

# Critical Decisions

## in Emergency Medicine™



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Elevated cardiac troponin concentrations have become a useful marker for acute coronary syndrome, but the definition of "elevated" is problematic, and other conditions, too, can cause increased troponin levels. Emergency physicians must understand the strengths and limitations of this marker in identifying acutely ill patients.
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Thermal burns are significant causes of injury and death; half a million people seek medical care for burn injuries each year in the United States. Emergency physicians must make rapid decisions regarding intubation, fluid resuscitation, pain management, and wound management in order to minimize morbidity and mortality in these patients.



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# SAMPLE ISSUE

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# Cardiac Troponin

Here's where we show you the most important take-home points of each lesson.



## ■ Objectives

**On completion of this lesson, you should be able to:**

1. Outline a plan for dealing with indeterminate or discordant cardiac biomarker results.
2. Discuss the kinetics of release into the serum of commonly used cardiac biomarkers.
3. Explain the significance of troponin elevations in patients with renal failure.
4. Describe the non-coronary artery disease causes of troponin elevations.
5. Describe the usefulness of cardiac biomarkers in non-coronary artery disease processes.
6. Explain the appropriate role for cardiac biomarkers in risk stratification for acute coronary syndrome.

## ■ From the EM Model

- 1.0 Signs, Symptoms, and Presentations
  - 1.3 Chest Pain

Alexander T. Limkakeng, Jr, MD, FACEP, and Jesmin Ehlers, MD

Cardiovascular disease remains a leading cause of death in the United States, and acute coronary syndrome (ACS), particularly, is concerning. Patients with ACS can present with a wide variety of symptoms, none of which is sufficient to rule in or rule out the disease.<sup>1</sup> This makes it difficult to identify which patients need extensive evaluation. Physicians fear missing a myocardial infarction (MI) in the emergency department because it is a leading cause of medicolegal liability. On the other hand, vast resources are spent on ruling out ACS in patients who present with concerning symptoms.

Previous studies have demonstrated that the patient history, physical examination, and ECG are insufficient to identify a patient population that can be safely discharged from the emergency department.<sup>2,3</sup> This challenge has led to the development of new technologies such as 15-lead ECGs, neural networks, emergency department stress testing, and computed tomography coronary angiography to improve identification of ACS in the emergency department.

Currently, however, serial ECG and serum cardiac biomarkers are the cornerstone of ACS evaluation. This is with good reason. Cardiac troponin elevations have been shown to predict which patients with ACS symptoms are at risk for major cardiac adverse events and therefore will benefit from aggressive treatment strategies.<sup>4</sup> Cardiac biomarkers are relatively inexpensive, noninvasive,

reproducible, fast, and objective.

They do not require specialty experts such as radiologists, cardiologists, or nuclear cardiologists to interpret.

As such, one would think that there are few issues surrounding the use of cardiac troponins. In reality, a number of clinical scenarios arise in which the correct interpretation of cardiac troponins becomes difficult. The emergency physician must be familiar with how troponins work and, just as with any other test, know how to interpret the results of these tests. As the number of options for ACS evaluation increases, correct interpretation of cardiac troponins becomes more important.

## Case Presentations

### ■ Case One

A 45-year-old man presents with exertional substernal diffuse chest pressure radiating to both shoulders; it has bothered him intermittently for the past 2 days. The most recent episode of pain is the most severe and started while the patient was carrying boxes up the stairs just prior to his presentation. Past medical history is significant for hypertension and hypercholesterolemia.

The patient called for an ambulance and was given aspirin and three nitroglycerin tablets sublingually, which provided relief of his pain. His vital signs are blood pressure 165/87, pulse rate 85, respiratory rate 17, and temperature of 37.8°C (100°F). On physical examination, the patient is an obese, diaphoretic man who appears to be in

## Critical Decisions

- What is the significance of discordance between CK-MB band and troponin values?
- What is the best approach to the patient with “indeterminate” troponin elevations?
- What is the best approach to patients with renal failure with increased troponin levels?
- What are the non-coronary artery disease causes of elevated troponin?
- What is the significance of elevated troponin in the setting of non-coronary artery disease presentations?

mild discomfort. The examination is otherwise unremarkable.

His ECG shows borderline ST-segment depression in leads aVF, II, and III, with normal sinus rhythm. His myoglobin level is 214 mcg/L (reference range for normal is <110 mcg/L), creatine kinase-MB (CK-MB) is 14 mg/dL (reference range for normal is <12 mg/dL), and troponin I is 0.9 ng/mL (reference range 0.02-1.2 ng/mL). His chest radiograph is normal.

A repeat ECG by a cardiologist is in the disposition of the patient. The cardiologist recommends serial ECGs in the observation room and testing the following:

Everybody likes cases!  
They help you relate  
what you learn to  
what you do.



### ■ Case Two

A 67-year-old man with end-stage renal disease and who is on dialysis presents with chest pain. The pain is diffuse across his chest and feels like a pressure. The pain started as a twinge last night, then worsened this morning. He feels like he cannot get a full breath in and is winded even with walking across the room. He has had some nonproductive coughing. He missed his dialysis session yesterday because he said he felt too ill to go. He notes that his legs are swollen but at their baseline levels. His past medical history is significant for hypertension, diabetes, and high cholesterol, and he has a 20 pack-year history of smoking.

Physical examination shows an obese man in moderate distress from pain. Vitals signs are blood pressure 187/101, pulse rate 101, respiratory rate 22, temperature 37.9°C (100.2°F), and pulse oximetry 95% on room air.

Further examination reveals normal S<sub>1</sub> and S<sub>2</sub>, 2/6 early systolic murmur, slightly labored breathing with slight bibasilar rales, nontender abdomen, 2+ bilateral lower extremity edema with good pulses, and mild jugular venous distention.

The ECG shows a rate of 101, sinus rhythm, and left ventricular hypertrophy with concave ST-segment elevations in leads V<sub>4</sub>-V<sub>6</sub>. The chest radiograph shows mild cardiomegaly and mild pulmonary edema, bilaterally. His electrolytes are notable for marked elevation of creatinine (7.9 mg/dL) and BUN (61 mg/dL), as well as potassium of 5.6 mg/dL. His first set of cardiac markers shows a creatine kinase level of 561 units/L (reference range normal <210 units/L), CK-MB of 12 mg/dL (reference range for normal is <12 mg/dL), and troponin T level of 1.2 ng/mL (reference range 0.01-0.1 ng/mL).

A cardiologist is consulted and recommends sending the patient to his nephrologist for dialysis and discharge, stating that renal failure patients always have false-positive troponin elevations.

### ■ Case Three

A 39-year-old woman presents because she has had constant chest pain for the past 3 days and then a syncopal episode. The chest pain developed suddenly following a car drive across country. It is diffuse across her chest and worse with deep breaths or coughing. Her cough is nonproductive. She does not have fever, nausea, or vomiting. She takes oral contraceptives and smokes.

The physical examination is remarkable for blood pressure 100/60,

pulse rate 111, respiratory rate 26, temperature of 36.7°C (98.1°F), and pulse oximetry 92% on room air. The patient appears mildly uncomfortable and is slightly diaphoretic. The rest of the examination, including examination of the heart and lungs, is unremarkable.

The ECG shows sinus tachycardia, without ST-segment elevations. There are T-wave inversions in leads V<sub>1</sub>-V<sub>3</sub>. The chest radiograph is normal. Creatine kinase level is 124 U/L, CK-MB is 7 mg/dL, and troponin I level is 2.4 mg/mL (0.04 - 2 mg/mL). A computed tomography scan of the chest reveals a large pulmonary embolism just distal to the bifurcation of the pulmonary artery. The patient is initiated on heparin, and the hospitalist accepts the patient for admission to a general telemetry bed.

## Cardiac Markers

Creatine kinase is an enzyme found in many parts of the body and can be fractionated into three isoenzymes: MM, MB, and BB. CK-MB is present in high concentrations in the myocardium but is also present in the lungs, small intestine, uterus, prostate, and healthy skeletal muscle. In response to cardiac injury, levels of CK-MB start to increase from baseline levels in 4 to 8 hours, peak in 12 to 24 hours, and usually return to normal within 3 days.<sup>5</sup>

Troponin is a structural protein found in striated muscle that is responsible for calcium processing. There are unique structural features of the cardiac muscle version of troponin that distinguish it from skeletal muscle versions of troponin and allow for detection in the serum. Cardiac

troponin occurs in three forms: I, T, and C. Most of the troponin is complexed to the contractile apparatus. A small amount (3% to 6%) exists that is not structurally bound. This small amount has been termed the “cytosolic pool.” It is released almost immediately, is detectable within 6 hours of coronary occlusion, and remains in the bloodstream for up to 10 days.<sup>6</sup>

**CRITICAL DECISION**

**What is the significance of discordance between CK-MB band and troponin values?**

For many years, measurement of CK-MB was the gold standard for diagnosis of MI. Troponins have rightly supplanted CK-MB as the gold standard because of their higher specificity to cardiac tissue. However, one advantage that CK-MB and other biomarkers still have is that elevations are detectable in the blood 1 to 2 hours faster than troponin using traditional assays. However, due to recent changes in proposed cutoffs and national consensus guidelines and the emergence of highly sensitive troponin assays, some experts now argue that troponin elevations can be detected much earlier—within 2 to 3 hours of infarction. These experts argue that CK-MB should no longer have a role in ACS risk stratification and, indeed, some institutions are discontinuing the use of CK-MB.<sup>7</sup> For physicians working at hospitals that still use CK-MB and who are not comfortable with the fact that troponin values at very low levels can be imprecise, CK-MB should be considered an early marker of infarction.

There is evidence to support this approach. Storrow et al evaluated the prognostic significance of discordant markers (when the results of two different markers conflict). Out of 8,769 eligible registry patients, CK-MB was positive but troponin negative in 4.9%. The odds ratio for ACS in these patients compared to patients for whom both markers were negative was 2.2 (95% confidence

interval 1.7-2.8).<sup>8</sup> Thus, patients with isolated CK-MB elevations should be considered higher risk for ACS, particularly early in the patient presentation.

**CRITICAL DECISION**

**What is the best approach to the patient with “indeterminate” troponin elevations?**

The most recent Joint European Society of Cardiology/American College of Cardiology Committee defines myocardial infarction by elevation of troponins. They define such an elevation as a measurement “exceeding the 99th percentile of a reference control group.” This is a lower level than almost all of the manufacturer-reported cutoffs. Unfortunately, at such low troponin levels, the precision of measurements decreases to unacceptable levels for almost all currently used troponin assays. The Committee statement also mandates that the coefficient of variation (a measure of assay precision that varies with the troponin level) be less than 10% at the cutoff level<sup>9</sup> (Table 1). The troponin level at which current assays achieve this mandated precision is higher than the aforementioned 99th percentile, creating an “indeterminate” zone between the Committee-recommended cutoff, the level at which the coefficient of variation is less than 10%, and the manufacturer-recommended cutoff. Nevertheless, there is some uncertainty as to what to do for patients with troponin in this range.

There have been several studies indicating that the risk associated with troponin elevation increases linearly above any detectable level. In one such study, the authors created a receiver operating curve for troponin using data from their own emergency department population and derived their own cutoff at which 99% of all ACS patients were identified. This derived cutoff level was lower than the manufacturer derived cutoff. They then determined the rates of cardiovascular events for patients with levels below normal (undetectable), below their cutoff, above their cutoff, and above the manufacturer cutoff. They found that the risk of adverse cardiovascular events essentially rose linearly in the “indeterminate” range between the manufacturer’s detectable and cutoff levels (Figure 1).<sup>12</sup>

Thus, cutoff values for “positive” and “negative” are necessarily artificial. Over the long term, it is necessary to ensure that there are no false-positives to prevent unnecessary interventions. However, for the emergency physician, it is probably more important to prevent any false-negatives—to avoid missing potential ACS. Keeping in mind that there are many reasons for chronic, low levels of troponin elevations, it is thus important to recognize the potential significance of troponin elevations below the manufacturer-recommended cutoffs. Particularly, in the course of disease when troponin might still be rising, patients with undetectable troponin should

**User-friendly quick-reference tables and figures – because sometimes words need pictures!**

**Table 1.**

Comparison of manufacturer 99th percentile and 10% coefficient of variation (COV) cutoffs<sup>10,11</sup>

| Manufacturer assay      | 99th percentile (ng/mL) | 10% COV cutoff (ng/mL) |
|-------------------------|-------------------------|------------------------|
| Abbott AxSYM            | 0.5                     | 0.8                    |
| Beckman-Coulter Access  | 0.04                    | 0.06                   |
| Biosite Triage          | 0.19                    | 0.5                    |
| Dade Behring Stratus CS | 0.07                    | 0.06                   |
| i-STAT                  | 0.08                    | 0.1                    |
| Boche Elecsys           | 0.01                    | 0.035                  |

be considered higher risk unless there is another explanation for their levels.

Recently, high-sensitivity troponin assays have been introduced that can detect troponin at levels that are orders of magnitude lower than those of current-generation assays. The role for these new assays in clinical care has not yet been definitively established.

## Renal Failure and Cardiac Biomarkers

The management of troponin elevations in patients with renal failure has been a source of controversy. In general, patients with chronic kidney disease are at almost six times higher risk for early death than those without renal disease, and many of these deaths are from cardiac causes.<sup>13</sup> However, patients with renal failure often demonstrate a chronic troponin elevation even in the absence of any identifiable coronary disease.<sup>14</sup> Since there is risk associated with aggressive intervention in these patients, false-positive troponin tests are potentially dangerous.

There is a common misperception that all troponin elevations in renal failure patients are false positives. Troponin is not renally cleared and therefore not affected by dialysis. Rather it is hypothesized that chronic troponin elevations in renal failure

are due to ventricular hypertrophy, chronic fluid overload, or endothelial dysfunction.<sup>6</sup> In fact, even in asymptomatic outpatients with renal failure, the identification of chronic troponin elevation is associated with poorer long-term outcome.<sup>15</sup> In one metaanalysis of such studies, troponin T but not troponin I elevation was associated with increased mortality in 12 to 24 months.<sup>16</sup>

Unfortunately, there is a relative dearth of high-quality clinical studies informing clinicians on how to rapidly differentiate a baseline chronic troponin elevation from an acute cardiac event. One sub-analysis of the large multicenter GUSTO IV trial revealed that patients with elevated troponin T levels were at a higher risk for adverse cardiac events and death in 30 days regardless of their renal function.<sup>17</sup> Kontos and colleagues<sup>18</sup> likewise found that troponin elevations were associated with increased mortality regardless of kidney function in 3,774 consecutive patients being admitted to the hospital for potential cardiac disease. On the other hand, Van Lente et al<sup>19</sup> found peak troponin T levels to have sensitivity and specificity of 45% and 72%, respectively in the renal disease group. Peak troponin I levels had a sensitivity and specificity of 21% and 89%, respectively. These values

are markedly lower than those for patients without renal disease.

### CRITICAL DECISION

#### What is the best approach to patients with renal failure with increased troponin levels?

The observation that elevated troponin and underlying renal disease are both independently associated with worse cardiovascular outcome needs to be balanced with an acknowledgment of the phenomena of chronic elevations and the risk of intervention. It is important to recall that the troponin test is one of several modalities of risk stratification. Therefore, troponin levels need to be interpreted in the context of the patient's other clinical risk indicators. Given renal disease patients' underlying increased risk, it is best to take a more conservative approach with regard to admission for serial markers and ECGs. Knowing the trend of troponin elevation can be useful. One available strategy is simply to repeat the test after several hours. The sensitivity and specificity of troponin tests increase with serial testing.<sup>10</sup> It is also recommended, whenever possible, to determine whether the patient's current level is an increase over a chronic baseline elevation.

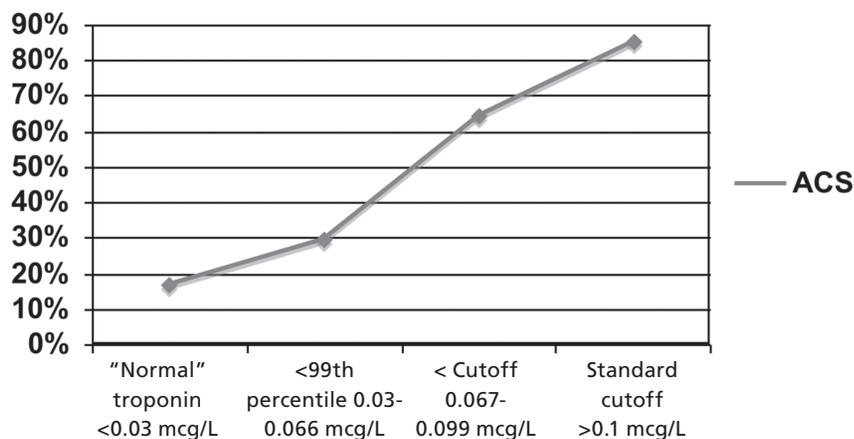
### CRITICAL DECISION

#### What are the non-coronary artery disease causes of elevated troponin?

Since the introduction of troponin assays, it has been observed that non-coronary artery diseases are associated with troponin elevations. Initially, there was some confusion as to whether this represented secondary ischemia from the primary disease process or an effect of the primary disease itself. It now is clear that troponin levels reflect the damage caused by disease processes other than acute cardiac ischemia.<sup>20</sup> Table 2 shows a partial list of the many conditions associated with serum troponin elevations. In the next section, we will discuss those causes that are commonly seen in the emergency department.

**Figure 1.**

Cardiovascular event rates in patients with troponin levels in the "indeterminate" zone<sup>12</sup>



**CRITICAL DECISION**

**What is the significance of elevated troponin in the setting of non-coronary artery disease presentations?**

In pulmonary embolism (PE), the sudden increase in pulmonary artery pressure is hypothesized to lead to right ventricular overload and cardiac cell injury. It has been shown that troponin elevations correlate with the size of the PE as well as with clinical indicators such as severe hypoxemia, prolonged hypotension, cardiogenic shock, and the need for inotropic therapy or mechanical ventilation.<sup>21</sup> A metaanalysis demonstrated that patients with confirmed PE and troponin elevations had a 30-day mortality rate of 19.7% compared to a rate of 3.7% for those with confirmed PE and normal troponin levels. This was true even for those with a normal blood pressure on admission.<sup>22</sup>

Troponin elevations have also been observed in patients with congestive heart failure (CHF), both during acute exacerbations and at baseline. As is the case for patients with renal failure, it is thought that troponin elevations in CHF are due to chronic fluid overload and that they identify patients with a worse long-term prognosis. In particular, they identify those with acute CHF exacerbations with worse prognosis.<sup>23</sup>

Patients with sepsis and a wide variety of other critical illnesses demonstrate troponin elevations. There is debate over which of the many proposed mechanisms is responsible, including regional wall motion abnormalities, microvascular thrombi, and direct toxic effect of endotoxins or inflammatory reactants.<sup>24</sup> It has also been shown that these elevations correlate with higher mortality rates, even when controlling for preexisting cardiac disease.<sup>25</sup>

It can be seen, then, that troponin elevations are present in a wide spectrum of non-coronary artery disease processes. The key is recognizing that such elevations do not always reflect cardiac ischemia,

but rather identify patients who may need more aggressive intervention and monitoring for their primary disease process. Once again, other tools for determining risk for ACS should be used in conjunction with markers to determine if the patient is suffering from both ACS and another disease process.

**Case Resolutions**

**■ Case One**

The emergency physician attending the 45-year-old man with exertional chest pain disagreed with the recommendation from the cardiology consultant. The emergency physician insisted that the cardiologist examine the patient in the emergency department before the patient was sent to the observation unit. After seeing the patient in the emergency department, the cardiologist admitted the patient to the cardiac ICU instead, where the patient's second set of cardiac markers showed markedly elevated levels. The patient was started on antithrombotic medications in preparation for cardiac catheterization the next day. The angiogram revealed diffuse three-vessel disease with more than 70%

stenosis. The patient underwent successful coronary artery bypass graft surgery and had an uneventful hospital course.

**■ Case Two**

The 67-year-old man with renal disease was sent to a dialysis center for treatment. He continued to feel worse, and 3 hours later he became hypotensive and increasingly dyspneic. An ambulance was called, and the patient was noted to decompensate, with alteration of mental status and evolution of sinus bradycardia into ventricular fibrillation. The paramedics started cardiopulmonary resuscitation and had given a first shock as they arrived in the emergency department. There, advanced cardiac life support protocols continued; the patient was intubated and was shocked a second time. The patient went into pulseless electrical activity arrest but responded to epinephrine, atropine, and bicarbonate, returning to sinus bradycardia. A repeat troponin level was 5.6 mg/mL. The patient was admitted to the ICU in unstable condition on vasopressor drugs, and hypothermia was initiated. He was

**Table 2.**

Non-coronary artery disease causes of troponin elevations<sup>6</sup>

|   |
|---|
| Acute neurologic disease (including cerebrovascular accident, subarachnoid hemorrhage)  |
| Aortic valve disease  |
| Burns, especially if more than 30% total body surface area is involved  |
| Cardiomyopathy (including hypertrophic obstructive cardiomyopathy, left ventricular hypertrophy)  |
| Congestive heart failure (acute and chronic)  |
| Drug toxicity (doxorubicin, fluorouracil, trastuzumab, snake venoms)  |
| Hypertension  |
| Hypotension   |
| Hypothyroidism  |
| Infiltrative diseases (amyloidosis, hemochromatosis, sarcoidosis, and scleroderma)  |
| Inflammatory diseases (myocarditis, pericarditis, Kawasaki disease)   |
| Pulmonary embolism, severe pulmonary hypertension   |
| Renal failure   |
| Sepsis  |
| Trauma (including blunt contusion, ablation, pacing, internal cardioverter-defibrillator firings, endomyocardial biopsy, cardiac surgery) |

believed to be too unstable for cardiac catheterization, and medical therapy was pursued. Two days later, in light of the patient's continued decline, the patient's family elected to withdraw aggressive measures, and the patient died.

### Helpful risk management and patient safety tips in every lesson.



#### Pearls

- Use serial testing of cardiac markers whenever possible.
- Compare current biomarker levels with previous values, if they are available, to identify patients with chronic baseline elevations.
- CK-MB and myoglobin may still have a role in identifying early ACS.
- The precision of troponin assay results is reduced at very low levels of troponin; however, even minor elevations are associated with a higher rate of cardiac events and death.
- Knowledge of test characteristics is sometimes required to risk stratify patients.

#### Pitfalls

- Relying on cardiac biomarkers and failing to consider the clinical context.
- Assuming that troponin elevation is always ACS.
- Ascribing an elevated troponin result in patients with chronic renal insufficiency to their renal disease without consideration of other possibilities.

transferred rapidly to the ICU where an echocardiogram revealed right ventricular dilation and hypokinesis. The patient was given thrombolytic therapy, and her condition improved over the next several days. She was transferred to the floor, and discharged 7 days later. When the case was raised in quality assurance committee, the question arose as to whether there were any clinical indicators of potential deterioration.

#### Summary

Cardiac biomarkers are an integral part of the risk stratification of ACS. Given this role, it is important for emergency physicians to be very familiar with their use and limitations. In particular, emergency physicians must be wary of patients with discordant or "indeterminate" markers. They also must use other factors such as the history and physical examination when assessing patients with potentially confounding conditions such as a history of renal failure or congestive heart failure. It is important not only to interpret marker levels in clinical context, but also to use past levels and serial markers to help make decisions in difficult cases.

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# The LLSA Literature Review

"The LLSA Literature Review" is a "2012 Lifelong Learning and Self-Assessment Reading List." These articles are available online in the ACEP LLSA (http://www.acep.org/llsa) and on the ABEM Web site.

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## Etomidate Versus Ketamine For Rapid Sequence Intubation in Acutely Ill Patients: A Multicenter Randomised Controlled Trial

Reviewed by Kohei Hasegawa, MD, and J. Stephen Bohan, MS, MD, FACEP; Harvard Affiliated Emergency Medicine Residency; Brigham and Women's Hospital

Jabre P, Combes X, Lapostolle F, et al. Etomidate versus ketamine for rapid sequence intubation in acutely ill patients: a multicenter randomised controlled trial. *Lancet*. 2009;374:293-300.

Critically ill patients often require emergency endotracheal intubation using rapid sequence intubation (RSI). Etomidate is the sedative-hypnotic medication that has hemodynamic tolerance and that is most often used in RSI. However, its use has been challenged because it can cause a reversible adrenal insufficiency by dose-dependant inhibition of 11 $\beta$ -hydroxylase. A possible alternative to etomidate is ketamine, which is not known to inhibit the adrenal axis. In this randomized, single-blind, controlled, double-arm trial, researchers compared early and 28-day morbidity after a single dose of etomidate (0.3 mg/kg) or ketamine (2 mg/kg) for induction during RSI at 65 ICUs and 12 emergency departments or prehospital systems in France. All patients received intravenous succinylcholine immediately after the trial medications and continuous sedation with midazolam combined with fentanyl or sufentanil after tube placement was confirmed.

Six hundred fifty-five adult patients who needed sedation for emergency intubation were enrolled; 469 patients were analyzed for primary and secondary endpoints, excluding patients who died before reaching the hospital and those discharged from the ICU within 3 days. Final diagnoses were categorized as trauma (22%), sepsis (16%), and other (stroke, drug poisoning, cardiogenic shock, respiratory failure; 62%). Adrenal insufficiency occurred significantly more often in the etomidate arm than the ketamine arm (86% vs. 48%; OR 6.7). However, there was no significant difference between

groups in the primary endpoint of maximum sequential organ failure assessment (SOFA) scores during the first 3 days in ICU or the secondary endpoints of intubation conditions, catecholamine use, length of stay in ICU, and 28-day mortality. No serious adverse events with either study drug were recorded.

This study demonstrated measurable adrenal suppression but no evidence of adverse outcome related to a single bolus of etomidate for RSI in critically ill patients. Additionally, ketamine may be a safe and effective alternative to etomidate for emergency endotracheal intubation.

### Highlights

- Despite the hemodynamic benefits of etomidate, some have challenged its use for RSI in critically ill patients, citing concerns about adrenal insufficiency.
- This study demonstrated that one etomidate bolus is not associated with a significant increase in morbidity or mortality compared with ketamine in patients admitted to the ICU even though the proportion of patients developing adrenal insufficiency was significantly higher in the etomidate arm.
- Ketamine may be a safe and effective alternative to etomidate for emergency endotracheal intubation.

## Lesson 2

# Thermal Burns

Ronald V. Hall, MD, Lisa S. Watts, MD, and Samer Bashiti, DO

## ■ Objectives

**On completion of this lesson, you should be able to:**

1. Discuss the classification of burns.
2. Explain how to approximate the percentage of total body surface area burned in adults and children.
3. Describe common clinical clues suggestive of inhalation injury.
4. Discuss optimal fluid resuscitation in burn victims.
5. Discuss the appropriate topical management and dressing of burns.
6. Explain the criteria for appropriate burn center referral.

## ■ From the EM Model

18.0 Traumatic Disorders

18.1 Trauma (Cutaneous injuries)

↑  
We choose topics from the official emergency medicine core content.

Approximately 500,000 burn injuries per year receive treatment at hospital emergency departments, outpatient clinics, free-standing urgent care centers, and private physician offices.<sup>1</sup> Of the patients seeking medical care, 40,000 are hospitalized for treatment of their injuries, with more than 60% admitted to burn centers. The percentage of burn center admissions has increased over the past several decades as a result of improvements in emergency care of burn patients and transportation. The survival rate of patients admitted to burn centers from 1995 to 2005 was 94.4%.<sup>1</sup> Males sustain more burns than females, in a ratio of approximately 2:1, and burn injuries are more common in Caucasians.<sup>1,2</sup> The two most common etiologies for burns are fire/flame and scalds. Most burn injuries occur in the home and often involve the consumption of alcohol.<sup>1,2</sup> There are an estimated 4,000 deaths per year from fire and burns. Risk factors for death include age above 60 years, inhalation injury, and involvement of more than 40% total body surface area (TBSA).<sup>2-4</sup>

## Case Presentations

### ■ Case One

A 90-kg, 33-year-old man is brought to the emergency department by fire rescue from the site of a house fire. Vital signs are blood pressure 106/55, pulse rate 117, respiratory rate 8, and temperature 35.9°C (96.6°F). He appears disoriented and combative and has soot around his face and

mouth, singed facial and scalp hair, and partial thickness burns to the abdomen and both lower extremities. On initial survey, his airway appears patent but his breathing appears labored, and he has inspiratory stridor.

### ■ Case Two

A 2-year-old girl is rushed to the emergency department by her parents with a scald burn. The child was burned when she pulled a pot of hot liquid down off of a kitchen counter. The patient weighs 12 kg and has a height of 85 cm. Vital signs are blood pressure 104/58, pulse rate 132, respiratory rate 32, and temperature 36.9°C (98.4°F). She appears severely agitated, is crying, is not consolable, and has burns to the right trunk, lower extremities, and face. Most of the injured areas have large blisters forming toward the center of the burns, and the peripheral areas have moist, bright red edges that are extremely painful to palpation and easily blanch with pressure.

### ■ Case Three

An 18-year-old man presents to the emergency department after sustaining several burns to his upper extremities and chest while playing with some fireworks. He has stable vital signs and no signs of respiratory involvement, although he appears to be in significant pain. The burn areas are mostly superficial, but he has small, discrete superficial and deep partial thickness burns that are approximately 5% of TBSA and spare his palms, elbows, neck, and face.

In every clinical presentation, the questions you ask yourself, the decisions you need to make.

## Critical Decisions

- How should the total body surface area involved in burns be calculated?
- Is it appropriate to clinically observe a stable patient with evidence of smoke inhalation?
- What methods are useful for achieving optimal fluid resuscitation in burn patients?
- How should adequate fluid resuscitation be assessed?
- What options exist for pain management in burn patients?
- Should emergency physicians unroof blisters?
- When should an emergency physician transfer a patient to a burn center?

## Classification of Burns

The traditional classification of burns (first-, second-, and third-degree) has been replaced with a classification that more accurately reflects depth. The term first-degree burn has been replaced with the term superficial burn, second-degree burn with superficial and deep partial-thickness burn, and third-degree burn with full-thickness burn. The term fourth-degree burn is still currently used.<sup>5-7</sup>

*Superficial burns* affect only the epidermis. They are typically nonbullous, painful, and red; they blanch with pressure and heal without scarring. They typically heal without intervention in 3 to 6 days. The most common etiologies are ultraviolet light (eg, sunburn) and flash burns.<sup>5-7</sup>

*Superficial partial-thickness burns* are usually caused by scalds (eg, spilled or splashed hot liquids). They involve the epidermis and superficial dermis. This type of burn is painful, moist, and erythematous and blanches with pressure. Blister formation distinguishes partial-thickness burns from superficial burns. Healing time is 7 to 20 days, and the burn usually heals without scarring. These injuries can cause changes to skin pigmentation.<sup>5-7</sup>

*Deep partial-thickness burns* extend into the deeper dermis and damage glandular tissue. These can develop from scald, flame, oil, or grease burns. They are wet or waxy dry, have blisters that are easily unroofed, and have a patchy, cheesy white to red variable color. Deep partial-thickness burns are painful to pressure only.

They will take more than 21 days to heal. There is a severe risk of contracture formation.<sup>5-7</sup> In partial-thickness burns, hair follicles may serve as a reservoir for undamaged epithelial cells that form the new skin layers; therefore, patients with these burns are much less likely to require skin grafting.

*Full-thickness burns* extend through and completely destroy the dermis, including the nerve endings. They can be caused by scald/immersion, flame, steam, oil, grease, chemicals, or high-voltage electricity. These injuries are nonbullous, nonpainful, dry, and inelastic; they can appear waxy white, leathery gray, or charred and black. Patients with this type of burn have sensation only to deep pressure. Spontaneous healing is not possible. If the burn is larger than 1 cm, skin grafting is always necessary. There is a very severe risk of contracture.<sup>5-7</sup> It is very difficult to distinguish deep partial-thickness burns from full-thickness burns; however, making the distinction is crucial in the consideration of skin repair. The absence or presence of a hair follicle can help distinguish the two and makes the difference in skin regeneration. If hair follicles are visible after débridement, the injury is a partial-thickness burn. If no follicle is visible, it is a full-thickness burn.<sup>6</sup> Skin repair requires a source of epithelial cells that can multiply and repair the defect.

*Fourth-degree burns* extend through the dermis into underlying tissue such as fascia, muscle, bone, or other tissues. These severe burns require extensive débridement and

complex reconstruction such as skin grafts or musculoskeletal flap placement. They are associated with significant functional and cosmetic impairment.<sup>6,7</sup>

### CRITICAL DECISION

**How should the total body surface area involved in burns be calculated?**

Accurately estimating the percentage of TBSA involvement in a burned patient is essential in guiding therapy. For the purpose of calculating the TBSA, only burns that are more severe than superficial burns are included. There are many ways to estimate TBSA. The “rule of nines” is a well-known method for approximating body surface area in adults. In this method, the head is counted as 9% of the body surface area (BSA), each upper extremity as 9%, the anterior and posterior trunk each comprise 18%, and each lower extremity is counted as 18%. The perineum get the remaining 1%.<sup>5,8,9</sup> In children, the rule of nines is less applicable. Children generally have a relatively larger head and relatively smaller lower extremities compared to adults. The Lund-Browder or Berkow chart is an excellent tool used in children to assess TBSA by correlating the percentage of TBSA of different parts of the body as a function of developmental age.<sup>6,8</sup> A third method used to approximate percentage of TBSA is the “rule of the hands.” This method uses the patient’s hand to assess the TBSA of small or irregular burns, and can be used in both children and adults. Traditionally, it was thought that the

size of the patient's handprint (palms and fingers) represented 1% of TBSA. However, studies have shown that this approach slightly overestimates the BSA; 0.8% is probably a more accurate estimation.<sup>10</sup>

## Initial Assessment and Treatment

Burn management involves the standard advanced trauma life support protocol of assessing airway, breathing, and circulation. However, the ABCs of burn management also require early recognition of even the most subtle clinical findings in all patients. Findings on the primary survey that can appear inconsequential can rapidly progress to life-threatening conditions. Thus, it is important to identify and address all examination findings that could affect airway, breathing, and circulation as soon as they are recognized. For example, burned facial hair, soot around the mouth, and facial flash burns may indicate an impending compromised airway and should prompt early intubation prior to the onset of clinically evident respiratory distress. Tetanus immunization should be updated in all burn patients, including those with minor burns.<sup>8</sup>

### CRITICAL DECISION

**Is it appropriate to clinically observe a stable patient with evidence of smoke inhalation?**

Inhalation injury is a common cause of death in burn victims. It is essential to have high degree of suspicion of inhalation injury and establish early intubation to secure the airway if it seems necessary.<sup>6</sup> Intubation is also recommended in any patient with burns covering 60% or more of TBSA, including the face, and in those being transferred when there is a going to be a long transport time.<sup>9,11</sup> Acute onset of airway obstruction may not be preceded by any changes in physical examination findings.<sup>11</sup> Common clinical clues suggestive of inhalation injuries are neck and face burns, cough, stridor,

wheezing, hoarseness, oropharyngeal blistering, carbonaceous sputum, tongue swelling, respiratory distress, and singed eyebrows and nasal hairs.<sup>6,11</sup> Patients with these signs and symptoms are candidates for early intubation rather than clinical observation.<sup>7</sup> Progressive edema of the airway can complicate intubation performed later in the clinical course.<sup>6</sup> Endotracheal intubation may be performed by the rapid sequence intubation (RSI) protocol. The risk of hyperkalemia does not develop until 48 to 72 hours following a severe burn. Unless otherwise contraindicated, succinylcholine may be used in the acute setting for RSI in patients following burn injury.<sup>12,13</sup>

Patients with inhalation injury have increased airway resistance caused by edema, bronchospasm, or debris within the airway. A common approach to mechanical ventilation in these cases is to minimize the mean airway pressure used in ventilation.<sup>7</sup> This can be achieved by using low tidal volumes (7 to 8 mL/kg) and higher respiratory rates (16 to 20 breaths per minute), thereby achieving permissive hypercapnia.<sup>7,14</sup> Hypercapnic acidosis may protect against endotoxin-induced lung injury and has protective effects on the myocardium and brain.<sup>15</sup>

Any patient who incurs a burn injury in an enclosed space or presents obtunded is presumed to have carbon monoxide intoxication until it is proved otherwise.<sup>6,9</sup> These patients should be placed on 100% oxygen, which decreases the half life of carbon-monoxide-bound hemoglobin from 4 hours on room air to 45 minutes.<sup>7</sup> A carboxyhemoglobin (HbCO) level should be obtained, and 100% oxygen should be continued until the HbCO level is less than 10%.<sup>6,7</sup> Carbon monoxide intoxication may be treated with hyperbaric oxygen when elevated HbCO levels are associated with a history of unconsciousness, presence of neurologic abnormalities, cardiac instability, or cardiac ischemia. Hyperbaric oxygen may also be

started as an adjunctive therapy to carbon monoxide toxicity, possibly helping to reduce the severity of neurologic sequelae or helping to sustain oxygenation in cases of severely elevated HbCO.<sup>7</sup>

Cyanide poisoning should be considered in patients with severe unexplained metabolic acidosis associated with a normal oxygen content or a low HbCO level or who have failed to clinically respond to oxygen.<sup>6,7</sup> Hydroxocobalamin is one of the treatments for cyanide poisoning.<sup>9</sup>

### CRITICAL DECISION

**What methods are useful for achieving optimal fluid resuscitation in burn patients?**

Achieving optimal fluid resuscitation is critical in managing burn patients. Burns greater than 20% of TBSA can cause large fluid shifts and depletion of intravascular volumes secondary to increased capillary permeability. This phenomenon is known as burn shock. It is imperative to achieve optimal fluid resuscitation to maintain end-organ perfusion. Decreased perfusion, acute renal failure, and death can be complications of under-resuscitation. On the other hand, overly aggressive fluid resuscitation can contribute to acute respiratory distress syndrome, worsening edema, multiple organ dysfunction, and abdominal, extremity, and orbital compartment syndromes.<sup>7,16</sup>

The standard of care is to institute fluid resuscitation in burn patients who have involvement of 20% of TBSA or more via intravenous cannulas. There are numerous formulas for calculating the fluid requirements in the first 24 hours, including the Parkland, modified Brooke, Shriners-Cincinnati, and the Galveston formulas. In the most common of these, the Parkland formula, crystalloid is given in a dose of 4 mL/kg of body weight per percentage of BSA burned. Half of the fluid is given over the first 8 hours, and the rest over the next 16 hours.

For further details regarding the most common formulas, see Table 1. The formulas are only a starting point for fluid resuscitation, and regardless of which formula is chosen, fluid administration should be adjusted based on urine output and other clinical parameters.<sup>16</sup>

Currently, a crystalloid solution (eg, Ringer lactate) is the most popular and safest fluid used to resuscitate burn patients despite the availability of other volume expanders. Because over-hydration is common and causes potentially devastating complications, other solutions have been employed. Hypertonic saline reduces the required volume of administered fluid; however, the use of hypertonic fluids is associated with a two-fold increase in mortality and a four-fold increase in acute renal failure. Therefore, hypertonic fluids are not recommended for burn resuscitation.<sup>7,16-18</sup>

There is great debate over the use of colloids as a resuscitation tool for burn victims. Examples of colloids used in burn resuscitation are plasma, albumin, and high-molecular-weight glucose polymers (dextran and hydroxyethyl starch).<sup>16</sup> Colloids can be used to mitigate the effects of decreased plasma proteins caused by large volumes of crystalloids, which could decrease the total amount of volume required to maintain adequate urine output.<sup>16</sup> Some burn units use colloids when, despite adequate

crystalloid resuscitation, burn shock worsens.<sup>9,19</sup> The use of plasma carries the risk of transmitting blood-borne infections and has also been associated with acute lung injury.<sup>16</sup> Plasma exchange shows promising outcomes. In burns, the increased capillary leak is partly caused by inflammatory mediators. Klein et al showed that following plasma exchange there was a decrease in fluid requirements, an increase in urine output, and improvement of base deficit and lactate and hematocrit levels.<sup>20</sup>

Blood transfusions have been shown to increase infectious episodes and mortality in burn patients.<sup>21,22</sup> Palmieri et al demonstrated a 13% increased risk of infection per unit transfused of packed red blood cells.<sup>21</sup> In the presence of ongoing blood loss or an acute drop in hemoglobin, blood transfusion may be indicated. However, some authors recommend not using the traditional 10 g/dL hemoglobin level threshold for transfusion except in patients with concomitant acute myocardial infarction, unstable angina, or a history of cardiac disease. Otherwise, using a more strict protocol for transfusion and a hemoglobin level of 7 to 8 g/dL as the threshold is advantageous in avoiding increased risk.<sup>14,21</sup>

Another important consideration in the administration of fluids is the need for maintenance fluids.

It is important to either manually calculate maintenance needs or to use one of the formulas in Table 1 that includes maintenance fluids. Although all patients need maintenance fluids, many authors think that it is especially important to ensure adequate maintenance fluid in children. Pediatric patients have a higher body-surface-area-to-weight ratio than do adults, predisposing them to higher insensible losses and relatively larger maintenance fluid needs than adults. The Parkland formula only calculates the volume required for adequate fluid resuscitation; the Galveston formula is sometimes preferred in children because it takes into account maintenance needs.<sup>7,11,16</sup>

Glycogen stores are depleted after 12 to 14 hours of fasting in children,<sup>16</sup> which makes this population susceptible to hypoglycemia. Dextrose should be added to resuscitation fluid, or early enteral nutrition should be employed.<sup>7,11,16</sup> Blood glucose levels should be frequently monitored to prevent hyperglycemia or hypoglycemia.<sup>11</sup>

**CRITICAL DECISION**

**How should adequate fluid resuscitation be assessed?**

The most commonly used physiological parameter to assess adequate fluid resuscitation is hourly urine output. Urinary catheters should be placed in all

**Table 1.**

Commonly used fluid resuscitation formulas

|  |   |
|--|---|
| Parkland formula                             | 4 mL/kg/percentage TBSA<br>½ of the total volume given over the first 8 hours<br>½ of the total volume given over the next 16 hours   |
| Modified Baxter formula                      | 2 mL/kg/percentage TBSA<br>½ of the total volume given over the first 8 hours<br>½ of the total volume given over the next 16 hours   |
| Shriners Burn Institute (Cincinnati) formula | 4 mL/kg/percentage TBSA + 1,500 mL/m <sup>2</sup> BSA<br>½ given as lactated Ringer with 50 mEq sodium bicarbonate given over the first 8 hours<br>¼ given as lactated Ringer given over the next 8 hours<br>¼ given as 5% albumin in lactated Ringer over the next 8 hours |
| Galveston Shriners Hospital formula          | 5,000 mL/m <sup>2</sup> burn surface area + 2,000 mL/m <sup>2</sup> BSA<br>½ of the total volume given over the first 8 hours<br>½ of the total volume given over the next 16 hours   |

patients requiring intravenous fluid resuscitation. The urine output goal is 1 mL/kg/hour in children younger than 2 years (or who weigh less than 30 kg) and 0.5 mL/kg/hour in adults and older children.<sup>7,8,11,19</sup> The intravenous fluid rate should be adjusted every 1 to 2 hours using urine output as an end point.<sup>8</sup> Other clinical parameters used to assess volume status include the patient's mental status, capillary refill rate, heart rate, mean arterial pressure, and distal pulses.<sup>6-8</sup> Laboratory values such as decreased mixed venous oxygen saturation, increased serum lactate, and significant base deficits suggest inadequate end-organ perfusion.<sup>23,24</sup> In studies comparing fluid resuscitation based on invasive hemodynamic monitoring versus administration based on a fluid resuscitation formula, invasive monitoring in burn shock results in a significant increase in fluid administration without improvement in outcome.<sup>25</sup>

### CRITICAL DECISION

#### What options exist for pain management in burn patients?

The degree of pain is inversely proportional to the depth of the burn injury. Full-thickness burns are painless because they destroy the pain nerve endings in the dermis. However, partial-thickness burns are extremely painful.<sup>7</sup> Acetaminophen and nonsteroidal anti-inflammatory drugs are appropriate for mild pain.<sup>26-28</sup>

Morphine, an opioid analgesic, is the most commonly used agent for moderate to severe pain. It is the mainstay of burn pain management because it is easily titrated to control pain, has a low protein binding, is conjugated in the liver, and is removed by glomerular filtration.<sup>7,29</sup> Anxiety can significantly add to a burn victim's perception of pain. A low dose of an anxiolytic such as a benzodiazepine is sometimes given along with an analgesic to improve pain control.<sup>9,29</sup> Wound manipulation such as dressing changes and

débridement can cause overwhelming pain, which could necessitate the use of procedural sedation.<sup>29</sup> Note that local or regional anesthesia may also be used.

Evidence has shown that cooling of burns reduces pain, the extent of injury, the depth of injury, the need for surgical excision of the burn, scarring, and mortality.<sup>26</sup> Tap-water or saline-soaked gauze cooled to between 12°C and 25°C (54°F and 77°F) can be applied to the burned tissues. Ice and ice water can cause local tissue injury and are not recommended as a cooling method.<sup>26-28,30</sup>

In patients undergoing external cooling who have burns covering 10% or more of TBSA, invasive core body temperature monitoring should be used to watch for hypothermia. Heat will be lost by local cooling of the wound, and skin disruption at the burn site can also cause heat loss via radiation, convection, conduction, and evaporation. Warm intravenous fluids and a warm environment can be used to maintain body temperature.<sup>11</sup>

### Burn Dressings

The purpose of proper local wound management is to reduce pain and contamination, prevent infection, and promote rapid healing with minimal scarring.<sup>26</sup> Initial management of burns consists of gentle cleansing of the wound with sterile saline, mild soap and tap water, or commercial products containing poloxamer 288.<sup>7,27</sup> Disinfectants such as chlorhexidine gluconate solution and povidone-iodine can inhibit the healing process, and their use is discouraged.<sup>27</sup> After the wound is cleaned, necrotic tissues and blisters are appropriately managed. Topical antimicrobial agents or bio-engineered substitutes should be applied to clean, débrided wounds.<sup>6</sup> However, if transfer to a burn center is imminent, no agent should be applied and the wound should simply be wrapped in a clean, dry sheet. If transfer is delayed, an antibacterial

ointment based on the burn center's preference should be applied.<sup>7</sup>

Moist dressings are superior to dry dressings. The moisture fosters re-epithelialization and angiogenesis of the wound.<sup>7,8,26</sup> Topical antimicrobial agents limit bacterial and fungal colonization in wounds.<sup>7</sup> Superficial burns do not require prophylaxis with topical antimicrobial medication,<sup>6,27</sup> but deeper burns should receive topical prophylaxis.<sup>27</sup> Commonly used topical agents are silver sulfadiazine, bacitracin, neomycin, mafenide acetate, and 0.5% silver nitrate solution.<sup>7,8,27</sup>

Silver sulfadiazine is effective against yeast, has bactericidal activity against gram-negative and gram-positive bacteria, and is more commonly used than bacitracin because it has a broader antimicrobial coverage.<sup>6</sup> Silver sulfadiazine contains sulfa and should not be used in patients with a known sensitivity to sulfa.<sup>6</sup> This medication causes permanent silver staining of the skin and is therefore contraindicated for facial applications. Severe hemolysis ensues if it is applied to patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency.<sup>5,6</sup> This agent should not be used in pregnant woman, newborns, or nursing mothers with children younger than 2 months because of the risk of sulfonamide kernicterus.<sup>5</sup>

Bacitracin is useful because it is cheaper than silver sulfadiazine, can be used around mucous membranes, and is less toxic to re-epithelialization of the burn wound.<sup>5-7</sup> Mafenide acetate is useful because it penetrates devascularized tissue and remains active in an acidic environment; however, the application can cause severe pain and metabolic acidosis, which may necessitate discontinuation.<sup>6</sup>

Unfortunately, traditional antimicrobial ointments require frequent dressing changes for optimal efficacy. For example, bacitracin ointment should be reapplied every 6 hours.<sup>6,7</sup> Dressing changes are extremely painful. Newer agents that

are silver-based have been developed to address this problem.<sup>6</sup> These silver-impregnated antimicrobial agents can be left on for several days and slowly release small amounts of silver.<sup>6,26</sup> They reduce the complexity and number of dressing changes, hospital length of stay, and pain and improve wound healing when compared with traditional topical antimicrobial agents.<sup>6,26,31</sup>

Biological dressings are effective with deep wounds. They gradually peel off as skin epithelializes underneath them.<sup>27</sup> These agents are associated with a lower infection rate and faster healing rate.<sup>8,27,32</sup> Demling et al demonstrated a significant decrease in wound care time, pain, and re-epithelialization time when these dressings were compared to topical antibiotics.<sup>32</sup>

**Table 2.**

Burn center referral criteria<sup>1,6-8,26</sup>

Partial-thickness burns covering more than 10% TBSA

Burns that involve the face, hands, feet, genitalia, perineum, or major joints

Full-thickness burns in any age group

Significant chemical injury

Significant electrical injury (including lightning injury)

Inhalation injury

Patients with significant concomitant mechanical trauma per physicians' judgment

Patients with preexisting medical disorders that may complicate management, prolong recovery, or affect survival

Pediatric patients initially presenting to hospitals without qualified personnel or equipment for the care of children

Patients who require special social, emotional, or long-term rehabilitative intervention

## CRITICAL DECISION

### Should emergency physicians unroof blisters?

The approach to intact burn blisters is a controversial topic. Advocates for the preservation of intact blisters think this method decreases healing time and wound infection and complications rates.<sup>7,33-35</sup> A small study from the 1950s showed blisters left intact healed faster than débrided or ruptured blisters.<sup>35</sup> It has been postulated that this phenomenon is due to mitogenic substances found in blister fluid.<sup>33</sup> In theory, a blister facilitates the migration of epidermal cells to the wound healing by providing a moist environment.<sup>33</sup> Swain et al showed microbe colonization was high in exposed wounds and lowest in intact blisters.<sup>36</sup> Intact blisters may offer pain relief by minimizing direct stimulation of superficial nerves.<sup>33</sup>

Advocates for the débridement of blisters think this method also decreases healing time and wound infection and complication rates. It is postulated that the pressure from the fluid in blisters may compromise circulation and cause progression of a partial thickness burn to a deeper injury.<sup>37</sup> Blister fluid has decreased leucocyte phagocytic ability and lymphocyte repression.<sup>33,38</sup> This, in conjunction with blister fluid's overall lack of antibacterial activity, creates a nidus for infection.<sup>39</sup> Garner et al concluded that re-epithelization may be inhibited beneath burn blisters, resulting in increased healing time.<sup>40</sup>

In general, the decision to unroof blisters in burns should be based on the appearance and location of the blister. Small blisters that are intact and that appear to have a strong covering may be left intact and allowed to re-epithelialize underneath. Larger blisters that have broken open or appear in danger of opening should be opened, débrided, and dressed. Blisters should also be débrided if they contain cloudy fluid or if they cause a functional impairment (ie, over joints).

## CRITICAL DECISION

### When should an emergency physician transfer a patient to a burn center?

Certain burn types or patterns of injury are more likely to cause significant morbidity or mortality. The American College of Surgeons (ACS) has developed criteria to identify those patients with clinically significant burns who may benefit from observation, evaluation, and treatment in a certified burn center. Referral to a burn center depends on the size, classification, and location of the burn. Age, comorbidities, concomitant injuries, and social factors also help to identify the patients who would benefit from specialized care.

"And then what happened?" See how the case resolved.



## Case Resolutions

### ■ Case One

The man brought in from a house fire was immediately given oxygen by a 100% nonbreathing mask, cardiac monitoring was initiated, and a peripheral intravenous line was placed. The patient was emergently intubated and placed on mechanical ventilation with a tidal volume of 550 mL and a rate of 20. Blood was drawn for analysis, including arterial blood gas measurement with Hbco levels. Burns were estimated to cover approximately 45% of his TBSA. Immediate fluid resuscitation was begun. Based on calculations using the Parkland formula, 8.1 liters of lactated Ringer should be given over the first 8 hours, and an additional 8.1 liters would be required over the next 16 hours. A core temperature monitor was placed, and hypothermia was combated using warmed intravenous fluids when appropriate. Immediate transfer to a burn center was initiated. Because transport time was estimated to be more than 30 minutes, all burns were cleansed with poloxamer 288, wounds were débrided of devitalized tissue and then dressed with topical antimicrobials and moist dressings. Prior to transport, the patient's tetanus immunization was also updated.

## ■ Case Two

In the case of the toddler with scald injury, a primary survey was completed, intravenous access was obtained, and cardiac monitoring was immediately begun. The TBSA of burns was estimated to be approximately 30%. Using the Galveston formula, it was determined that 1,855 mL total would be needed during the next 24 hours. Lactated Ringer solution was initiated at a rate of 116 mL/hr, to deliver half the requirement in 8 hours. The second half would be given over the next 16 hours. Urine output was monitored to ensure a rate of at least 1 mL/kg/hr. Analgesia was immediately started with intravenous morphine and was titrated to effect. Temperature was monitored with a core temperature probe, and hypothermia was combated with warm intravenous fluids when appropriate. All of the burns were cleansed with poloxamer 288, and the blisters were left intact. Transfer to a burn center 15 minutes

away was arranged. The burns were then wrapped in a dry, sterile sheet, and the patient transferred to the burn center for further management. The parents confirmed that the child's vaccinations were up-to-date, so no tetanus prophylaxis was needed.

## ■ Case Three

The young man involved in the fireworks accident required only conservative management. A nonsteroidal anti-inflammatory medication was given for pain management. All burned areas were washed with poloxamer 288. Blisters were left intact. After ensuring that the patient had no history of sulfa allergy, silver sulfadiazine was placed on all partial-thickness burns and they were covered with moist dressings. The patient was given tetanus prophylaxis before discharge home. He was instructed to follow up with his primary care physician within 48 to 72 hours. He was given a prescription for appropriate analgesic medications and silver sulfadiazine and instructed to change the dressings twice daily and to avoid sun exposure to the burned areas.

## Conclusion

Injuries from thermal burns are commonly encountered in emergency departments. The severity of burns can range from mild and self-limited to severe, life-threatening, and quickly fatal. Some complications may arise only after a delay and require appropriate preventive interventions. A focused examination is required to screen for the most devastating injury patterns. Any signs of potential inhalational injury should prompt consideration of intubation for immediate airway control. Patients who may have sustained smoke inhalation area at risk of carbon monoxide exposure and require targeted testing and treatment. Adequate management depends on correctly classifying burn size and severity. Patients with clinically significant burns may experience shifts in fluid balance, difficulty regulating body temperature, and

loss of natural barriers to infections; these complications must be promptly addressed with tailored fluid replacement, temperature monitoring with prevention of hypothermia, and wound management. Additionally, burns that leave the dermis partially or completely intact can cause moderate or severe pain. Clinicians should be prepared to manage pain with opioids and to control anxiety with appropriate anxiolytics. Short procedures such as wound débridement warrant consideration of procedural sedation. Finally, because of the potential for a prolonged course, patients may require delayed completion of the workup for surgical intervention. Meeting criteria for management shifts to specialized care and stabilization.

**"Says who?" We show you where the research and recommendations came from.**



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## Pearls

- Patients with burns involving 20% or more of TBSA will require intravenous fluid resuscitation.
- In fluid resuscitation, the urine output goal is 1 mL/kg/hour in children younger than 2 years (or weighing less than 30 kg) and 0.5 mL/kg/hour in adults and older children.
- If signs of inhalation injury are present, early intubation should not be delayed, even if the patient initially appears clinically stable.

## Pitfalls

- Applying wound dressings if transfer to a burn center is imminent; instead apply a simple clean, dry dressing.
- Unroofing blisters on the palms and soles.

Emergency medicine's favorite ECG teacher, Amal Mattu, gives you a consult in every issue.



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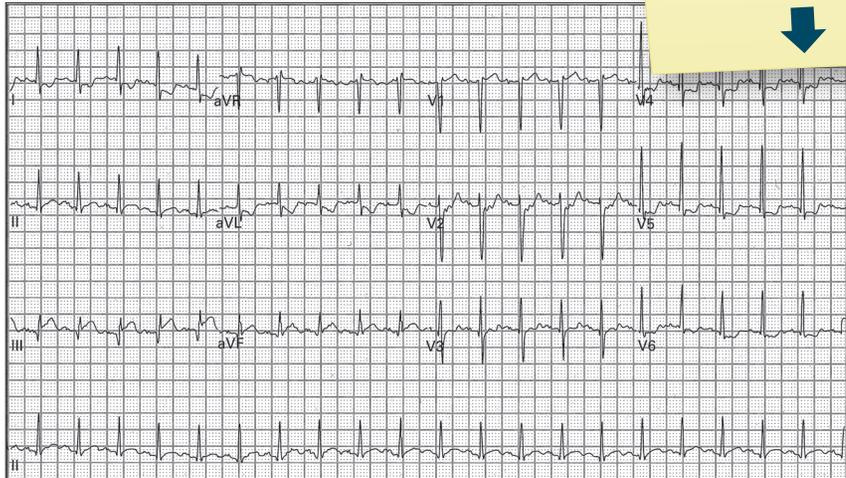


More patient safety tips.



## The Critical ECG

A 75-year-old man with dyspnea and nausea.



**Sinus tachycardia, rate 124, acute inferior and right ventricular (RV) myocardial infarction (MI).** Q waves and ST-segment elevation in the inferior leads indicate acute MI. RV extension of the MI is diagnosed based on the presence of simultaneous ST-segment elevation in lead V<sub>1</sub> and ST-segment depression in lead V<sub>2</sub>. RV MI was confirmed using right-sided chest leads and echocardiography. Reciprocal ST-segment depression is noted in the lateral leads.

Feature Editor: Amal Mattu, MD, FACEP

## The Drug Box

Asif Rahman, MD; Summa Akron City Hospital, Department of Emergency Medicine

### Magnesium Sulfate

|   |  |
|---|--|
| <b>Mechanism of Action</b>                    | NMDA-receptor blockade, inhibition of presynaptic glutamate release, and increase in action potential threshold; indirect inhibition of calcium channels leading to smooth muscle relaxation   |
| <b>Indications</b>                            | For peripartum patients: blood pressure higher than 140/90 plus one of the following: abdominal pain, visual disturbances, or severe headache<br>In all populations: severe asthma exacerbations, torsade de pointes, refractory ventricular tachycardia or ventricular fibrillation, other cardiac dysrhythmias |
| <b>Dosing</b>                                 | 4-6 grams IV over 15 minutes followed by 1-2 grams IV every hour<br>Onset of action is immediate, and duration is approximately 30 minutes.  |
| <b>Side Effects</b>                           | Common side effects include flushing, nausea, and vomiting.  |
| <b>Estimated Cost to Hospital and Patient</b> | Hospital cost is roughly \$4 per 4 mg IV. Patient cost is estimated at \$10-\$20 per 4 mg IV.  |
| <b>Contraindication/Precautions</b>           | Complete heart block is an absolute contraindication.<br>Magnesium toxicity: CNS depression, respiratory depression, hyporeflexia, hypotension<br>Monitor for magnesium toxicity with frequent, serial exams. IV calcium gluconate or calcium chloride is the treatment for magnesium toxicity.                  |

Feature Editors: Michael S. Beeson, MD, MBA, FACEP; Amy K. Niertit, MD

## The Critical Image

A 37-year-old woman presenting with sudden onset of intense right lower quadrant pain beginning 90 minutes prior to arrival. Her last menstrual period was 3 weeks ago. She has no fever, chills, diarrhea, nausea, vomiting, hematuria, dysuria, vaginal bleeding, or discharge. There is no history of renal stones or abdominal surgery. Blood pressure is 135/94, heart rate 54, respiratory rate 16, and temperature 36.7°C (98.1°F). Her examination demonstrates right lower quadrant tenderness and guarding. Her urine HCG is negative. The emergency physician suspects ovarian torsion and obtains immediate pelvic ultrasound.

The patient's initial ultrasound (A) reveals an enlarged right adnexa, which could represent an engorged torsed ovary or ovarian pathology in the absence of torsion. Ovarian enlargement increases the risk of torsion but is not sufficient to prove it. Conversely, a structurally normal ovary is less likely to be torsed but does not rule out the diagnosis.

An absence of both arterial and venous blood flow by Doppler ultrasound is consistent with ovarian torsion, but studies show that arterial or venous blood flow is present in 30% to 50% of patients with ovarian torsion.<sup>1-3</sup> A possible explanation is intermittent ovarian torsion, in which blood flow may be preserved at the time of the ultrasound study. A second possibility is that the low-pressure ovarian venous system may be the first to be compromised by partial or early torsion, preserving some higher-pressure arterial flow. Consequently, when ovarian torsion is strongly suspected, blood flow demonstrated on ultrasound is insufficient to rule out the diagnosis, and early gynecology consultation is prudent. The patient's Doppler ultrasound (B) demonstrates an apparently normal arterial waveform from the right adnexa.

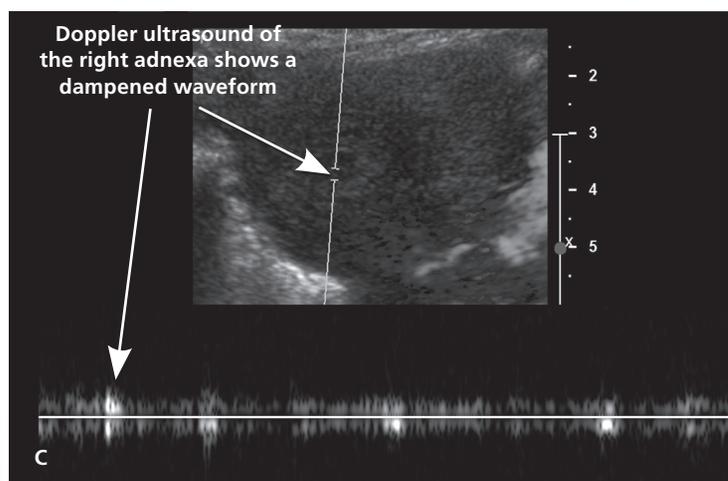
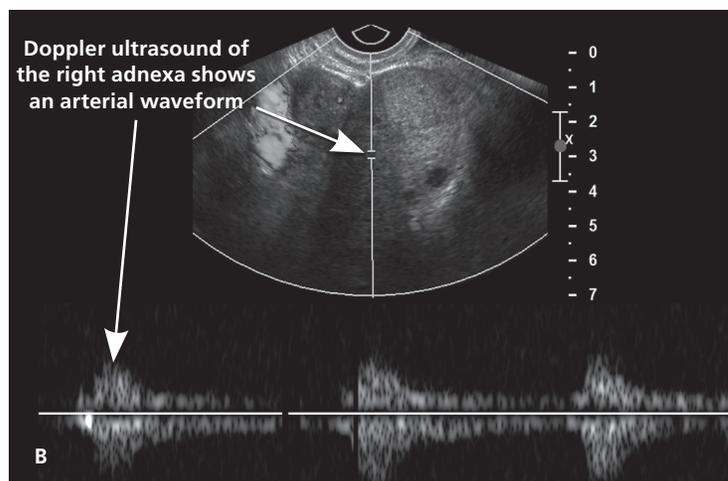
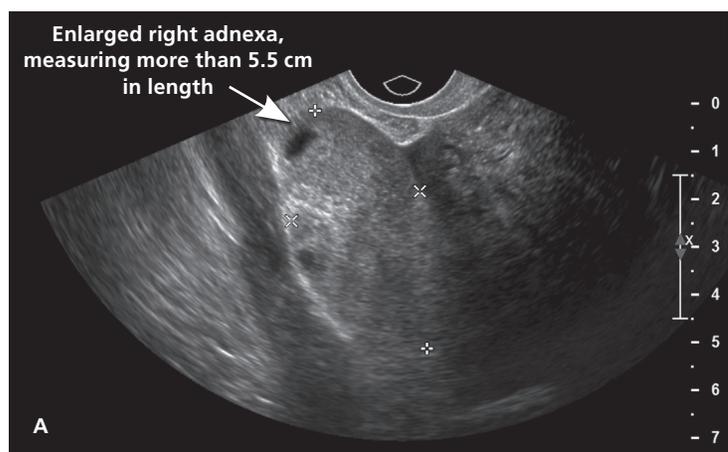
A CT performed to further evaluate the etiology of the patient's ovarian mass and continued pain confirmed an adnexal mass; a followup ultrasound performed 3 hours later (C) demonstrated dampened blood flow.

Laparoscopy was performed, demonstrating a bluish ovarian mass torsed four times. This was removed, and pathology demonstrated only ovarian necrosis without other underlying pathology.

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**Feature Editor:** Joshua S. Broder, MD, FACEP.  
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- A 40-year-old man presents because for the past 3 days he has been weak and nauseated. On initial evaluation, he has stable vital signs but is found to have a serum potassium level of 6.8 mEq/L. Which of the following studies dictates the next step in management?

  - 12-lead ECG
  - arterial blood gas
  - blood glucose
  - chest radiograph
  - serum creatinine
- A young woman with glomerulonephritis and acute renal failure complains of weakness and palpitations. Her 12-lead ECG shows peaked T waves, absent P waves, and a QRS duration of 240 milliseconds. Which of the following is the next step in emergency department management?

  - calcium gluconate
  - furosemide
  - hemodialysis
  - insulin with glucose
  - nebulized albuterol
- A 40-year-old pregnant woman thought to have preeclampsia is transferred to the emergency department on a magnesium sulfate drip. She is noted to be lethargic, with respiratory depression. On examination, she has diffuse crackles in her lungs and absent reflexes. After providing oxygen and attaching a monitor, what the best treatment?

  - activated charcoal
  - calcium gluconate
  - glucagon
  - naloxone
  - sodium bicarbonate
- For which of the following patients would it be most appropriate to supplement potassium to keep serum levels above 4 mEq/L?

  - a 23-year-old woman with palpitations and no past medical history
  - a 42-year-old woman with poorly controlled diabetes mellitus
  - a 51-year-old man with hypertension, recently started on lisinopril
  - a 55-year-old man with chest pain and premature ventricular complexes
  - a 68-year-old man with chronic bronchitis and fever
- Paramedics bring in a 42-year-old alcoholic man found wandering near a bus stop. He appears disheveled and malnourished. Approximately 4 hours after receiving intravenous glucose and thiamine, he complains of severe muscle aches and diffuse weakness. Supplementation with which of the following might have prevented his new symptoms?

  - calcium gluconate
  - diazepam
  - normal saline
  - potassium phosphate
  - sodium bicarbonate
- A woman with diabetes is receiving normal saline and insulin for suspected diabetic ketoacidosis. Two hours later, she complains of severe, diffuse muscle weakness. Her cardiac monitor shows broad T waves, U waves, and frequent premature ventricular complexes. Supplementation with which of the following might have prevented this presentation?

  - calcium gluconate
  - dextrose
  - potassium chloride
  - sodium bicarbonate
  - sodium phosphate
- A 58-year-old man with history of small-cell lung cancer is brought in for weakness and pleuritic chest pain. He is alert, with normal vital signs, and has a nonfocal neurologic examination and a serum sodium of 129 mEq/L. What is the appropriate management of his hyponatremia?

  - free water restriction
  - hypertonic saline bolus
  - intravenous furosemide
  - normal saline bolus
  - sodium chloride tablets
- A 45-year-old woman presents with a 1-day history of cramping in the legs and forearms. She is one week postoperative from a thyroidectomy for follicular carcinoma. When her nurse inflates the blood pressure cuff, her ipsilateral hand contorts into a rigid fist. What is the treatment of choice?

  - calcium gluconate
  - diphenhydramine
  - levothyroxine
  - lorazepam
  - magnesium sulfate
- A marathon runner is brought in after he collapsed at the 15th mile marker. He had been hydrating at water fountains placed every mile along the route. Suddenly, he begins to seize and does not stop despite 10 mg of diazepam. What is the most appropriate treatment?

  - 5% dextrose solution
  - fosphenytoin
  - hypertonic saline
  - lorazepam
  - pentobarbital
- A 74-year-old woman with diabetes is brought from her nursing home for altered mental status. She is lethargic, with thready peripheral pulses, cool extremities, dry mucous membranes, and clear breath sounds. Her serum sodium level is 162 mEq/L with a blood glucose of 830 mg/dL. What is the best treatment?

  - half-normal saline, 250 mL/hr
  - half-normal saline bolus, 1 liter
  - intravenous insulin, 10 units
  - normal saline, 250 mL/hr
  - normal saline bolus, 1 liter

11. Regarding the epidemiology and risk factors surrounding burns, which of the following is correct?
- burn injuries occur mostly in African American women
  - compared with women, men have a much lower risk of death from burns
  - most burns occur in the home and incidents usually involve ethanol intoxication
  - survival for patients admitted to burn centers has been declining in the last 5 years
  - the two most common etiologies for burns are exposure to tar and sun exposure
12. A 34-year-old firefighter presents with burns covering his left arm, left leg, the upper half of his anterior trunk, and his face, head, and neck. Using the "rule of nines" what total body surface area (TBSA) is involved in this injury?
- 9%
  - 27%
  - 45%
  - 63%
  - 75%
13. Early intubation for patients with evidence of inhalational injury is recommended because:
- airway obstruction can occur rapidly and without clinical signs on initial physical examination
  - hyperkalemia always develops later in the clinical course, complicating intubation
  - inhalational injury is always associated with carbon monoxide poisoning, requiring intubation for resolution of the exposure
  - intubation always improves outcomes in patients with burns covering more than 30% TBSA
  - mechanical ventilation directed toward keeping the patient in state of respiratory alkalosis has shown to have a protective effect against lung injury
14. A 100-kg, 47-year-old woman presents with multiple partial-thickness burns. The TBSA of injury is approximately 40%. Initial management of the patient includes:
- immediate referral for tissue débridement and skin grafting
  - rapid administration of lactated Ringer solution or normal saline to combat burn shock
  - tetanus immunization
  - type and cross-match for immediate administration of 2 units of packed red blood cells
  - washing all wounds with chlorhexidine gluconate and povidone-iodine followed by application of dry dressings
15. Which of the following is true regarding the use of hypertonic saline to treat or prevent burn shock?
- acute tubular necrosis, hypernatremia, and hyperchloremic acidosis are all possible adverse effects of hypertonic saline administration
  - although not without potential side effect, hypertonic saline eliminates the possibility of overhydration, especially in pediatric patients
  - hypertonic saline increases intravascular volume while decreasing urinary output
  - hypertonic saline increases the risk of edema formation in the extremities
  - unlike lactated Ringer solution, hypertonic saline has no association with acute renal failure
16. A 25-year-old man with minor superficial burns is brought in after being found trapped inside a burning building. The man appears lethargic and is incoherent; his neurologic examination is nonfocal. Which of the following is an important first step in his management?
- administration of 100% oxygen and a stat HgCO level
  - a head CT to rule out underlying trauma
  - ice-water immersion of injured areas
  - immediate covering with warm blankets to minimize heat loss
  - placement of a central venous catheter
17. Which of the following patients requires referral to a burn center?
- a 4-year-old girl with burn injuries totaling 9% of TBSA
  - a 27-year-old man with superficial partial-thickness scald burns of both of his forearms from boiling water
  - a 33-year-old woman with deep partial-thickness burns on the knees and perineum
  - a 45-year-old man with superficial burns to his wrists from contact with a hot stove
  - a 57-year-old otherwise healthy woman with partial-thickness burns on approximately 5% of her trunk
18. A woman presents with a burn on her calf from the exhaust pipe of a motorcycle. The burn has a blister that breaks with minimal pressure, appears patchy white and pale red underneath, has a waxy appearance, has visible hair follicles at the burn site, and is exquisitely painful to pressure. What class of burn does she have?
- deep partial-thickness burn
  - fifth-degree burn
  - full-thickness burn
  - superficial burn
  - superficial partial-thickness burn
19. Proper care of burn wounds includes:
- application of silver sulfadiazine after cleansing except to burns on the face or in patients with sulfa hypersensitivity
  - dry dressings over wounds if patients can be transferred to a burn center within 24 hours
  - oral antibiotic prophylaxis to all patients with burns more severe than superficial
  - tetanus prophylaxis in all patients with deep partial-thickness or more severe burns
  - thorough cleansing of all wounds with povidone-iodine
20. A 100-kg man with significant burns requires intubation. Which of the following is the correct mechanical ventilation setting for this patient?
- high tidal volumes (800 mL), high respiratory rate to maintain respiratory acidosis
  - high tidal volumes (800 mL), low respiratory rate to maintain respiratory alkalosis
  - low tidal volumes (600 mL), high respiratory rates to maintain hypercapnic acidosis
  - low tidal volumes (600 mL), low respiratory rates to maintain hypercapnic acidosis
  - pressure support ventilation with high positive airway pressure and high respiratory rate to maintain respiratory alkalosis

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