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## INSIDE



# Looking for a Clinical Decision Rule to Allow Safe Discharge of Young Chest Pain Patients

ABSTRACT & COMMENTARY

**Source:** Marsan RJ, et al. Evaluation of a clinical decision rule for young adult patients with chest pain. *Acad Emerg Med* 2005;12:26–32.

THE AUTHORS DEVELOPED A CLINICAL DECISION RULE FOR adult (younger than 40 years) chest pain patients, minimizing the risk of 30-day adverse cardiovascular (CV) events. Slightly more than 1000 patients ages 24–39 years who received electrocardiograms (ECG) for chest pain during a 33-month period met criteria for enrollment; cocaine users were excluded. The main outcome was 30-day adverse CV events (e.g., death, acute myocardial infarction, percutaneous intervention, and coronary artery bypass graft); 30-day follow-up was done by telephone. The majority of patients were female (61%) and African American (73%). Overall, the risks of acute coronary syndrome (ACS) and 30-day adverse CV events were 5.4% and 2.2%, respectively. However, for the patients with no cardiac history and no cardiac risk factors, the risk of ACS and 30-day adverse CV event was 1.8%. Furthermore, in patients with no cardiac history and a normal ECG, the risk was reduced to 1.3%. Finally, patients with no cardiac history, no cardiac risk factors, and a normal ECG had a risk of 1.0%. A modified clinical decision rule using the above factors plus serum markers for cardiac ischemia, found that in young adult patients: 1) without a known cardiac history, 2) with either no classic cardiac risk factors or a normal ECG, and 3) initially normal cardiac marker studies, the risk of ACS also was extremely low (0.14%); there were no adverse CV events at 30-day follow-up (95% CI, 0.1% to 0.2%). ❖

### COMMENTARY BY RICHARD HAMILTON, MD, FAAEM, ABMT

There is a great deal of wisdom in the approach the authors take to our daily confrontation with low-risk chest pain patients— especially in the young. Simply put, their research takes the position that it may be easier to determine which patients seemingly never have

an ACS, rather than to ascertain which patients are more likely to have it. As I often remind my exasperated residents, you could admit every single patient with chest pain and still miss a myocardial infarction; some patients with ACS, especially the elderly, present with other complaints, such as dyspnea. The more enlightened approach is take each patient and combine the information from the history and physical examination and the data from the ECG and cardiac markers to stratify and manage the risk—not always resulting in an admission. A far cry from the day when some ED physicians were compelled to admit anyone they had tested for CK-MB elevation, under the adage, "you can't rule out an MI with one set of enzymes." In fact, the authors present a very convincing argument that you can—but only if you carefully select and screen your population, increasing the prior probability that your approach will be successful. That also reveals the only real flaw in this paper: The population was overwhelmingly female and African American. To ascertain the validity of the approach, it will be necessary to repeat the study in other demographically diverse populations. Until then, the advocated concept of risk stratification, with a more liberal approach to the outpatient workup in the low probability patient age range 25 to 40 years, is an intelligent and acceptable, if not entirely proven, approach.

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# Statin Metabolism Interactions

ABSTRACT & COMMENTARY

**Source**: Jacobson TA. Comparative pharmacokinetic interaction profiles of pravastatin, simvastatin, and atorvastatin when coadministered with cytochrome P450 inhibitors. *Am J Cardiol* 2004;94:1140-1146.

STATINS HAVE BECOME THE MAINSTAY OF PREVENTIVE cardiology. However, concern continues regarding the potential for rhabdomyolysis, especially at higher doses of these agents. Thus, Jacobson studied four groups of healthy subjects to assess the pharmacokinetics of: 1) 40 mg pravastatin or 40 mg simvastatin coadministered with 480 mg verapamil; 2) 40 mg pravastatin or 80 mg atorvastatin plus 100 mg mibefradil; 3) 40 mg pravastatin or 80 mg atorvastatin plus 200 mg itraconazole; and 4) 40 mg pravastatin, 40 mg simvastatin, or 80 atorvastatin plus 500 mg clarithromycin.

When compared with pravastatin alone, coadministration of verapamil, mibefradil, or itraconazole with pravastatin did not alter pravastatin pharmacokinetics. Clarithromycin did increase the area under the curve (AUC) of plasma pravastatin (100% $P < .001$), but increased the AUC of simvastatin 219% and atorvastatin 343%. Verapamil increased simvastatin AUC four-fold. Mibefradil increased atorvastatin AUC more than four-fold, and itraconazole increased atorvastatin AUC 47%. Clarithromycin increased the AUC of all three statins; simvastatin ten-fold, atorvastatin more than four-fold, and pravastatin almost two-fold. Jacobson concluded that pravastatin is the statin with the fewest interactions with cytochrome P450-(CYP) 3A4 inhibitors. ❖

**COMMENTARY BY MICHAEL H. CRAWFORD, MD**

The withdrawal of cervastatin from the market, and the recent physician notification that the starting dose of rosuvastatin is 10 mg, underscores the concern about anything that may increase the incidence of the rare, but serious, adverse reaction of rhabdomyolysis, which is dose related. Coadministration of other agents that share

the CYP receptor may lead to increased serum levels of certain statins for prolonged periods. These agents include gemfibrozil, calcium-channel blockers, immunosuppressives, macrolide antibiotics, certain antifungal agents, protease inhibitors of HIV, amiodarone, and grapefruit juice.

Some have estimated that more than half the rhabdomyolysis cases reported to the FDA involved coadministration of other CYP inhibitors. In fact, the package insert for simvastatin recommends a daily dose no higher than 20 mg with the coadministration of verapamil or amiodarone. Simvastatin and atorvastatin are lipophilic statins that demonstrate profound increases in drug levels over time when given with CYP inhibitors. Pravastatin is hydrophilic and is not a CYP substrate. Mibefradil is a T-channel calcium blocker, which is a strong CYP inhibitor and was withdrawn from the U.S. market because of numerous serious drug interactions. However, in this study, it did not interact with pravastatin. Other studies have shown an overall extremely low incidence of rhabdomyolysis with pravastatin, 0.04 per million prescriptions vs 0.12 for simvastatin, 0.10 for lovastatin and 3.2 for cerivastatin.

The down side of pravastatin is that it is not a particularly potent statin and may require concomitant lipid-lowering agents to achieve the desired effect. This may be the reason that Merck developed Vytorin, which combines lower doses of simvastatin with ezetimibe. Thus, when using more potent statins, one must weigh the tiny risk of rhabdomyolysis against the major benefits of effective lipid-lowering in patients with vascular disease. However, care must be taken when the coadministration of CYP inhibitors is necessary. In these situations, pravastatin may be an alternative if lipid targets can be met.

*Dr. Crawford, Professor of Medicine, Associate Chief of cardiology for Clinical programs, University of California San Francisco, is Editor of* Clinical Cardiology Alert.

# Search for a Safe Algorithm to Exclude PE Continues

ABSTRACT & COMMENTARY

**Source**: Brown MD, et al. An emergency department guideline for the diagnosis of pulmonary embolism: An outcome study. *Acad Emerg Med* 2005;12:20-25.

THIS PROSPECTIVE OBSERVATIONAL STUDY WAS designed to determine the safety of an emergency department (ED) pulmonary embolism (PE) diagnostic guideline. The study population consisted of all adult patients presenting with clinical suspicion of acute PE during a four-month period. The guideline recommended D-dimer testing in those patients younger than 70 years with a low (less than 20%) clinical suspicion of PE and no unexplained hypoxemia, unilateral leg swelling, recent surgery, hemoptysis, pregnancy, or prolonged duration of symptoms. Diagnostic imaging was recommended for patients having a positive D-dimer (ELISA) ≥500 ng/mL. For those patients with moderate to high probability of PE, the guideline recommended diagnostic imaging without a D-dimer level. The primary outcome was the identification of venous thromboembolism during a three-month follow-up period. The electronic medical record, telephone contact, questionnaires sent by mail, medical examiner records, and the Social Security Death Index were the sources of follow-up information.

Of 1207 patients evaluated for suspected PE, 71 (5.8%) were diagnosed with venous thromboembolism. There was one missed case of PE on follow-up, for a negative predictive value of 99.9% (95% CI, 99.5% to 100%). There were 677 patients with D-dimers performed and subgroup analysis yielded a sensitivity of 0.93 (95%CI, 0.77 to 0.98) and a specificity of 0.74 (95%CI, 0.70 to 0.77). ❖

## COMMENTARY BY STEPHANIE ABBUHL, MD, FACEP

The authors found that the guideline resulted in a negative predictive value (NPV) for PE of 99.9%, and thus, concluded that it is safe and feasible in a community hospital ED. It is possible that one of the reasons the guideline had such a high NPV was the low prevalence of PE in the population tested (only 5.8% overall), lower than in many other studies and much lower than the 20% prevalence the authors estimated to determine their sample size. This should not be mistaken as a negative predictive value of the D-dimer test (only sensitivity and specificity of the D-dimer was calculated). In fact, the sensitivity of the D-dimer was 0.93 with a wide confidence interval that included a lower limit of 0.77. The authors note that in 40 cases the clinician pursued radiographic studies in spite of a negative D-dimer and two (5%) patients had a PE.

A significant limitation of this study is the questionable reliability of the follow-up. As in many outcome studies for PE, the gold standard for ruling out PE—in those patients who did not have an imaging study— was the absence of an identifiable thromboembolic event in the three-month follow-up period. To determine this, the researchers reviewed the Spectrum Health electronic

medical records of all 1137 patients. But for the 896 patients who did not confirm that Spectrum Health was their usual source of care, the researchers attempted phone calls and sent letters with only a 52% success rate. County medical examiner's records were searched for matches of all subjects; when telephone and mail follow-up were both unsuccessful, a search of the Social Security Death Index was performed. The concern is that this follow-up could have missed a significant number of patients who went to another health system with a thromboembolic event.

Before considering implementation of this guideline in your ED, it should be remembered that the guideline only recommended D-dimer testing in patients who were considered by gestalt to be in a low (less than 20%) pretest probability group, who had none of four strong predictors (unexplained hypoxia, unilateral leg swelling, recent surgery or hemoptysis), and who also were younger than 70 years, not pregnant, and did not have a duration of symptoms greater or equal to four days. Even after this attempt to reduce the false positives and false negatives, the D-dimer test is still far from ideal in its test characteristics and requires a good understanding of its limitations for safe use.

## Prevalence of UTI and STD in Women with UTI Symptoms

ABSTRACT & COMMENTARY

**Source:** Shapiro T, et al. The prevalence of urinary tract infections and sexually transmitted disease in women with symptoms of a simple urinary tract infection stratified by low colony count criteria. *Acad Emerg Med* 2005;12:38-44.

THERE IS CONSIDERABLE OVERLAP BETWEEN THE presentation of lower urinary tract infection (UTI) and sexually transmitted disease (STD) in women, making the evaluation somewhat complicated. Are symptoms indicative of UTI, STD, or both? Research in this area has been hampered by a retrospective approach in some studies, as well as the inclusion of voided (and thus, potentially contaminated) specimens for UTI evaluation in others. Shapiro and colleagues sought to address those issues in their prospective study looking at the prevalence of UTI and STD in patients presenting to the ED with symptoms suggestive of the former.

Nonpregnant, female patients ages 18-55 years presenting to an urban ED were eligible if their complaint was consistent with a simple UTI—dysuria, frequency, urgency, or suprapubic pain/pressure without significant vaginal discharge. Exclusion criteria included UTI or antibiotic use within the past two weeks; vaginal or pelvic infection with chlamydia, gonorrhea, trichomonas, or yeast within the past four weeks; significant vaginal discharge; febrile presentation (T>100.4°F), or prior hysterectomy. Emergency medicine residents, medical students, and attending physicians were trained regarding study protocol and data collection, which included collection of urine specimens (by bladder catheterization with the Female Speci-Cath Kit), STD cultures (via endocervical swab sent for polymerase chain reaction for gonorrhea and chlamydia), as well as performance of microscopic analysis of the vaginal wet mount (for trichomonas or bacterial vaginosis, as well as yeast). Treatment was at the discretion of the treating physicians, and was based upon the history and physical examination, the urinalysis, and the examination of the wet mount. Presence or absence of UTI was determined by laboratory culture of the catheterization specimens using two threshold criteria: the traditional $10^5$ colony-forming units (CFU)/mL of bacteria as well as a perhaps more sensitive $10^2$ CFU/mL. The authors offered literature support from the infectious disease/internal medicine literature of the 1990s for this approach, and presented a cogent argument in the discussion section regarding this issue.

Of 528 patients ultimately diagnosed with UTI during the study period, 290 (55%) were study eligible; 94 patients were approached and ultimately 92 (32%) were enrolled, examined, and treated. Due to an unforeseen laboratory mishap, 17 of those 92 did not have UTI culture performed, leaving 75 patients for data analysis. The presence of UTI in these 75 women with UTI symptoms and urine cultures performed was 57% ($10^2$ CFU/mL) or 43% ($10^5$ CFU/mL), depending upon the criterion threshold. *Escherichia coli* was the predominant organism (54%), although nine others had only gram-negative rods (i.e., no organism) reported. The prevalence of STD in these 75 patients with UTI symptoms and urine cultures performed was 17%; the STD rate in the 91 (one missing value from the original 92) women who were enrolled, examined, and treated was not much different—14%—and no significant difference was found in rate of STD between urine culture positive and negative groups, regardless of CFU/mL criterion. Using logistic regression analysis on the 91 patients, number of sex partners during the past year was the only variable found to significantly predict who had an STD. Fifty percent of those patients ultimately found to have an STD were not diagnosed and treated as such at the end of their ED visit. The authors conclude that women with classic UTI symptomatology often do not

have UTI, and that diagnosis of either entity—UTI or STD—based solely on clinical evaluation (history, physical examination, urinalysis, and vaginal wet prep) is problematic. ❖

### COMMENTARY BY RICHARD HARRIGAN, MD, FAAEM

This topic reminds me of the approach to the patient with pharyngitis: It is a common problem we see everyday, yet the *right* way to diagnose and treat is not abundantly clear. Why does such a simple problem have such complex issues? Like most complex things, the reasons are multiple. First, doing a definitive study of such an issue requires that most, if not all, eligible patients be captured. The authors admit that their study is limited by low enrollment numbers: 198 potential study patients were not approached in this case, partly because those physicians not involved intimately in the study were less likely to enroll patients. This is a chronic frustration at academic medical centers; without a SWAT team of research associates to hound faculty and assist them with patient enrollment, busy EDs are tough places to take the time to enroll patients in a study when the caregivers are also the research data gatherers. Thus, the specter of selection bias enters this study. Another problem is defining the gold standard of disease: a colony count that defines UTI, diagnosis of trichomoniasis, or adequacy of vaginal swab for detection of gonorrhea and chlamydia. (In this study, where seven people had trichomoniasis, the diagnosis rested upon real-time identification on the ED microscope; reality in my ED, but not truly a gold standard. Assumably, we are not as good as polymerase chain reaction at detecting this disease.) Like the throat swab for streptococcal pharyngitis, the STD will go undetected if the specimen is inadequate. The authors are to be congratulated on controlling many variables that have been omitted in prior research in this area, such as in their design (prospective rather than retrospective) and their use of straight catheterization of the bladder.

So what can we take away from this study? The UTI/STD dilemma in women with symptoms of UTI remains a dilemma. Remember, women with simple UTI complaints make up the denominator here; any change in usual vaginal discharge by history or finding of significant vaginal discharge on physical examination led to exclusion. And the STD rate in this group (in this environment) was high (17%). Moreover, only 54% of those women with UTI complaints had a UTI—even using a liberal gold standard of low bacteria counts. Thus, it is probably prudent to evaluate patients completely for both STD and UTI, despite symptomatology that favors the former diagnosis.

*Special Feature*

# Right Ventricular Myocardial Infarction

*By William J. Brady, MD*

MYOCARDIAL INFARCTION OF THE RIGHT VENTRICLE (RV) most often occurs in the setting of inferior wall myocardial infarction; the culprit artery is most often the right coronary artery (RCA). RV infarction complicates approximately 25% of inferior wall acute myocardial infarctions (AMI); it is very uncommon in anterior and lateral wall AMI. Isolated RV infarction is exceedingly rare.[1] This co-infarction pattern of the inferior wall of the left ventricle and the RV is attributable to the coronary anatomy; the occlusive lesion is found in the right coronary artery (RCA), usually in the proximal third of the vessel, with involvement of various downstream arterial branches. The RCA and related branches perfuse the right atrium, RV, and additional portions of the left ventricle. The right conus artery, which usually originates from the aorta, supplies the upper anterior wall of the RV. Lastly, the posterior descending artery supplies a segment of the RV.

The classic triad of hypotension, clear lung fields, and jugular venous distension in a patient with an inferior wall AMI on electrocardiogram (ECG) is highly suggestive of RV infarction. Left ventricular filling pre sures are dependent upon cardiac preload; with RV infarction, a significant reduction in preload occurs, and hypotension likely results; this hypotension may be worsened by nitroglycerin and morphine administration due to their vasodilating properties. However, the clinician is cautioned regarding hypotension in inferior AMI; other etiologies must be considered as well.

## Electrocardiographic Manifestations of Right Ventricular Infarction

The most frequent electrocardiographic presentation is acute inferior wall ST-segment elevation AMI (STEMI).[2] (See Figure 1.) A specific pattern of ST-segment elevation in the inferior wall STEMI suggests RV AMI. ST-segment elevation of greatest magnitude in lead III (compared with leads II and aVF) is highly predictive of acute RV infarction (*Figures 1 and 2*); it suggests the right coronary artery is more likely the culprit vessel, rather than the left circumflex artery.[2] The single electrocardiographic lead from the traditional 12-lead ECG, which best reflects RV injury is lead $V_1$ – ST-segment elevation in lead $V_1$ usually is seen in RV

AMI, if co-existing acute posterior infarction is not present (*See Figure 3*). RV AMI occasionally may present with ST-segment elevation in other standard precordial leads ($V_1$ to $V_3$, or even continuing to $V_5$) mimicking an antero-septal AMI. In RV AMI, ST-segment elevation decreases in magnitude from $V_1$ to $V_5$, whereas the opposite pattern is noted in anterior wall infarction.

**Figure 1. 12-lead ECG with STEMI of the Inferior Wall**

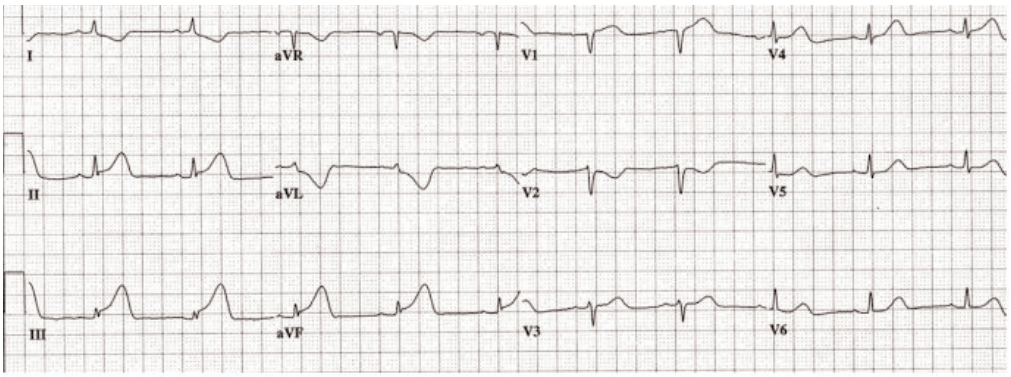


**Figure 1.** Note that the degree of ST- segment elevation is greatest in lead III compared with leads II and aVF. Also, note the slight ST-segment elevation in lead $V_1$. After nitroglycerin treatment, the patient developed hypotension, which promptly responded to IV normal saline infusion. The ECG findings, along with the clinical information, strongly suggest a right ventricular infarction in the setting of inferior wall ST-segment elevation infarction.

An important electrocardiographic abnormality suggestive of RV AMI is ST-segment elevation in additional right-sided leads. Although all six right-sided leads—$V_1$R to $V_6$R—may be used (*Figure 3A*), lead $V_4$R has been found to be the single best lead for detection of RV infarction (*Figure 3B*). The degree of ST-segment elevation in any electrocardiographic lead, whether a standard or additional lead, may be of a small magnitude due to the relatively small muscle mass in the RV. With less ventricular myocardium, a reduced current of injury is noted, producing lower magnitude ST-segment elevation.

**Figure 2. Lead $V_1$ in a Patient with Inferior Wall AMI**

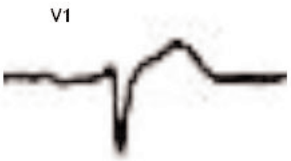


**Figure 2**. The ST-segment elevation (note its subtlety) in this lead suggests right ventricular infarction; this is true if ST-segment elevation also exists in the inferior leads. Reciprocal ST-segment depression usually is seen in leads aVL and I as well.

## Diagnostic Adjuncts and Therapeutic Options

Additional diagnostic options include echocardiography and right heart catheterization. Echocardiography may demonstrate regional wall motion abnormalities, decreased right ventricular systolic function, and/or right ventricular dilitation. Right heart catheterization, or Swan-Ganz catheter monitoring, may demonstrate elevated right atrial pressures that equal or exceed pulmonary capillary wedge pressure, resulting from a noncompliant RV and impaired left ventricular filling.

Management priorities include standard therapy (e.g., anti-thrombotic, anti-coagulant, fibrinolytic) for an acute coronary syndrome, as well as attention to preload augmentation, inotropic support, and afterload reduction. To restore preload, the clinician should administer an IV fluid bolus to maintain a blood pressure of at least 90 mmHg and/or a pulmonary capillary wedge pressure of 15 mmHg. Vasodilating (e.g., nitroglycerin) and diuretic agents should be used cautiously, if at all. When aggressive saline loading does not correct the hypotension, inotropic medication may be required. Ionotropic agents, such as dobutamine or amrinone, improve RV function, thereby improving perfusion. Severe right heart failure unresponsive to ionotropic support may respond to afterload reduction or pressor support.

Patients with inferior wall STEMI with RV infarction have a markedly worse prognosis (both acute cardiovascular complications and death) compared with patients with isolated inferior wall STEMI.[3] The su set of patients with RV infarction who develop decreased cardiac output and hypotension has increased, early in-hospital mortality. In most cases, RV function typically returns to normal. Patients sustaining severe RV damage, however, may have persistent RV dysfunction and develop chronic right heart failure. ❖

## References


1. Anderson HR, et al. Right ventricular infarction; frequency, size and topography in coronary artery disease: A prospective study comprising 107 consecutive autopsies from a coronary care unit. *J Am Coll Cardiol* 1987;10:1223-1232.
2. Saw J, et al. Value of ST elevation in lead III greater than lead II in inferior wall acute myocardial infarction for predicting in-hospital mortality and diagnosing right ventricular infarction. *Am J Cardiol* 2001;87;4:448-450.

**Figure 3. Right Ventricular Infarction: Right-sided Leads**

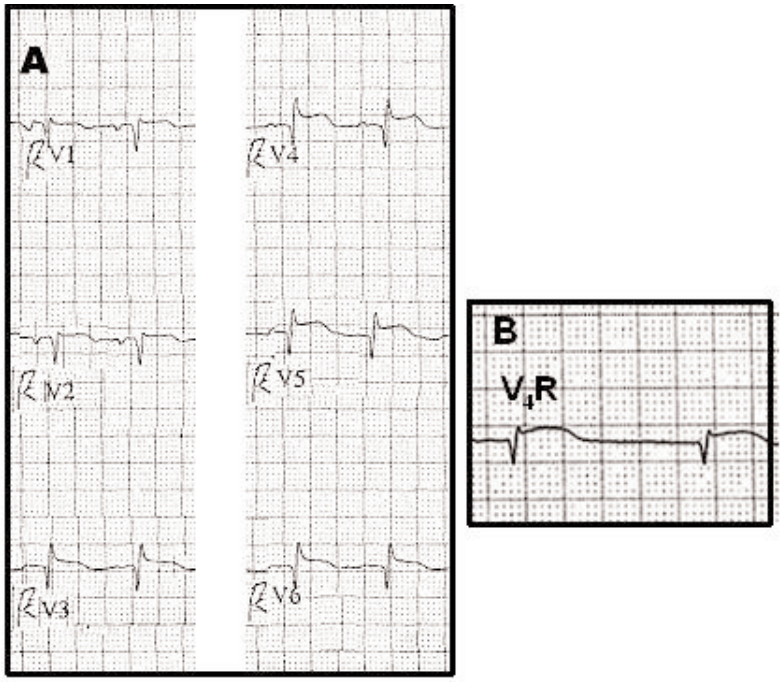


**Figure 3.** Figure A: Right precordial leads $V_1R$ to $V_6R$ demonstrating ST-segment elevation of RV infarction; the Q waves are normal and don't reflect chronicity. Figure B: Single lead $V_4R$ with ST-segment elevation.

3. Zehender M, et al. Eligibility for and benefit of thrombolytic therapy in inferior wall myocardial infarction: Focus on the prognostic importance of right ventricular infarction. *J Am Coll Cardiol* 1994;24:362-369.

# *Physician CME Questions*

**17. The most sensitive ECG lead for right ventricular infarction is the:**
a. III
b. V1
c. V2
d. V4R

**18. Hypotension, jugular venous distention, and clear lungs in the setting of acute MI are suggestive of _____ involvement.**
a. posterior wall
b. anterior wall
c. right ventricular
d. high lateral wall

**19. In the recent study by Brown and colleagues, a low D-dimer value proved to:**
a. be an effective means to exclude pulmonary embolism in pregnant patients.
b. have a 99% negative predictive value for thromboembolic events during a three-month follow-up period—in a restricted population, with a low-pretest probability of disease by clinical gestalt.
c. be useful in excluding thromboembolic disease in patients older than 70 years.
d. have a high positive predictive value for thromboembolic disease in that restricted clinical population.

**20. Using a clinical decision rule, Marsan and colleagues were able to reduce the risk of acute coronary syndrome to 0.14% with no adverse cardiovascular outcomes at 30 days. This rule specified the patients (age younger than 40 years) must have initially normal serum cardiac markers, no cardiac history, and:**
a. either a normal ECG or no cardiac risk factors.
b. two normal ECGs in the ED.
c. be female.
d. have no cardiac risk factors and a normal ECG.

**21. Using a clinical decision rule, Marsan and colleagues were able to reduce the risk of acute coronary syndrome to 0.14% with no adverse cardiovascular outcomes at 30 days. This rule was developed in a cohort of:**
a. female patients.
b. female patients younger than 40 years.
c. patients younger than 40 years.
d. male patients younger than 40 years.

**Answers:** 17. d; 18. c; 19. b; 20. a; 21.c.

## CME Objectives

To help physicians:

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

## CME Instructions

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

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

# A Subtle Reality Rhythm?

*By Ken Grauer, MD*

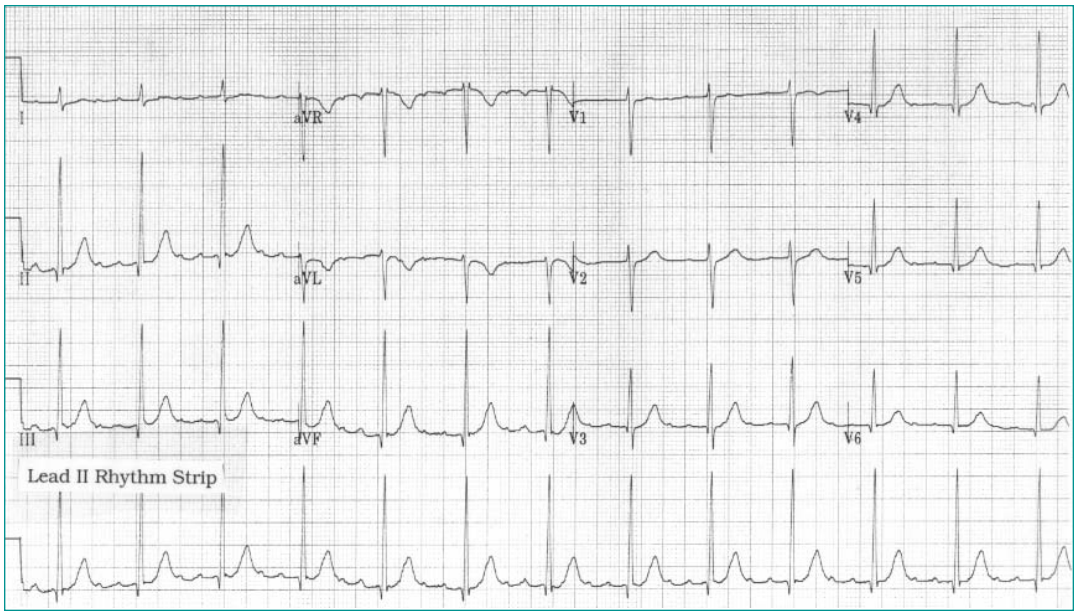


**Figure.** 12-lead ECG and rhythm strip obtained from a 74-year-old man with severe pulmonary disease.

**Clinical Scenario:** The 12-lead electrocardiogram (ECG) and accompanying lead II rhythm strip in the Figure were obtained from a 74-year-old man with severe pulmonary disease. The computer interpreted this tracing as showing normal sinus rhythm. Do you agree?

**Interpretation:** This is a difficult tracing. There are actually *two* small upright deflections seen in lead II during the interval that extends from the end of the T wave until the next QRS complex. Each of these 1-mm deflections in lead II look alike. The distance between them is approximately one large box in duration, which would correspond to a rate of 300 per minute if these deflections represented regularly occurring atrial activity.

In favor of these subtle deflections being real (i.e., not due to artifact) is the observation that they are seen in several leads at approximately the same point in the cardiac cycle. Thus, in addition to their consistent appearance throughout the lead II rhythm strip, these dual deflections also are seen in leads I, III, aVR, aVF, and $V_3$. This occurrence in multiple leads should suggest the possibility of atrial flutter. However, flutter waves generally tend to be larger in amplitude and typically manifest a sawtooth pattern, at least in the inferior leads and/or in leads $V_1$ and $V_2$. In addition, a suggestion of flutter activity in one or more leads usually is apparent throughout other points in the cardiac cycle.

Often, these can be mapped with calipers at a regular rate consistent with the rate of flutter activity. Other than the two deflections described above that occur between the end of the T wave and the next QRS complex, there is no definite indication of potential flutter activity in any other part of the cardiac cycle. This should raise the possibility of artifact (from body tremor or other reason) as the cause of these dual deflections identified in the leads we describe.

Subsequent tracings on this patient however, confirmed that these subtle dual deflections did in fact represent flutter activity. The ECG and rhythm strip in the figure are therefore an excellent example of how subtle atrial flutter can be! Clearly, definitive diagnosis of this rhythm can not be made by assessment of only the 12-lead ECG and rhythm strip shown in the figure. Instead, diagnostic maneuvers (e.g., carotid massage) and/or additional tracings are needed for diagnosis. Nevertheless, an extra deflection is present in several leads of this ECG, and recognition of this repetitive at a rate of approximately 300/minute should be enough to raise suspicion of atrial flutter and spur further investigation.❖

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# Trauma Reports

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## The Burned Patient: Assessment, Diagnosis, and Management in the ED

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*Burn injuries frequently present to the emergency department (ED). In the majority of cases, the burns are minor, yet, they require a careful assessment, cleaning, dressing, and careful follow-up. In the pediatric and geriatric populations, careful attention, to the history and physical examination, and an awareness of burn patterns associated with abuse, may protect the patient from further inflicted injury.*

*Patients with more severe burn injuries, especially those associated with house fires or explosions, should be assessed carefully for multiple trauma, and care should be taken to protect the spine until injury can be excluded clinically or radiographically. The airway of a burn patient may be particularly challenging; early aggressive intervention, when indicated, may make a potentially disastrous situation manageable. The authors review the diagnosis, classification, and management of patients who have sustained burns.* — *The Editor*

### Epidemiology

More than one million burn injuries occur in the United States each year; 700,000 of these injured individuals will seek care in an ED, and 45,000 will be hospitalized for their injuries.[1] In the United States, $2 billion are spent on burn care annually.[2]

Seventy percent of burned patients are male, and the average age of patients sustaining burn injury is 30 years. Infants account for 13% of cases, and adults older than 60 years for 11%. The extremes of age have been associated with an increased risk of death from burns and burn-related injuries.[3]

Burns can occur by several mechanisms. Scald burns arise from exposure to hot liquids or steam. Thermal burns are the result of contact with flames. Contact burns are caused by contact of the skin with hot or cold surfaces. Burns also occur from exposure to radiation, chemicals, and electricity.

Thermal, scald, and contact are the most common categories of burn injuries. In particular age groups, certain types of burns occur more commonly: scald and contact burns are prevalent from birth to 2 years of age, whereas thermal burns are common in the 5- to 20-year range.[3]

Most children dying as a result of burns sustained their injuries in house fires, and as many as 20% of pediatric burns are the result of abuse or neglect.[4] Low income, minority children are

three times more likely to die in a house fire than children in high-income categories.[5,6]

Likely causes of burn injury also vary among different ages. Mishaps with flammable liquids are common causes of burn injury in teenagers and young adults,[7] but for the elderly population, kitchen accidents and the resultant exposure to hot liquid or to an open flame are most prevalent.[8]

Burn injury also may occur in connection with industrial incidents as well as other major trauma. One-fifth to one-fourth of severe burns are work related, and 5% to 10% of burned patients sustain multisystem trauma concurrent with their burn injury.[9-11]

A study performed at Massachusetts General Hospital and Shriners Burns Institute in Boston found three distinct risk factors associated with higher mortality: age more than 60 years, burn injury greater than 40% of the total body surface area (TBSA), and the presence of concomitant inhalation injury. If none of these risk factors were present, the death rate associated with burn injury was 0.3%. The presence of one risk factor raised the mortality to 3%. Two risk factors correlated with a 33% mortality, and when all three risk factors were present, the risk of death jumped to 90%. These numbers apply only to adults younger than 90 years. If a burned patient survives the first week after the incident, the chance of death drops to 2.5%. Only 1.1% of burn injured patients surviving after 2 weeks will die from their injuries.[2]

A recent study from the Shriners Burn Institute at the University of Texas sheds some light on pediatric burn mortality numbers. On average, the investigators found that burns of 85% of the TBSA were 30% lethal. Concomitant inhalation injury increases the risk of death. The mortality rate for patients with burns affecting 50% of the TBSA and concomitant inhalation injury was 10%. In comparison, patients with burns covering 73% of the TBSA without inhalation injury had the same 10% mortality. This study also found that males younger than 3 years had a higher risk of death from their injuries than males of other ages. Lastly, Hispanic teenagers had a higher mortality from burn injury than African-American or Caucasian teenagers of the same age.[5]

## Pathophysiology

Burn wounds have three distinct zones of tissue damage: the coagulation zone, the stasis zone, and the hyperemic zone. The coagulation zone consists of the tissue that has been irreversibly destroyed from the primary injury and cannot recover. Surrounding this area is the stasis zone, where damaged tissue with decreased perfusion is located, but the potential for recovery still exists. Adjacent to the stasis zone is the hyperemic zone, the tissue that has sustained minimal damage and will recover spontaneously.[7,12,13] The tissue in the zones of stasis and hyperemia, while still viable, is at risk of destruction from poor perfusion, edema, and inflammation.[13]

Blisters form when damaged capillaries, with increased vascular permeability, leak plasma into the interstitial space. The damaged epidermis separates from the underlying dermis. The fluid of the blister contains inflammatory mediators, such as arachidonic acid metabolites, thromboxane, and calmodulin, as well as plasma proteins and cellular debris. The high osmolarity of this fluid can cause additional water absorption from underlying tissue into the blister, which increases local wound tissue pressure and may cause ischemia in the already compromised tissue.[12]

As the largest organ in the human body, the skin provides protection, immunologic defense, and acts as a barrier to fluid loss. When the skin is burned, these functions are lost, enhancing the victim's risk of systemic illness, sepsis, and multiple organ failure.

Loss of the barrier function of the skin leads to massive fluid losses from evaporation. This fluid loss impedes tissue perfusion and oxygenation. The resultant hypovolemia can result in relative hypoperfusion to distant organs.[7] Injured tissues release inflammatory mediators and vasoactive substances, causing interstitial edema and organ dysfunction. Multi-organ failure usually develops between the second and eighth week after injury and accounts for one-third of burn-related deaths.[2,14] Increased age, increased TBSA burned, male sex, and the presence of inhalation

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**Figure 1. Inflicted Immersion Burn**

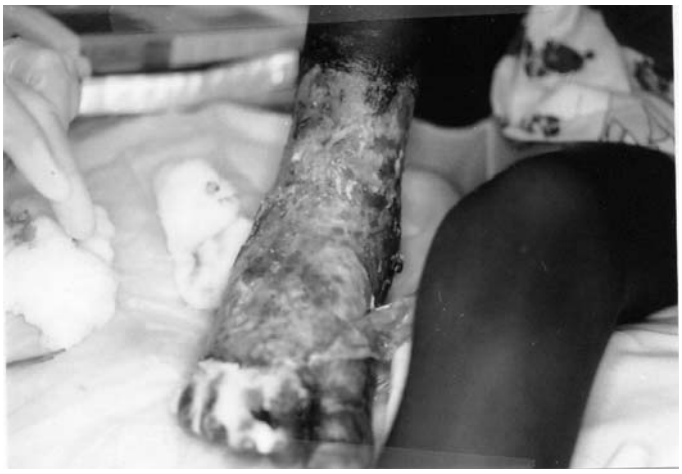


**Figure 1.** Photograph shows stocking distribution of an inflicted immersion burn. Note the clear demarcation between burned and spared skin. The child told medical staff of being forced to stand in hot water for several minutes.

*Reprinted with permission from Bechtel, K. Identifying the subtle signs of pediatric physical abuse.* Ped Emerg Med Rep *2001;6:61.*

injury all are associated with an increased likelihood of the development of multi-organ failure after burn injury.[14,15]

Loss of the skin's immunologic defenses leads to an increased susceptibility to infection. Systemic infection results from invasion of bacteria into the body through the burn eschar. Immediately following injury, the burned area is sterile, but then bacteria quickly colonize the wound. Rapidly reproducing in this avascular environment, the bacteria are able to gain access to the rest of the body.[8] Sepsis is common in burned patients and has been noted to precede the development of multi-organ failure. Advanced age and the presence of full-thickness burns are risk factors for the development of severe sepsis.[15]

In the first 1 to 3 hours after burn injury, edema develops and may increase up to 24 hours after injury.[16-18] The development of edema in the setting of burn injury is multifactorial. Vasodilation and increased transcapillary pressure in conjunction with increased extravascular osmotic activity of the burned tissue, increased microvascular permeability, and impaired cell membrane function with swelling of the cells all contribute to the development of edema.[19,20]

**Cardiac Effects**. Immediately after injury, myocardial function may be depressed; however, this typically improves within three days. This depression in myocardial function may be caused by circulating myocardial depressants or persisting hypovolemia, despite agressive fluid therapy and the lack of classic signs of hypoperfusion.[21] A hyperdynamic cardiovascular response then occurs, with an up-to-twofold increase in cardiac output.[8,22]

**Metabolic Effects.** Metabolic derangements (e.g., metabolic acidosis, respiratory alkalosis, and electrolyte disturbances) are common in patients with burn injury. Intracellular concentrations of sodium and calcium rise, while intravascular levels of potassium increase as the result of cell membrane alteration.[23]

In comparison with other critically ill patients, patients suffering from burn injuries have the highest metabolic rate.[24] The burned patient has increased energy expenditure, accelerated glycogen and protein breakdown, and lipolysis. This hypermetabolic state is the result of increased circulating catabolic hormones, catecholamines, cortisol, and glucagon.[22,24,25]

Catabolism begins by five days after injury.[24,26] Although it was once thought that catabolism resolved with wound closure, catabolism actually continues up to nine months after the initial injury.[27] The level of catabolism is increased with age, weight, and delay in surgical treatment. Catabolism intensifies with increases in TBSA burned, up to a TBSA of 40%. Sepsis, hyperglycemia, and decreased ambient air temperature also increase catabolism in these critically ill patients.[24,26,28]

Thermoregulation is altered after burn injury. In addition to the loss of the skin's protective function, which results in loss of body heat and hypothermia, the hypothalamic temperature regulation set point increases by 2º C above normal body temperature.[24,29]

The metabolic effects of burn injury can have serious sequelae for the patient. Patients experience loss of lean body mass and body weight, delayed wound healing, and immune depression as a result of their hypermetabolism. Fatty infiltration of the liver develops as a result of the increase in lipolysis.[30] Pediatric patients with severe burns have been found to have a delay in linear growth for two years after injury.[31]

## Clinical Features

**History.** A thorough history is required from every burned patient and may require information from paramedics, family, or witnesses to the injury. Standard elements, such as medical history, surgical history, medications, allergies, and last tetanus immunization, are essential. Beyond those, the mechanism of injury is perhaps the most important information. Knowing how the burn was incurred will help direct the workup and physical examination and help delineate whether the burn injury occurred intentionally. The mechanism of burn injury also may indicate whether the inhalation of toxic gases, such as carbon monoxide or cyanide, may have occurred. Current use of alcohol or illicit drugs also is important to ascertain; it may have contributed to an alteration in mental status, the mechanism of injury, or comorbidities. In pediatric patients, it is essential to ascertain the circumstances surrounding the injury, maintaining a high index of suspicion for intentional acts.

**Physical Examination.** The physical examination of the burned patient begins with assessment of the ABCs (airway, breathing, circulation). After management of these crucial elements, a secondary survey focusing on recognizing concurrent traumatic injuries, if there is associated trauma, should be completed. Evaluate the patient's face and oropharynx for carbonaceous sputum, circumoral burns, and singed nasal hair, which could indicate the presence of an inhalation injury. During this survey, patterns of injury in pediatric patients that may indicate abuse, such as a stocking or glove-like appearance with sharp

## Figure 2. Rule of Nines (Adult)

| ADULT BODY PART | % OF TOTAL BSA |
|---|---|
| Arm | 9% |
| Head | 9% |
| Perineum | 1% |
| Leg | 18% |
| Anterior trunk | 18% |
| Posterior trunk | 18% |

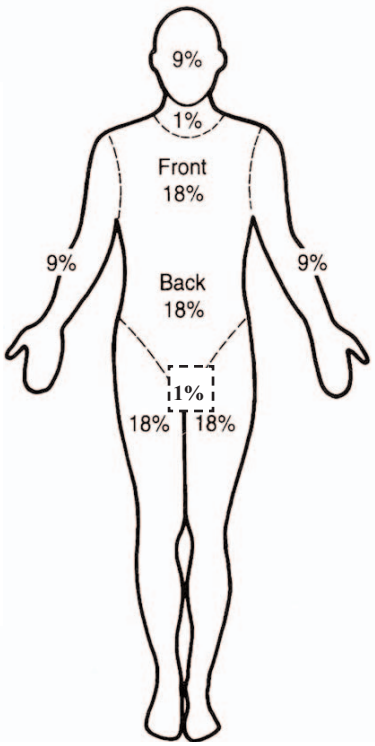
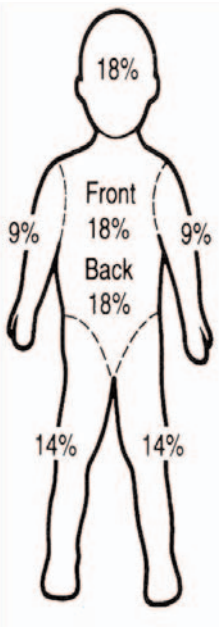



**Figure 2**. The Rule of Nines is designed to rapidly access the percentage of TBSA for a burn injury.

*Illustration courtesy of the authors.*

## Figure 3. Rule of Nines (Child)




| CHILD BODY PART | % OF TOTAL BSA |
|---|---|
| Arm | 9% |
| Head and neck | 18% |
| Leg | 14% |
| Anterior trunk | 18% |
| Posterior trunk | 18% |

**Figure 3.** Modification to the adult Rule of Nines to reflect the different proportions in the pediatric population.

*Illustration courtesy of the authors.*

margins, should be recognized. *(See Figure 1.)* The severity of the burn injury also should be appraised.

**Severity of Burn Injury**. To determine the severity of a burn injury, assess both the TBSA burned and the depth of the burn injury. The TBSA measurement is used to estimate fluid resuscitation requirements and to assess the risk of death. Burn depth is used to assess the burned patient's need for hospitalization, the need for surgical intervention, as well as the probability of scar development after the wound heals.[32]

There are three methods of estimating the total body surface area burned (First-degree or superficial burns are excluded from BSA calcuations). The Rule of Nines is the most commonly used system to estimate the extent of burn injury. It is much more accurate for adults than for children. The body is divided into areas that represent 9% of body surface area or multiples of 9%. The exception is the perineum, which is assigned the value of 1%. *(See Figure 2.)* For the pediatric patient, the Rule of Nines is altered by taking 4% from each leg and 1% from the perineum to add an additional 9% to the surface area of the head. *(See Figure 3.)* The values associated with affected areas are summed to estimate the TBSA burned.[3,33]

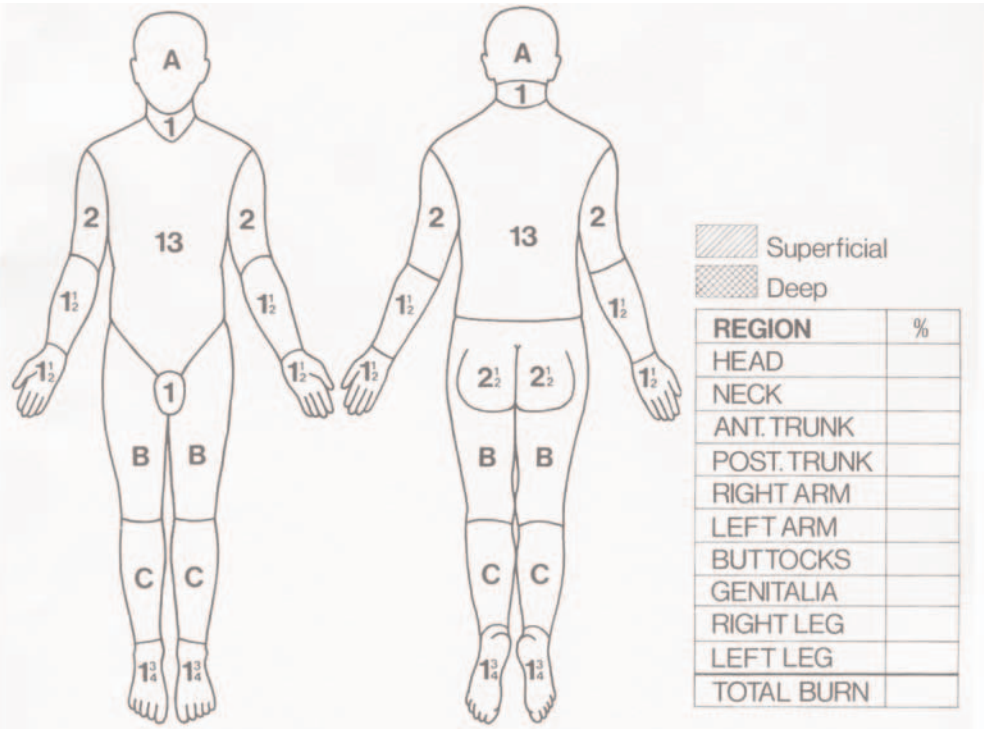
The Lund and Browder chart is considered a more accurate estimation tool to determine TBSA, especially for pediatric patients. This chart also divides the body into areas and assigns a percentage body surface area based on the patient's age. This chart accounts for the differences in proportionality between newborns, children, and adults.[3,34] *(See Figure 4.)*

Another frequently used technique for estimation of injury uses the surface area of the patient's palm, considered to represent 1% of the TBSA. This method is best used for patients with scattered small burns and is believed to be the least accurate of the three methods. Investigators at the University College London Medical School called this method into question. Their research found that the palmar surface of the hand represented only 0.4% body surface area of adults and 0.45% body surface area in children.[35]

Given the increasing incidence of obesity in our society, it is important to consider how estimation of TBSA in the obese patient differs from that process in nonobese patients. Underestimation of burned area on the trunk and legs becomes more common with increasing obesity. The trunk may constitute up to 50% of TBSA in the obese patient, while each leg may account for 20%. The head and arms of the obese patient account for a smaller body surface area than that assigned to them by the Rule of Nines.[36]

The depth of burn injury commonly has been referred to in terms of first-, second-, and third-degree burns. First-degree burns affect only the epidermis. They are warm and red, have no blisters, and generally are painful. In second-degree burns, both the dermis and epidermis are involved. Most burns of this type will be painful, red, and blistered, with moist bases. In third-degree burns, the entire dermis and epidermis are destroyed.

**Figure 4. Lund and Browder Chart**




| REGION | % |
|---|---|
| HEAD | |
| NECK | |
| ANT. TRUNK | |
| POST. TRUNK | |
| RIGHT ARM | |
| LEFT ARM | |
| BUTTOCKS | |
| GENITALIA | |
| RIGHT LEG | |
| LEFT LEG | |
| TOTAL BURN | |

RELATIVE PERCENTAGE OF BODY SURFACE AREA AFFECTED BY GROWTH

| AREA | AGE 0 | 1 | 5 | 10 | 15 | ADULT |
|---|---|---|---|---|---|---|
| A = ½ OF HEAD | 9½ | 8½ | 6½ | 5½ | 4½ | 3½ |
| B = ½ OF ONE THIGH | 2¾ | 3¼ | 4 | 4½ | 4½ | 4¾ |
| C = ½ OF ONE LEG | 2½ | 2½ | 2¾ | 3 | 3¼ | 3½ |

**Figure 4.** The Lund and Browder Chart is the most accurate estimation tool to determine TBSA and accounts for differences in proportions between children and adults.

*Illustration courtesy of the authors.*

These burns appear leathery and dry and are typically tan in color. Third-degree burns usually are painless, secondary to destruction of pain receptors in the dermis.[37] In some instances, the term "fourth-degree burn" also is used. These are burns that involve underlying tissue such as muscle, bone, or fascia.

However, these categories have been replaced recently by the more accurate and less confusing terms of superficial, partial-thickness burns (which include both superficial and deep partial thickness), and full-thickness burns. This system of categories accounts for the anatomic structures affected by the burn (Table 1).[38]

Superficial burns affect only epidermis. Erythema is present, but no blisters form. The surface is usually dry and painful to touch. In certain areas, such as around the eyes, there also may be edema. These burns usually heal in 3 to 7 days and generally do not lead to scarring.

In superficial partial-thickness burns, injury extends through the epidermis into the superficial layers of the dermis. These burns are extremely painful. The burned area is erythematous, and the surface blanches readily, with brisk capillary refill. Blisters develop rapidly. Owing to the extensive vasculature of the epidermis, these are moist wounds and produce moderate edema.

Extending to the deeper layers of the dermis, deep partial-thickness burns may have a red and white waxy or mottled appearance. Although these wounds continue to blanch, capillary refill may be slow or entirely absent. Blisters usually are not present. The surface of the wound is moist, notable edema is present, and sensation is altered.[39] Most partial-thickness burns (both superficial and deep) heal spontaneously within 14 days. The extent of scarring resulting from these burns depends upon the depth of the burn. If located over a joint, these burns may require skin grafting.[38]

Full-thickness burns destroy epidermis and dermis and extend into the subcutaneous tissue. They have a white or charred appearance without any blistering. These burns are insensate secondary to destruction of sensory nerves; however, the area of a full-thickness burn will be bordered by an area of less severely burned tissue, which is painful. Subdermal burns extend into muscle, fascia, and bone. Burns of this magnitude require surgical intervention and have an associated risk of systemic disease. Fluid and protein shifts cause intense edema.[38,39] Skin grafting leads to extensive scarring, development of contractures, and impaired mobility.

Customarily, the determination of burn depth is made clinically; however, in serious burn injury, this assessment can be complicated. Wound biopsy has been used to histologically identify burn depth more accurately by examining the tissue for evidence of blocked and patent vasculature.[40] The less invasive technology of laser Doppler imaging (LDI) also is being used to assess microvascular blood flow in the dermis to determine burn depth.

LDI combines laser Doppler technology with scanning techniques to produce an image of tissue perfusion by tracking red blood cell movement.[41,42] This technology has been found to assess burn depth accurately in 97% of injuries compared with 60% to 80% by clinical assessment alone for adult patients.[41] In pediatric burned patients, this technology has been found to have a sensitivity of 90% and specificity of 96% for the detection of deep partial or full-thickness burns when used 36 to 72 hours after injury.[42] This technology has yet to become widespread in

**Table 1. Burn Depth**

| CLASSIFICATION | FORMER CLASSIFICATION | STRUCTURES INVOLVED | COLOR | BLISTERS | PAIN |
|---|---|---|---|---|---|
| Superficial | First degree | Epidermis | Red, dry | No | Yes |
| Partial-thickness, superficial | Second degree | Epidermis into superficial dermis | Red, moist | Yes | Yes |
| Partical-thickness, deep | Second degree | Epidermis into deep dermis | Red/white, waxy | No | Altered |
| Full-thickness | Third degree | Epidermis, dermis, into subcutaneous tissue | White or charred | No | None |
| Subdermal | Fourth degree | Epidermis, dermis, into fascia, muscle, bone | White, charred, variable depending on involved structures | No | None |

use, but it has the potential to become a useful adjunct in the assessment of burn injury depth in the future.

## Diagnostic Studies

Burned patients should be placed on a cardiac monitor, and pulse oximetry should be assessed and monitored, if indicated. Basic laboratory studies should be obtained in patients with severe burns or concomitant trauma, including a complete blood count, type and crossmatch, chemistries, coagulation profiles, arterial blood gas measurement, and a pregnancy test, when appropriate. All patients with thermal burns should have arterial or venous blood sent for measurement of the carboxyhemoglobin level to evaluate for carbon monoxide poisoning.

An initial chest radiograph is warranted in all burned patients when an inhalation injury is possible. A normal study does not rule out pulmonary injury however, and serial chest radiographs may show delayed development of pulmonary edema or findings of pulmonary contusions. Computed tomography scans should be obtained as indicated in the patient with accompanying traumatic injuries or decreased mental status. In addition, the history and physical examination should guide radiologic examination of the extremities and cervical spine.

## Initial Management

Stabilization of the ABCs is essential in managing any medical emergency. The initial approach to managing severe burn victims is no exception. Because severe burns often are associated with nonthermal injuries, seriously burned patients must be viewed as trauma patients and should be stabilized according to Advanced Trauma Life Support and the American College of Surgeons Committee on Trauma protocol.[8,43]

The first priority in stabilizing these patients is ensuring a patent airway, which can be challenging, secondary to oropharyngeal and laryngeal edema. Airway edema may progress rapidly in a burned patient who has inhaled heated gases or toxic products of combustion. Signs that indicate the patient may have had a significant inhalational injury include singed nasal hairs, facial burns, oral burns, sooty sputum, and stridor or wheezes. Fiberoptic laryngoscopy or bronchoscopy may be helpful in assessing the degree of airway trauma.

Once the airway is established, it is paramount to secure it; laryngeal edema, even more than oropharyngeal edema, makes intubation difficult in burn patients. If circumferential burns of the chest or an extremity are present, emergent escarotomy may be necessary. If the chest eschar compromises ventilatory motion incisions along the the costal margin, anterior axillary lines and across the top of the chest may be necessary to allow adequate chest movement. Circumferential eschar of an extremity may act as a tourniquet restricting adequate blood flow to an extremity. In this case, an escarotomy should be performed along the lateral aspect of the extremity, through the entire depth of the eschar, to allow the return of adequate blood flow. Obtaining intravenous access also is extremely important for adequate fluid resuscitation. If peripheral access is unobtainable, central access, or intraosseous access in children, generally is required.

## Fluid Resuscitation

Fluid resuscitation in severe burn victims is controversial in many aspects. Despite many years of research in the area of burns, there does not seem to be standardization regarding the type of infusion fluid to use or definitive resuscitation endpoints.

The American Burn Association (ABA) suggests that patients with burns greater than 15% TBSA should undergo fluid replacement according to the Parkland Formula:[3]

**4 mL (of IV fluid) x weight (kg) x % TBSA burn = Total amount of fluid for the first 24 hours**

## Table 2. Consensus Formulas for Fluid Resuscitation of Burned Patients after 24 Hours

| TBSA BURNED (%) | FLUID ADMINISTRATION |
|---|---|
| 0-30 | None |
| 30-50 | 0.3 mL/kg/%burn/24 hr |
| 50-70 | 0.4 mL/kg/%burn/24 hr |
| 70-100 | 0.5 mL/kg/% burn/24 hr |

**Key**: TBSA = total body surface area.

## Table 3. Resuscitation Endpoints

| | |
|---|---|
| • Sensorium | Arousable and comfortable |
| • Digital temperature | Warm peripherally |
| • Systolic blood pressure | |
| - For infants – | 60 mmHg systolic |
| - For older children – | 70–90 mmHg + 2 x age in years |
| - For adults – | mean arterial pressure >60 mmHg |
| • Pulse | 80–180/min (age dependent) |
| • Urine output | 0.5–1 mL/kg/hr (glucose negative) |
| • Base deficit | <2 mEq/L |

*Adapted from Sheridan RL. Comprehensive treatment of burns.* Curr Probl Surg *2001;38:641-756.*

This formula currently is the gold standard and applies only to adults. However, the Parkland formula is for replacement fluid administration and does not include approximation of maintenance fluids. Pediatric considerations will be discussed later in this article. Half of the calculated amount is given intravenously during the first 8 hours, and the rest is given during the remaining 16 hours. If initial fluid administration is delayed, half of the calculated volume is to be completed by the end of the eighth hour after injury. Interestingly, even though current teaching is to give fluids aggressively to burn victims, some studies have suggested that this type of fluid management increases oxygen delivery to ischemic tissue, triggering free radicals that can further damage tissue.[44]

The fluid of choice for initial resuscitation is an isotonic crystalloid fluid, such as lactated Ringer's solution. The lactate replaces the chloride in the solution, decreasing the likelihood of hyperchloremic acidosis.[8] Many studies have explored the use of hypertonic solution (i.e., 3% saline solution) as an alternative for burn victims.[22] Some studies have shown that use of a hypertonic solution may decrease the extent of edema.[45,46] However, the outcomes of studies that compared hypertonic solution versus crystalloid solution are inconclusive and suggest the use of such hypertonic solution does not improve, and may worsen, outcomes.

During the second 24 hours of resuscitation, fluid administration should decrease. This premise is based on the belief that after 18 to 24 hours, if resuscitation is successful, capillary integrity improves and therefore fluid requirements decrease.[8] The consensus formulas for fluid administration after the first 24 hours are listed in Table 2.

At this point in fluid management, the fluid of choice is a colloid formula, such as 5% albumin lactated Ringer's solution. In addition, electrolytes should be monitored closely.

The aforementioned formulas should be used in burn victims to achieve suggested endpoints of resuscitation. Infusion rates of fluids can be adjusted based on the resuscitation endpoints. *(See Table 3.)*[8] Resuscitation endpoints include stable vitals signs, normal mentation and sensorium. One important endpoint is maintaining adequate urine output, specifically 30-50 mL per hour in adults and 1mL/kg per hour in children.[47,48] `

## Wound Care

Immediate wound care for burns is important for many reasons. Topical agents for less serious burns provide a means of pain control and decrease the rate of bacterial growth. Deeper burns may require surgical management or subsequent transfer to a burn center. How a burn wound is treated depends upon the depth of the wound. Overall, outpatient management of burns can be divided into the six Cs: clothing, cooling, cleaning, chemoprophylaxis, covering, and comforting.[32]

**Clothing.** Clear the patient's body of all materials that are hot or burned. In addition, clothing that appears to have come into contact with any chemicals also should be removed.

**Cooling.** Cold water has many purposes for burn wounds. Applying gauze soaked with cold water stops the burning process, relieves pain, and removes chemicals from direct contact with skin. Use caution with cooling methods for patients with burns greater than 10% TBSA; they may cause hypothermia, especially in children. Cold water should be applied to the burn area for at least 10 minutes and a maximum of 20 minutes.[49]

**Cleaning.** Cleaning a wound is essential to prevent infection; however, the procedures can cause a great deal of pain. Local, regional, or systemic anesthesia should be induced before cleaning a wound; topical anesthesia and injection of anesthetic agents directly into the wound should be avoided. There is increasing support for using mild soap and tap water to wash burns.[50-53] Disinfectants, such as povidone–iodine solution or chlorhexidine gluconate solution, should be avoided because the agents will hamper the healing process. If there are any residues adhering to the wound, such as tar or asphalt, they should be removed with the aid of large amounts of bacitracin ointment applied during a period of many days.

Blister formation commonly is associated with burns. Intact blisters can allow re-epithelialization 40% faster than blisters that are aspirated or deroofed.[13] However, studies have shown that blister fluid contains proteins that increase the likelihood of sepsis by decreasing normal lymphocytic and neutrophilic function. Deroofing minor blisters is controversial and needs further research. Some study results have suggested that small-to-moderate-sized blisters be covered with occlusive dressing for the first 72 hours after injury. Large blisters or blisters over a joint may be aspirated with a needle and a syringe leaving the roof of the blis-

ter intact. After 72 hours, the blistered skin may be excised using aseptic technique.[12]

**Chemoprophylaxis**. Burns are considered to be tetanus-prone wounds. Active immunization against tetanus should be given to burn patients when tetanus status is in doubt. For patients who are not immunized or who have incomplete tetanus status, passive tetanus immunization is recommended.[22]

Burn wounds are more vulnerable to infections and, ultimately, sepsis. The common pathogens that cause infection in burn victims are *Staphylococcus aureus, Pseudomonas aeruginosa, Streptococcus pyogenes*, and other coliform bacilli.[54]

Topical antibiotics are an essential element in burn wound management. Classically, the medication of choice is silver sulfadiazine. It is a good selection for most burns, especially for deep partial-thickness burns, because it may permit wound healing without the need for a skin graft. Silver sulfadiazine cream should not be used on the face or in patients who are pregnant, newborns, or nursing mothers with children younger than 2 months because of the risk of sulfonamide kernicterus. In addition, silver sulfadiazine is contraindicated in patients with sulfa allergies. A cerium nitrate silver sulfadiazine cream also is commercially available. Cerium is a lanthamide metal that interacts with calcium, which is an important element of epidermal cell growth. A few studies suggest that cerium, in conjunction with silver sulfadiazine, can decrease local inflammation and sepsis.[55]

Bacitracin also can be used as a topical antibiotic for wound management. The advantage of using bacitracin over silver sulfadiazine is its lower cost. However, studies have not compared the efficacies of one topical antibiotic with another.

Biologic dressings, such as xenograft and allograft, also may be used to prevent wound contamination and fluid loss. These dressings are associated with lower infections rates and faster healing compared with silver sulfadiazine.[39] Biologic dressings need to be applied within 6 hours after burn injury. These dressings allow skin epithelialization and eventually will peel off as the skin heals.

Nonbiologic dressings provide a moist wound environment and fast healing. These dressings require fewer changes and induce less pain compared with topical antibiotics.[39] However, nonbiologic dressings need a bulky dressing that must be changed daily.

**Covering.** Superficial burns generally do not need wound dressings. Patients with this type of burn should be instructed to see their physician if blisters form. Also, a skin lubricant, such as aloe vera, can be applied to the burn wound.

Partial and full-thickness burns should be covered with sterile dressings after the wound is cleansed and a topical antibiotic is applied. Patients should be instructed to change dressings with recommended frequencies of twice a day to once a week.[56] At each dressing change, the wound should be cleaned gently, a topical antibiotic should be applied, and the wound re-dressed.

**Comforting**. A burn injury can be extremely painful. Patients with small burns may be instructed to take nonsteroidal anti-inflammatory drugs and acetaminophen. Nonsteroidal anti-inflammatory medications also decrease inflammation and edema and increase blood flow. Opioids taken orally may be added for pain control. For more painful burns, adults should be given morphine intravenously or intramuscularly. Aggressive pain control should be pursued with some adults requiring large doses of intravenous morphine.

**Table 4. Burn Center Referral Criteria**

**PATIENTS WHO MEET THE CRITERIA LISTED BELOW SHOULD BE REFERRED TO A BURN CENTER**

- Partial-thickness and full-thickness burns greater than 10% of the total body surface area (TBSA) in patients younger than 10 years or older than 50 years
- Partial-thickness and full-thickness burns greater than 20% TBSA in other age groups
- Partial-thickness and full-thickness burns involving the face, eyes, ears, hands, feet, genitalia, or perineum, or those that involve skin overlying major joints
- Full-thickness burns greater than 5% BSA in any age group
- Electrical burns, including lightning injury. (Significant volumes of tissue beneath the surface may be injured and result in acute renal failure and other complications.)
- Significant chemical burns
- Inhalation injury
- Burn injury in patients with pre-existing illness that could complicate management, prolong recovery, or affect mortality
- Any burn patient in whom concomitant trauma poses an increased risk of morbidity or mortality may be treated initially in a trauma center until stable before transfer to a burn center.
- Children with burns seen in hospitals without qualified personnel or equipment for their care should be transferred to a burn center with these capabilities.
- Burn injury in patients who will require special social and emotional or long-term rehabilitative support, including cases involving suspected child abuse or neglect

*Adapted from Committee on Trauma, American College of Surgeons. Guidelines for the operation of burn units. Resources for Optimal Care of the Injured Patient 1999. 1998:55.*

## Special Considerations

**Pediatric Burns.** In general, the management of pediatric burns is similar to that of adult burns. Children have smaller and shorter airways, which can make intubation difficult, especially if the child has fast-developing edema. Children require larger amounts of fluid during resuscitation because they have larger insensible fluid losses. Pediatric patients also have a larger surface-to-mass ratio, which makes temperature control difficult. As always, in managing any emergency, the ABCs in the pediatric burned patient must be secured during initial resuscitation. To make up for fluid loss, including insensible fluid loss, the following formula can be used:

**(5,000 mL/M$^2$ BSA burned/24 hr) +**
**(2,000 mL/M$^2$ BSA nonburned/24 hr)[9]**

Half the calculated fluid is given during the first eight hours, and the rest is given during the next 16 hours. As in adults, pain

**Figure 5. Oral Commissure Burn**

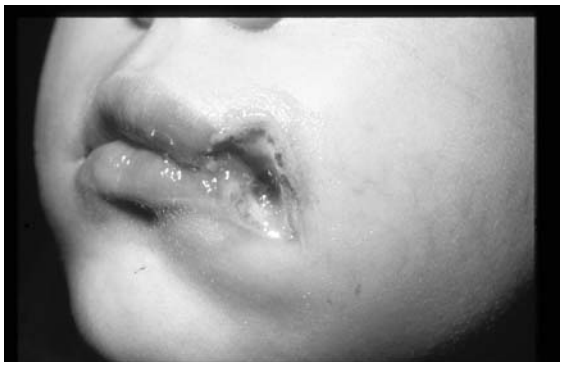


**Figure 5.** An oral commissure burn may occur when a child chews on a live electrical cord.

*Reprinted with perrmission from Stewart C. Electrical injuries. Ped Emerg Med Rep 2001:1:4.*

medication is fundamental in managing burns in children. A variety of pain medications can be used, such as nonsteroidal anti-inflammatory agents, opiates, benzodiazepines, neuroleptics, and dissociative drugs.[57] For example, a suggested starting dose of opiates (e.g., morphine) can be given in a dose of 0.1 to 0.2 mg/kg intravenously every 30 to 60 minutes as the patient's blood pressure and respiratory status tolerate. Some patients may require significantly higher doses.

**Geriatric Patients.** As the population ages, geriatric patients become increasingly common in EDs throughout the United States and other countries. Elderly females are more likely than elderly males to sustain a burn injury, and a lack of supervision is common.[58] The elderly are more likely to sustain a flame injury or a scald injury, and the majority of burn injuries have been reported to occur secondary to impaired judgment, mobility or both.[58] With increasing age, the survival decreases with reported survival rates in one series 86% in the 59-69 year old age group, 69% in the 70-79 year old age group, and 47% in patients older than 80 years.[58] In addition, the degree of concomitant chronic illness has a detrimental effect on survival.

**Inhalation Injury.** Inhalation injury is common in burn patients. Many people who sustain a burn injury were confined in a smoke-filled area and subsequently were exposed to large amounts of carbon monoxide. Usually 100% oxygen via face mask or intubation will reduce the half-life of carbon monoxide and is sufficient to treat an inhalational injury. However, hyperbaric oxygen also is a treatment option and usually is reserved for patients with carbon monoxide levels greater than 25%.[59] Other potential criteria for use of hyperbaric oxygen include patients with coma, transient loss of consciousness, ischemic electrocardiogram changes, focal neurologic deficits, and who are pregnant.[60]

**Electrical Injuries.** Electrical injuries also are considered a type of burn injury and often are managed at burn centers. Exposures to voltages of 200 to 1000 volts are considered moderate and are associated with local injury. Exposure to more than 1000 volts induces high-voltage injuries. They are associated with compartment syndromes, loss of consciousness, and myoglobinuria. Patients with these injuries must be monitored for cardiac arrhythmias for at least 24 to 72 hours.[8] In addition, delayed ophthalmologic and neurologic injuries can occur. Urine also must be monitored for myoglobinuria, and appropriate fluid must be administered.

**Oral Electrical Burns in Children.** Oral electrical burns in children usually are incurred by the sucking or chewing on the female end of a live extension cord or by biting through an electrical cord.[61] This type of injury is most common in children younger than 2 years, with a male predominance.[61] Electrolyte-rich saliva completes the circuit between the two electric poles. This results in an arc burn generating intense heat between 2500° and 3000° C that causes tissue necrosis.[62,63] The severity of the burn depends upon the length of contact, type of current, voltage, resistance of the tissue, and path of the electrical current through the body. [62,63] The low-voltage mechanism of this injury, usually resulting from household appliances consuming between 110 and 220 volts, leads to muscle contraction actually prolonging the length of exposure to the current.[63]

Oral electrical burn wounds have a central necrotic zone that appears gray or white. Surrounding this area, the tissue is edematous and raised. The most common area of involvement is the oral commissure. (*See Figure 5.*) The lower lip and cheek more likely are involved than the upper lip.[64] The heat of the electrical arc causes coagulation of tissues and thrombosis of blood vessels. As a result, bleeding is not very common in the acute phase of the injury. Edema of the surrounding tissues peaks within the first 24 hours.[65] Due to involvement of neural structures, the burns are usually painless and may result in deficits in sensory and motor function.[61,62]

In the acute care setting, these burn patients initially should be evaluated for systemic effects including arrhythmias and other injuries, which are unusual.[65,66] The wound should be irrigated, and topical antibiotics should be applied. Distinguishing between the viable and non-viable tissue is difficult in the acute phase, and debridement should not be attempted.[67] After an observation period in the ED, patients with isolated oral burn injury may be managed as outpatients with expeditious follow-up by a burn specialist and by a pediatric dentist.[64]

One of the most serious complications of oral electrical burns is delayed bleeding from the labial artery. This is most common 7 to 10 days post burn when swelling subsides and the eschar separates from the underlying tissue.[63,66] The patient's guardians should be warned of this possibility and should receive instruction on the proper application of pressure during transport back to the hospital should that occur. In the ED, the bleeding should be managed by applying direct pressure, if necessary, followed by applying epinephrine-soaked packing, and ultimately by suturing.[62]

Oral electric burns can be treated by different modalities. The use of oral splints to reduce wound contracture and to maintain position of the affected tissues during the healing process has been advocated.[62] The appliance is created by a pediatric dentist

and applied in the first few weeks post injury and commonly is worn for 6 to 12 months.[62] Surgical repair of tissue defects usually is delayed for at least 6 months after injury. At this time, the amount of scarring and the functional deficit can be better assessed.[2] The goals of reconstructive surgery are restoration of functionality and aesthetics.[67] These patients should be followed by a plastic surgeon well into their adolescence until fully grown.[7] Ultimately, most of these patients will have almost complete functional recovery of the mouth.[64]

### Disposition

Most small superficial burns and some partial-thickness burns can be managed on an outpatient basis. Adequate patient education on dressing changes, topical medications, and appropriate follow-up is necessary. A patient who does not have follow-up resources should be encouraged to return to the ED for wound checks.

Some patients with partial-thickness burns need to be transferred to a burn center. The American Burn Association has published criteria for patients needing specialized care that are listed in Table 4.[68] Refer questions regarding specific patients to a burn center physician for consultation.

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## CME/CE Questions

1. Burns can occur by several different mechanisms. What are the three most common types of burn injuries?
   A. chemical, scald, and electrical
   B. electrical, thermal, and scald
   C. scald, contact, and thermal
   D. radiation, chemical, and contact

## CE/CME Objectives

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

a) recognize or increase suspicion for traumatic injuries that present to the emergency department;
b) describe the various modalities used to identify different traumatic conditions covered in the newsletter;
c) describe how to correctly and quickly stabilize, and then to manage patients with the particular condition covered in the newsletter; and
d) identify both likely and rare complications that may occur with traumatic injuries.

## CE/CME Instructions

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

2. Burn wounds consist of three distinct zones of tissue damage: the coagulation zone, the stasis zone, and the hyperemic zone. Which of the following is an accurate description of the stasis zone?
   A.. Tissue irreversibly destroyed by heat, which cannot recover
   B. Damaged tissue with decreased perfusion and the potential for recovery
   C. Intermediate damage to tissue, with no chance of recovery
   D. Minimal damage to tissue, with the chance of spontaneous recovery

3. The depth of burn injury commonly has been classified in terms of first, second, and third degree. However, these categories have been replaced recently by the terms superficial, partial thickness, and full thickness. Which statement below best describes partial-thickness burns?
   A. They only affect the epidermis, have no blister formation, heal in 3 to 7 days, and do not lead to scarring.
   B. They involve the underlying tissue, such as muscle, bone, or fascia; are extremely painful; and lead to systemic disease.
   C. They destroy the epidermis and dermis, extend into the subcutaneous tissue, and are insensate secondary to the destruction of nerves.
   D. They include the subcategories of superficial and deep and extend through the epidermis into the superficial and deeper layers of the dermis, respectively.

4. Which of the following statements accurately describes why it is important to assess the total body surface area (TBSA) of the burned patient?
   A. It is used to estimate fluid resuscitation requirements and to assess the risk of death.
   B. It is used to assess the patient's need for surgical intervention and stabilization.
   C. It is used to determine the probability of scar development after the wound heals.
   D. It uses the Rule of Nines, equally accurately in adults and children, to estimate the extent of burn injury.

5. To evaluate the burn patient for carbon monoxide poisoning, one must complete which of the following tests?
   A. serial chest radiographs and pulse oximetry
   B. measurement of arterial or venous blood carboxyhemoglobin level
   C. complete blood count, chemistries, and coagulation studies
   D. arterial blood gas measurements and liberal CT scans

6. The American Burn Association (ABA) recommends using which of the following for burns greater than 15% TBSA during the first 24 hours?
   A. The Resuscitation Consensus Formula
   B. Hypertonic solution (i.e., 3% saline solution)
   C. The Parkland Formula as the gold standard for adults
   D. A combination of colloid formula and isotonic crystalloid fluid

7. What is the essential reason to clean burn wounds?
   A. To ensure decreased blister formation
   B. To immediately remove adherents such as tar and asphalt
   C. To prevent the separation of the epidermis from the dermis
   D. To prevent future infection of the burn wound

8. Which are the most common pathogens that cause burn wound infections?
   A. *Staphylococcus aureus, Streptococcus pneumoniae, and Clostridium perfringens*
   B. *Pseudomonas aeruginosa, Streptococcus pyogenes, and Staphylococcus aureus*
   C. *Streptococcus pyogenes, Clostridium perfringens, and Staphylococcus epidermidis*
   D. *Staphylococcus epidermidis, Pseudomonas aeruginosa, and Streptococcus pneumoniae*

9. Topical antibiotics are an essential element in burn wound management. Classically, silver sulfadiazine is the medication of choice and can be used in which of the following patients?
   A. A pregnant woman with ≥ 15% TBSA burns
   B. A patient who has burn wounds to the face
   C. A patient with deep partial-thickness burns and no sulfa allergies
   D. A newborn with superficial extremity burn wounds only

10. Management of burns in children is similar to that of burns in adults with a few exceptions. Which of the following statements is true?
   A. Children require larger amounts of fluid during resuscitation because they have larger insensible losses.
   B. Pediatric patients have smaller surface-to-mass ratio, which makes their temperature easier to control.
   C. Children have a smaller and shorter airway, which makes intubation easier and right main stem intubation less likely.
   D. Pain medication is not as important in managing pediatric burns, and nonsteroidal anti-inflammatory agents usually are adequate.

**Answer Key:**

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

## In Future Issues:

**Maxillofacial trauma**