine
PEM Fellowship Director, Attending Physician
Children's Hospital of Columbus
Columbus, Ohio

Lawrence N. Diebel, MD

Professor of Surgery
Wayne State University
Detroit, Michigan

Robert Falcone, MD, FACS

Clinical Professor of Surgery
The Ohio State University
College of Medicine
Columbus, Ohio

Theresa Rodier Finerty, RN, MS, CNA, BC

Director, Emergency and Trauma

Services,
OSF Saint Francis Medical Center
Peoria, Illinois

Dennis Hanlon, MD, FAAEM

Vice Chairman, Academics
Department of Emergency Medicine
Allegheny General Hospital
Pittsburgh, Pennsylvania

Jeffrey Linzer Sr., MD, FAAP, FACEP

Assistant Professor of Pediatrics and Emergency Medicine
Emory University School of Medicine
Associate Medical Director for Compliance
Emergency Pediatric Group
Children's Healthcare of Atlanta at Egleston and Hughes Spalding
Atlanta, Georgia

S.V. Mahadevan, MD, FACEP, FAAEM

Associate Professor of Surgery/
Emergency Medicine
Stanford University School of Medicine
Associate Chief, Division of
Emergency Medicine
Medical Director, Stanford University
Emergency Department
Stanford, California

Janet A. Neff, RN, MN, CEN

Trauma Program Manager

Stanford University Medical Center
Stanford, California

Ronald M. Perkin, MD, MA, FAAP, FCCM

Professor and Chairman
Department of Pediatrics
The Brody School of Medicine at East Carolina University
Medical Director, Children's Hospital
University Health Systems of Eastern Carolina
Greenville, North Carolina

Andrew D. Perron, MD, FACEP, FACSM

Professor and Residency Program Director,
Department of Emergency Medicine,
Maine Medical Center
Portland, Maine

Steven A. Santanello, DO

Medical Director, Trauma Services
Grant Medical Center
Columbus, Ohio

Eric Savitsky, MD

Associate Professor Emergency Medicine
Director, UCLA EMC Trauma Services and Education
UCLA Emergency Medicine
Residency Program
Los Angeles, California

Thomas M. Scalea, MD

Physician-in-Chief
R Adams Cowley Shock Trauma Center
Francis X. Kelly Professor of Trauma Surgery
Director, Program in Trauma
University of Maryland School of Medicine

Perry W. Stafford, MD, FACS, FAAP, FCCM

Professor of Surgery
UMDNJ Robert Wood Johnson Medical School
New Brunswick, New Jersey

Steven M. Winograd, MD, FACEP

St. Barnabus Hospital, Core Faculty
Emergency Medicine Residency Program
Albert Einstein Medical School,
Bronx, New York

CNE Nurse Reviewer

Sue A. Behrens, APRN, BC

Director of Emergency/ECU/Trauma Services
OSF Saint Francis Medical Center
Peoria, IL

© 2012 AHC Media. All rights reserved.

Trauma Reports[™] (ISSN 1531-1082) is published bimonthly by AHC Media, a division of Thompson Media Group, LLC, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Senior Vice President / Group Publisher: Donald R. Johnston

Executive Editor: Shelly Morrow Mark

Managing Editor: Leslie Hamlin

POSTMASTER: Send address changes to *Trauma Reports*, P.O. Box 105109, Atlanta, GA 30348.

Copyright © 2012 by AHC Media, Atlanta, GA, a division of Thompson Media Group LLC. All rights reserved. Reproduction, distribution, or translation without express written permission is strictly prohibited.

Subscriber Information

Customer Service: 1-800-688-2421

Customer Service E-Mail:
customerservice@ahcmedia.com

Editorial E-Mail:
shelly.mark@ahcmedia.com

World Wide Web page:
<http://www.ahcmedia.com>

FREE to subscribers of *Emergency Medicine Reports* and *Pediatric Emergency Medicine Reports*

Subscription Prices

United States

\$249 per year. Add \$17.95 for shipping & handling

Multiple Copies

Discounts are available for group subscriptions, multiple copies, site-licenses or electronic distribution. For pricing information, call Tria Kreutzer at 404-262-5482.

All prices U.S. only. U.S. possessions and Canada, add \$30 postage plus applicable GST.

Other international orders, add \$30.

Accreditation

AHC Media is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

AHC Media designates this enduring material for a maximum of 2.5 *AMA PRA Category 1 Credits*[™]. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Approved by the American College of Emergency Physicians for a maximum of 2.5 hour(s) of ACEP Category 1 credit.

AHC Media is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.

This activity has been approved for 1.5 nursing contact hours using a 60-minute contact hour.

Provider approved by the California Board of Registered Nursing, Provider # 14749, for 1.5 Contact Hours.

This is an educational publication designed to present scientific information and opinion to health professionals, to stimulate thought, and further investigation. It does not provide advice regarding medical diagnosis or treatment for any individual case. It is not intended for use by the layman. Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. Clinical, legal, tax, and other comments are offered for general guidance only; professional counsel should be sought for specific situations.

This CME/CNE activity is intended for emergency, family, osteopathic, trauma, surgical, and general practice physicians and nurses who have contact with trauma patients.

It is in effect for 24 months from the date of publication.

© 2012 AHC Media. All rights reserved.

AHC Media