Enclosed in this issue: CME Evaluation

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INSIDE

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For Best Outcomes: Have Your MI During Normal Business Hours

ABSTRACT & COMMENTARY

By ***Andrew D. Perron, MD, FACEP, FACSM,***
Residency Program Director, Department of Emergency Medicine, Maine Medical Center, Portland, ME.
Dr. Perron has reported no relationships with companies having ties to the field of study covered by this CME program.

Source: Magid DJ, et al. Relationship between time of day, day of week, timeliness of reperfusion, and in-hospital mortality for patients with acute ST-segment elevation myocardial infarction. *JAMA* 2005;294:803-812.

NO ONE CAN DEBATE THAT REPERFUSION THERAPY WITH EITHER percutaneous coronary intervention (PCI) or fibrinolysis reduces mortality for patients with ST-segment elevation myocardial infarction (STEMI). Additionally, it has been clearly proven that the shorter the time interval from symptom onset to treatment, the greater the benefit, regardless of which therapy is chosen. The purpose of this study was to determine the impact of time of day and day of week on the ability to meet American College of Cardiology/ American Heart Association guidelines for door-to-drug or door-to-balloon time expectations (≤ 30 and 90 minutes, respectively).[1] A secondary goal of the study was to determine if identified time differences contributed to mortality.

This was a retrospective cohort study based on the National Registry of Myocardial Infarction (NRMI) database, which enrolled patients from 1999-2002. For this investigation, the authors examined 68,439 patients treated with fibrinolysis and 33,647 treated with PCI; patients were further divided into groups treated during *regular hours* (7 am-5 pm) or *off-hours* (5 pm-7 am on weekdays, and weekends). Door-to-drug and door-to-balloon times, as well as in-hospital mortality, were analyzed.

The findings (not surprisingly) demonstrated no significant differences in door-to-drug times between the two time periods (34.3 min off-hours vs 33.2 min regular hours, 95% CI 0.7-1.4, $p <$ 0.001), but marked differences in door-to-balloon times (116.1 min

off-hours vs 94.8 min regular hours (95% CI 20.5-22.2, $p < 0.001$). When teased out, the longer door-to-ballon time during off-hours almost entirely was caused by longer decision-to-cardiac-cath-lab-arrival times. The delay in door-to-balloon time during off-hours resulted in significantly higher adjusted in-hospital mortality for patients who presented during these times (OR 1.07, 95% CI 1.01-1.14, $p = 0.02$). Predictably, two-thirds of the patients presented during off-hours, and one third presented during regular hours. ❖

COMMENTARY

Do the terms *off-hours* and *regular hours* make anyone else bristle as much as they do me? I read these terms repeatedly in both the cardiology and (again, not surprisingly) the radiology literature. I know I am preaching to the converted, but patient care is a 24/7/365 proposition, and studies that show that I provide worse care at various times of the day or week that are completely out of my control drive me absolutely nuts. My chairman has a sage standard answer whenever we are presented with a new clinical pathway or patient care service in faculty meeting: it must work the same whether it is 1 pm on a normal weekday or 2 am on Christmas Eve. In general, those pathways/services that meet these criteria succeed, and those that don't fail.

A few important lessons can be taken from this large study. First, two-thirds of patients presenting with STEMI will come to the ED during the off-hours; therefore, whatever reperfusion strategy one pursues, this factor must be acknowledged. A facility that excels in door-to-balloon times during the 7 am-5 pm period but fails to approach the 90-minute mark during off-hours does a disservice to two-thirds of their STEMI patients.

Secondly, while we all acknowledge that time is muscle, this study outlines the potential in-hospital mortality consequence of this off-hour delay. If you come in during off-hours with a STEMI, your chance of surviving that hospitalization is diminished as compared with those patients who come in during regular hours.

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Please call **Martha Jo Dendinger,** Managing Editor, (404) 262-5514, or martha.dendinger@thomson.com.

Shock Teams Bring Favorable Effects to Patient Outcomes

ABSTRACT & COMMENTARY

*By **Nathan Shapiro, MD, MPH,***
Research Director, Harvard Medical School, Beth Israel Deaconess Medical Center, Department of Emergency Medicine, Boston, MA.
Dr. Shapiro has reported that he is on the speaker's bureaus of Eli Lilly, Edwards Life Sciences, and is a researcher for Biosite.

Source: Sebat F, et al. A multidisciplinary community hospital program for early and rapid resuscitation of shock in nontrauma patients. *Chest* 2005:127:1729-1743.

Cardiogenic shock mortality ranges from 50-80% and septic shock 40-60%. The hallmark of shock is inadequate tissue perfusion, a condition that should be addressed immediately and reversed promptly to prevent organ failure and death. The team approach with a mobilization of resources has been well described in specific disease processes such as cardiac arrest and trauma; the authors of this manuscript proposed that all forms of shock deserve the same level of attention.

The objective of this study was to "determine the effect of a community hospital-wide program enabling personnel to mobilize institutional resources for the treatment of patients with nontraumatic shock." Proto-

cols were developed that included screening criteria of hypotension (SBP < 90 mmHg or mean arterial pressure < 60 mmHg) plus one clinical criterion: respirations 20/min or more; oliguria less than 30 cc/hr; lactic acid more than 2.0 mmol/L or base excess -5 mmol/L or less; temperature 36° C or lower, cool extremities, or skin mottling; or anxiety, apathy, agitation, coma, or lethargy; or normotension plus three clinical criteria. If criteria still were present after a fluid bolus, then a shock alert was called. Personnel including nurses, pre-hospital providers, or physicians were empowered to call an alert.

Once a shock alert was called, a team of dedicated health care providers was mobilized, along with equipment and resources to perform an aggressive resuscitation. Patients were resuscitated using a combination of fluids and set goals to restore adequate perfusion. In some cases, these goals included invasive monitoring to meet a minimum SVO_2 and cardiac index, using vasopressors and dobutamine in coordination with other therapies (see appendix in manuscript for details). There were established emergency department (ED) and intensive care unit (ICU) protocols based on the providers' classification of shock (e.g., anaphylactic, hypovolemic, cardiogenic, or septic). The septic shock pathway included an antibiotic algorithm along with a protocol very similar to that published by Rivers and colleagues.[1] An ICU bed was reserved for a shock patient at all times to facilitate rapid transfer to the critical care setting.

During the one-year treatment period, there were 103 patients included in the protocol group; they were compared with 86 patients from the 30-month historical control period. The overall mortality was 40.7% for historical controls compared with 28.2% for the treatment group. The treatment group seemed to have benefited from earlier and more aggressive therapies. Notably, the protocol group had significant reduction in times to interventions: time to operating room/ICU admission 167 min vs 90 min ($p < 0.002$), 2 L fluid infused (232 min vs 105 minutes ($p < 0.0001$), intensivist arrival 120 min vs 50 min ($p < 0.002$). These markers represent a more aggressive and earlier resuscitation effort. ❖

COMMENTARY

This is a provocative article with some compelling findings. It promotes the concept that shock in general —not just traumatic— perhaps deserves increased attention and a plan to mobilize additional hospital resources. It shows that through multidisciplinary teamwork, planning, and good resource utilization, we can improve the timeliness and strength of a resuscitation to improve outcomes. The overarching idea is that critically ill patients need ICU level care, no matter where they are. In the study, 66% of the patients were admitted from the ED, and many of the alerts were called either by the ED or by pre-hospital personnel transporting a patient. This approach attempts to change the timeline of the needed therapies as well as the location where they are initiated and the providers (including ED physicians and nurses) who are involved.

Although the study clearly represents an ambitious collaborative effort, some unanswered questions remain regarding the study design. The fact that it took 30 months to select 86 patients in the historical control group, compared with 103 patients enrolled during 12 months in the treatment group, suggests that there may have been some selection bias. Although the authors credited a hospital capacity expansion, with increased attention to shock patients as part of a protocol, patients may have been more aggressively identified. They tried to account for this by doing an ICD-9 search, which is helpful, but still leaves some doubts. They also performed a regression analysis to control for important differences. All of these techniques improved the validity, but still, some questions remain, and a definitive answer may be obtained only from a randomized trial.

There are important lessons to be learned from the methods and results of this paper: 1) early identification and treatment of shock is critical; 2) multidisciplinary collaboration is important; 3) the use of coordinated protocols tailored to the needs and abilities of an individual institution is a key component to change practice. There is a clear role for the ED —the principal portal of entry to the hospital — to assist in coordinating these efforts.

Reference

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Esophageal Coins in Asymptomatic Children: Watchful Waiting or Early Retrieval?

ABSTRACT & COMMENTARY

*By **Raemma Paredes Luck, MD,***
Assistant Professor of Pediatric Emergency Medicine, Director of Continuing Medical Education, the International Health Program,

Temple University Children's Medical Center, Philadelphia.
Dr. Luck has reported no relationships with companies having ties to the field of study covered by this CME program.

Source: Waltzman ML, et al. A randomized clinical trial of the management of esophageal coins in children. *Pediatrics* 2005;116:614-619.

INGESTION OF FOREIGN BODIES IS A FREQUENT REASON for seeking emergency care in the pediatric population. Coins, in particular, are commonly encountered, and often are lodged in the esophagus. The management in symptomatic children is immediate removal of the coin, usually by endoscopy. The management of esophageal coin ingestion in the asymptomatic child has been controversial. Some practitioners recommend immediate removal while others advocate observation. This study by Waltzman was undertaken to answer these key issues: to compare the outcomes of two commonly used strategies and to determine which clinical factors can predict spontaneous passage of an esophageal coin in the asymptomatic pediatric patient.

Sixty patients younger than 21 years seen in the pediatric ED were admitted and randomized to equal two groups: 1) defined observation period (~ 16 hours, while remaining NPO) and repeat radiographs afterward; and 2) immediate endoscopic removal. Patients were excluded if they were symptomatic, had previous esophageal or tracheal surgery, or presented more than 24 hours after ingestion (or ingestion time could not be determined). Results showed that the rate of spontaneous passage beyond the esophageal sphincter was similar in both groups (around 27%), with all coins having passed by 19 hours. Nine of 30 patients in the immediate endoscopy group had a delay in their procedure of more than 2 hours. Repeat radiographs just before endoscopic removal showed that all coins had passed spontaneously to the stomach. There were no complications in either group. The authors found that coins located more distally in the esophagus (in the distal third), in males, and in older patients (66 vs 46 months) were more likely to pass spontaneously beyond the esophageal sphincter. ❖

COMMENTARY

It is interesting to note that in the immediate removal group, where there was a delay in endoscopy of more than 2 hours, approximately the same percentage of patients had spontaneous passage of the coins as in the observation group. One can argue that the percentage could be higher if the patients were observed longer.

The small sample size is probably this study's greatest limitation. However, the findings of the study are similar to other studies and provide further understanding on the natural history of coin ingestions. Previous researchers have shown that coins lodged for less than 24 hours, especially those more distally located and in children without a history of tracheosophageal surgery or pathology, are more likely to pass.[1-3] This led to the recommendation that asymptomatic children be observed for 12-24 hours before endoscopic removal.[1-4]

In an anonymous home-based survey of parents who had children with coin ingestions, Conners[5] found that the majority of parents (85%) managed these cases at home, often without calling their physicians or the poison center. Parents did not report any adverse complications. None of the children underwent endoscopic removal. This study suffered somewhat by the possibility of sample bias, in that only 35% of those surveyed responded. This finding was reinforced in a small study by Sharieff and colleagues who also found that asymptomatic patients sent home and followed-up the next day experienced no adverse outcomes.[1] Obviously, this strategy is more cost effective and convenient for patients.

A large prospective study comparing inpatient and home observation also may provide better answers to this commonly encountered condition.

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Special Feature

Management of the Difficult Airway

Michael Gibbs, MD, FACEP,
Chief, Department of Emergency Medicine, Maine Medical Center, Portland, ME.
Dr. Gibbs has reported no relationships with companies having ties to the field of study covered by this CME program.

Table 1. Difficult Airway Predictors

- Long upper incisors and/or prominent overbite
- Inter-incisor distance < 3 finger breaths
- Mandibular floor distance <3 finger breaths
- Thyromental distance < 2 finger breaths
- Mallampati score > 2
- Highly arched palate
- Large, thick tongue
- Short, thick neck
- Patient unable to touch chin to chest/extend head

Adapted from ASA Difficult Airway Taskforce. Practice guidelines for management of the difficult airway. Anesthesiology *2003; 98:1269-1277.*

Table 2. The LEMON Law

L =	**Look externally** (facial trauma, large incisors, beard or moustache, large tongue)
E =	**Evaluate** The 3-3-2 (incisor distance < 3 fingerbreadths, hyoid/mental distance <3 fingerbreadths, thyroid-to-mouth distance < 2 fingerbreadths)
M =	**Mallampati** (Mallampati score ≥3)
O =	**Obstruction** (presence of any condition that could cause an obstructed airway)
N =	**Neck mobility** (limited neck mobility)

Adapted from Reed MJ, et al. Can an airway assessment score predict difficulty at intubation in the emergency department? Emerg Med J *2005;2:99-109.*

Effective and timely management of the airway is our paramount responsibility. Once the decision to intubate has been made, the emergency physician faces a number of daunting questions — often with little information, no time, and zero tolerance for failure. *Does the patient have a difficult airway? Can this be predicted using physical examination? What is the best approach if standard techniques are unsuccessful?* To avoid disaster, these questions must be considered before the succinylcholine is pushed. This article provides a rational framework for doing that.

Step 1. Defining the Problem

In 2003 the American Society of Anesthesiology (ASA) Difficult Airway Task Force provided specific definitions for both the *difficult* and the *failed* airway that since have become widely accepted.[1] A *difficult airway* is broken down into three unique attributes that may exist alone or in combination: 1) difficult bag-valve-mask ventilation; 2) difficult laryngoscopy and intubation; and 3) difficult surgical airway. The *failed airway* is present when either of the following exists: 1) inability to ventilate or intubate the paralyzed patient; and 2) three failed intubation attempts by an experienced operator. Strict adherence to this level of definition specificity helps guide effective clinical decision-making; it raises the bar for performance, and it allows clinicians to communicate accurately.

Step 2. Understanding the Scope of the Problem

A recent review of the National Emergency Airway Registry (NEAR) described data from more than 6,000 airway cases at 31 university-affiliated EDs in the United States and Canada.[2] In this large, diverse cohort of adults and children, the first intubator was successful 90% of the time (95% CI 89-91%), including 83% (95% CI 82-84%) on the first attempt. The overall success rate for the first intubator was highest with rapid sequence intubation (RSI) (91%). Intubation success rates were directly proportional to years of training. Further analysis of this group demonstrates that the first technique failed in about 3% of cases.[3] Two-thirds of these first technique failures occurred when methods other than rapid sequence intubation were used (e.g., nasotracheal intubation and intubation with sedation alone). This fact provides additional compelling evidence that RSI should be the technique of choice in the majority of our patients. Rescue techniques included RSI (49%), cricothyroidotomy (21%), and a number of alternative airway techniques (30%).

These two studies illuminate several important principles. First, we are good at what we do. Second, we will fail. Third, because we don't fail very often, becoming an airway rescue expert is a challenge.

Step 3. Predicting the Problem

A number of clinical features have been proposed as potential markers of difficult intubation (*Table 1*). The accuracy of any single feature or combination of features for predicting the presence or absence of a difficult airway is not clear. An evidence-based literature review performed by the ASA Difficult Airway Taskforce lacked sufficient evidence to definitively evaluate the accuracy of the physical examination at predicting the presence of a difficult airway, although data suggested that "abnormal findings obtained during an airway exam may be associated with a difficult airway."[1] Shiga and colleagues performed a meta-analysis of 35 studies (n = 50, 760) relating to the accuracy of predictive tests for difficult intubation. Study selection criteria included: 1) prospective design, 2) at least one bedside diagnostic test performed, 3) data reported for true positives, true negatives, false positives, false negatives, 4) adequate blinding and consecutive enrollment. Once again, the features examined (Mallampati score of 3 or more, thyromental distance < 2 fingerbreadths, sternomental distance < 3 fingerbreadths, and mouth opening < 3 finger-

Table 3. Airway Rescue Devices

SUPRAGLOTTIC AIRWAY RESCUE DEVICES

- Combitube™
- Standard laryngeal mask airway™
- Intubating laryngeal mask airway™
- Lighted stylet™
- Intubating stylet

FIBEROPTIC INTUBATION

INFRAGLOTTIC AIRWAY RESCUE DEVICES

- Transtracheal jet ventilation
- Retrograde intubation
- Percutaneous cricothyroidotomy
- Open cricothyroidotomy

breadths) were associated with difficult airways, but had unacceptably low specificity as single markers. Adding several markers did increase specificity.[4]

More recently, a prospective single-center study evaluated the predictive value of the 10-point LEMON criteria (*Table 2*) in 156 consecutive ED patients.[5] For each patient, the score was compared with the widely accepted Cormack laryngoscopic view score (1 = full view, 4 = glottis not visualized). At intubation, 114 patients were classified as Cormack grade 1 (easy intubation), and 42 were classified as grade 2 or higher (difficult intubation). Patients in the difficult intubation group had significantly higher LEMON scores. Of the criteria used to calculate the score, only large incisors, inter-incisor distance less than 3 fingerbreadths, and thyromental distance less than 2 fingerbreadths were associated with difficult intubation. These data underscored the critical function of a focused and sophisticated airway evaluation whenever intubation is planned. However, it must be acknowledged that there is no perfect formula. I like the LEMON law, and use it routinely.

Step 4. Managing the Problem

Dealing with a perilously difficult or failed airway is perhaps the most treacherous set of circumstances facing the emergency physician. While a number of difficult airway algorithms have been published in the anesthesia literature,[1,6,7] these are not relevant to the ED. In addition, evidence-based, prospectively validated guidelines are lacking in our specialty. That being said, a number of fundamental principles can help guide decision-making. There are multiple alternative airway management devices available today, with more on the way (*Table 3*). Cricothyroidotomy is no longer the only option. Airway rescue devices can be classified logically as supraglottic (inserted above the glottis) and infraglottic (inserted below the glottis). With the exception of fiberoptic-guided devices, supraglottic rescue devices are blind—they are inserted without direct

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vision of the airway. As such, these generally are not recommended in patients with significant disruption of airway anatomy (e.g., hemorrhage, edema, tumor, or foreign body). Obviously this recommendation is not absolute; clinical judgment and experience are key. The element of time is critical. In patients who can be ventilated effectively, our menu of choice is potentially wide. Conversely, it should be recognized that the solution to a can-not-intubate-can-not-ventilate scenario must be an immediate definitive airway. Using the device you know will be the most effective method in your hands. It is our responsibility to become proficient with a number of devices that will allow each of us to manage the spectrum of airway disasters landing on our doorstep. ❖

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Physician CME Questions

27. A physical examination finding consistent with an anticipated difficult airway is a _____ distance less than 2 fingerbreadths.
a. mylohyoid
b. thyrohyoid
c. thyromental
d. intercalary

28. If your patient has _____, it is reasonable to anticipate a difficult airway.
a. large molars
b. a long neck
c. a long uvula
d. a thick tongue

29. Regarding radiographically proven esophageal coins in pediatric patients, which asymptomatic patient below merits emergent endoscopic removal?
a. Penny ingested 4 hours ago located in the distal esophagus
b. Nickel ingested 7 hours ago located in the mid-esophagus
c. Dime ingested 48 hours ago located in the distal esophagus
d. Quarter ingested within the hour, located in the proximal esophagus

30. A recent study examining the effect of time of day and day of the week on outcomes in the reperfusion of acute ST-segment elevation myocardial infarction found that:
a. there was no difference in mortality based on time of presentation.
b. off-hours presentation was associated with longer times to fibrinolysis.
c. off-hours presentation was associated with longer times to percutaneous coronary intervention.
d. mortality was highest in those patients receiving off-hours fibrinolysis followed by emergent percutaneous coronary intervention.

31. Establishing a shock team was shown to:
a. improve mortality.
b. improve procedural complication rates without affecting mortality.
c. decrease iatrogenic drug-drug interaction errors.
d. be beneficial to mortality only if the team was directed by traumatologists.

Answers: 27. c; 28.d.; 29. c; 30. c; 31.a

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.

ECG Review

Torsades Fabriquées?

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

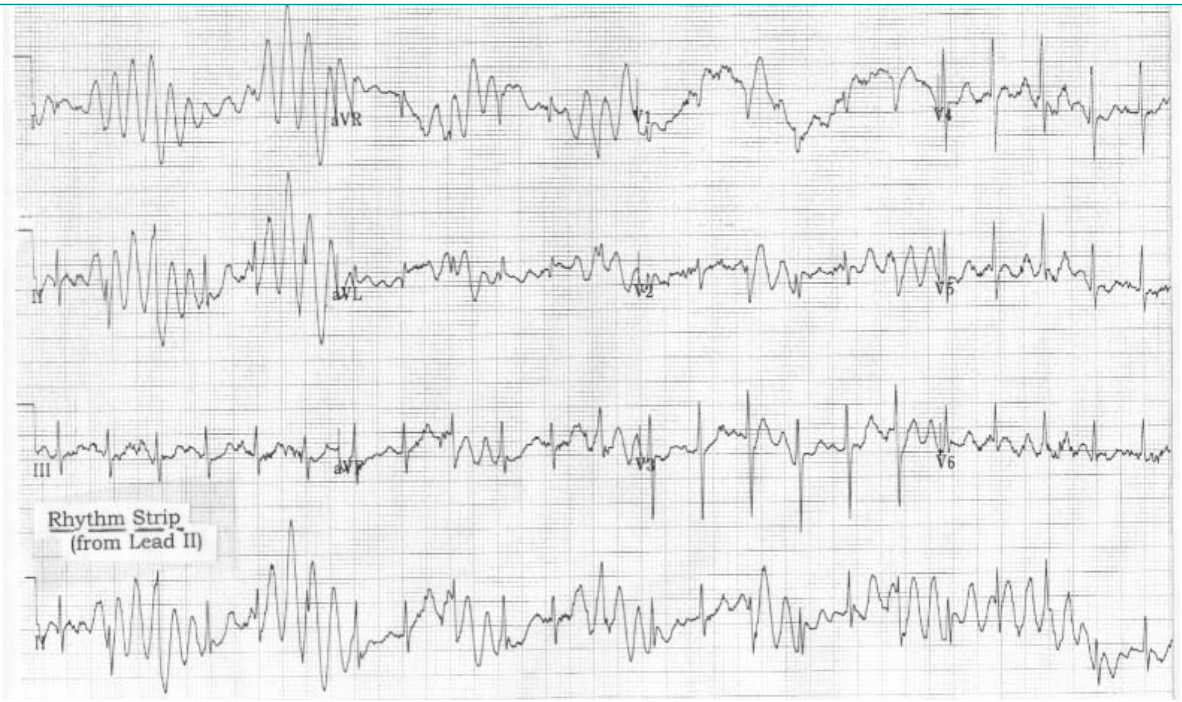

Figure. 12-lead ECG and accompanying rhythm strip recorded from an older man on multiple medications.

Clinical Scenario: The 12-lead electrocardiogram (ECG) and accompanying rhythm strip shown in the Figure were obtained from an older man who presented with acute dyspnea from pneumonia. He was on multiple medications, and was in moderate-to-severe respiratory distress at the time this ECG was recorded. Is the rhythm *Torsades de Pointes?*

Interpretation/Answer: At first glance, the tracing in the lead II rhythm strip clearly resembles Torsades, as there appears to be an intermittently large amplitude sinusoidal pattern with alternating polarity. However, this is not what is happening. The clue to the true etiology of the rhythm lies with careful surveillance of all leads on the 12-lead ECG. This reveals an underlying regular supraventricular (narrow QRS complex) tachycardia that is best seen in leads III, V3, V4, and V5. The relatively small baseline undulations in these leads clearly are identified as artifact. Armed with this knowledge, one can prove artifact as the cause of baseline distortion in the simultaneously recorded rhythm strip at the bottom of the Figure by beat-to-beat comparison with the rhythm in leads III and V3. Doing so should make it apparent that the underlying narrow QRS rhythm continues without interruption throughout the lead II rhythm strip. This underlying rhythm is sinus tachycardia, with intermittent sinusoidal-appearing artifact produced by this patient's respiratory distress.

Recognition of artifact is often a challenging endeavor. Perhaps the two most helpful clues toward facilitating recognition are: 1) Look first at the patient for correlation with the clinical setting (i.e., unresponsiveness consistent with malignant arrhythmia?—or "telltale activity" such as hand tremor, performance of CPR, seizure, or respiratory distress?); and 2) Look carefully for an underlying rhythm, which, if present continuously throughout the tracing despite distorting activity, proves artifact as the cause. ❖

In Future Issues: Atrial fibrillation management

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Hypothermia: A Cold Weather Hazard

Authors: **Ken Zafren, MD, FACEP**, EMS Medical Director, State of Alaska; Staff Emergency Physician, Alaska Native Medical Center, Anchorage, AK, Clinical Assistant Professor and Staff Physician, Division of Emergency Medicine, Stanford University Medical Center, Stanford, CA; and **Ingrid T. Lim, MD**, Senior Emergency Medicine Resident Physician, Stanford/Kaiser Emergency Medicine Residency, Stanford, CA.

Peer Reviewer: **Mary Jo Bowman, MD, FAAP,** Associate Professor of Clinical Pediatrics, Ohio State University; Attending Physician and Pediatric Emergency Medicine Fellowship Director, Columbus Children's Hospital, Columbus, OH.

Hypothermia causes 700-800 deaths per year in the United States alone.[1] Half of those who die are older than 65 years.[1] Many patients are hypothermic when they arrive in the emergency department (ED), but hypothermia may result from or be aggravated by failing to protect the patient from heat loss, administering room temperature or chilled IV fluids or blood, or using drugs that suppress shivering. Failing to recognize and treat hypothermia or allowing hypothermia to develop in a patient can lead to avoidable morbidity or mortality. Early recognition and aggressive management may prevent the life-threatening complications associated with this disease process.

This article will discuss the recognition and classification of hypothermia, how to prevent patients from becoming hypothermic, and options for treatment. The authors address some controversial areas, such as the best way to measure core temperature, the most effective methods for rewarming, and limitations to resuscitation. — The Editor

Definition and Classification

Humans are homeothermic organisms, functioning best within a relatively narrow range of core temperatures, between 36°C to 37.5°C (97°F to 99°F). Hypothermia is a decline in the core temperature to less than 35°C (< 95°F).[2] *Accidental hypothermia,* sometimes called primary hypothermia, results from exposure to cold environments without adequate protection. *Secondary hypothermia* is a complication of underlying disorders that decrease heat production, such as trauma, sepsis, and hypoendocrine states. *Iatrogenic hypothermia* can be caused by resuscitation with intravenous (IV) fluids at room temperature, by infusing chilled blood products, or by administering drugs that decrease shivering.

Epidemiology

Accidental hypothermia is the reported cause of death in approximately 700 to 800 people per year in the United States.[1]

Statement of Financial Disclosure

In order to reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, Dr. Dietrich (editor in chief) reports that she is the medical director for the Ohio Chapter of the American College of Emergency Physicians. Drs. Zafren and Lim (authors), Bowman (peer reviewer), Ms. Finnerty (nurse reviewer), and Ms. Neff (nurse reviewer) report no relationships with companies related to this field of study.

The elderly are particularly vulnerable to hypothermia, with 50% of all deaths occurring in patients older than 65 years.[1]

Other groups at risk include the homeless, chemically dependent, and mentally ill individuals, outdoor workers, trauma victims, and children. Although most cases occur during winter months, hypothermia can occur throughout the year, even in mild climates, and in individuals without outdoor exposure.[2] Elderly and ill patients may become hypothermic, even in well-heated houses.

Etiology

The ability to adapt to cold varies among individuals. Conditions that make some patients more susceptible to cold exposure include those associated with impaired heat production, increased heat loss, or impaired thermoregulation. Endocrine disorders, hypoglycemia, malnutrition, and extremes of age are associated with decreased ability to increase heat production by shivering.[3] Impaired thermoregulation occurs in both peripheral and central nervous system dysfunction. Individuals with peripheral neuropathies or spinal-cord injuries may be unaware of environmental conditions or may be unable to compensate by mechanisms such as vasoconstriction. Stroke, trauma, neoplasms, neurodegenerative disorders, and drugs can act centrally to decrease hypothalamic function. In addition, sepsis, pancreatitis, carcinomatosis, uremia, vascular insufficiency, and multisystem trauma also are associated with hypothermia.[4] Numerous drugs, most commonly alcohol, impair temperature regulation. Alcohol causes peripheral vasodilation, leading to increased heat loss. Alcohol and other drugs also may interfere with the ability to make appropriate behavioral choices to seek shelter or wear protective clothing. Some medications (e.g., benzodiazepines, meperidine, and chlorpromazine) suppress shivering, and predispose to iatrogenic hypothermia.[5,6]

Physiology

Body temperature is determined by the balance between heat production and heat loss. Increased heat production can be achieved by voluntary activity, by increased metabolism, by shivering, and is mediated by the thyroid and adrenal glands.

Heat is lost from the body in four ways: radiation, conduction, convection, and evaporation. *Radiation* is the direct exchange of heat (by radiant energy) between two objects separated in space. It depends upon surface area and the temperature difference between the objects. *Conduction* is heat transfer between two objects that are in direct contact. *Convection* is heat transfer by a moving fluid. This can occur when the body heats air or water surrounding its surface by conduction and movement (wind or water currents) and displaces the warmed fluid. *Evaporation* is heat lost by vaporizing a liquid. A large amount of heat can be lost from moist skin surfaces. The lungs also lose heat through evaporation (insensible respiratory loss).

The relative amount of heat lost by each mechanism depends upon conditions. Wet clothing, wet skin, and wind are factors that can dramatically increase heat loss. Wet clothing can increase the rate of heat loss by a factor of five, while immersion in water causes an increase of 25 to 30-fold.[7] Children are more susceptible to hypothermia because they have a large body surface area relative to body mass.

Peripheral, especially cutaneous, vasoconstriction reduces heat loss. Shivering generates heat, increasing metabolism up to five times the baseline rate.[8]

However, humans are tropically adapted animals with limited ability to maintain core temperature in the face of environmental stresses encountered under more temperate conditions. The thermoneutral temperature of humans in still air is 28°C (82°F).[7] Doubling the metabolic rate decreases the thermoneutral temperature only to 20°C (68°F). The ability to create a warm microclimate by the use of clothes and shelter is critical to human survival in temperate and polar climates.

Pathophysiology

Hypothermia affects multiple organ systems.

Cardiovascular. Initially, the patient's heart rate, blood pressure, and cardiac output increase because of increased catecholamine production with peripheral vasoconstriction. After further cooling, there is an almost linear decrease in these parameters. At core temperatures below 28°C, bradycardia is a direct consequence of slowed conduction and decreased spontaneous depolarization of pacemaker cells.[9] The myocardium becomes irritable, with a lower threshold for ventricular fibrillation.

Circulating blood volume markedly decreases to as little as one-third of normal, even in moderate hypothermia.[10] As a consequence, even relatively modest infusions of fluids can have a significant effect on core temperature.

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Editorial Group Head: Glen Harris
Managing Editor: Martha Jo Dendinger

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Central Nervous System (CNS). Hypothermia results in decreased cerebral blood flow, with decreased neuronal metabolism. This cerebroprotective response explains why the brain can endure ischemic injury in the setting of hypothermia. Clinically, there is a progressive decline in level of consciousness, manifesting as lack of fine motor coordination and clumsiness, followed by confusion, dysarthria, and impaired judgment and memory.

Renal. With mild hypothermia, peripheral vasoconstriction results in increased renal blood flow and a cold-induced diuresis, which depletes intravascular volume. As the temperature drops, the kidneys lose the ability to reabsorb water as the tubules become insensitive to antidiuretic hormone (ADH). In moderate hypothermia, the glomerular filtration rate declines as cardiac output falls and vascular tone increases. At lower temperatures, tubular capacity for hydrogen ion secretion is reduced, resulting in acidosis.[11]

Pulmonary. In mild hypothermia, initial tachypnea, is followed by a reduction in minute ventilation and reduced oxygen consumption.[6] Bronchospasm and bronchorrhea develop. As the temperature drops into the range of moderate hypothermia, ciliary function is impaired, which can lead to aspiration and pneumonia. In severe hypothermia, progressive hypoventilation and apnea may develop. Pulmonary edema is a rare complication.

Oxygen Delivery. Falling temperatures cause a left shift of the oxyhemoglobin dissociation curve, resulting in impaired oxygen delivery and tissue hypoxia. However, this left shift is balanced by a rightward shift caused by acidosis that occurs with shivering and anaerobic metabolism. The problem of impaired oxygen delivery to the tissue is mitigated by the decreased oxygen demand at lower temperatures.

Hematologic. Hypothermia affects the erythrocytes, leukocytes, platelets, and clotting factors. The hematocrit increases by about 2% for every one degree Celsius decline in temperature.[2]

A normal hematocrit in a moderately or severely hypothermic patient should suggest anemia or blood loss. Leukocyte depletion can occur, along with impaired neutrophil migration and ineffective bacterial phagocytosis.[12]

Both decreased platelet numbers from sequestration in the liver and spleen and decreased platelet function occur with decreased production of thromboxane B_2.[13] Hypothermia can impair both the endothelial synthesis of prostacyclin (PGI_2) and its inhibitory action on platelet aggregation, promoting platelet activation and thrombosis.[11] Elevated cryofibrinogen levels raise the blood viscosity.

Cold also inhibits the enzymatic reactions of the coagulation cascade. The prothrombin (PT) and partial thromboplastin time (PTT) can be deceptively normal if measured at 37°C, but can be significantly increased if measured at lower temperatures. Instead of replacing clotting factors, rewarming is the appropriate treatment for this type of coagulopathy.[14] Disseminated intravascular coagulation can also occur.

Gastrointestinal. Below 34°C, intestinal motility decreases. Autopsy results can show shallow gastric ulcers known as Wischnevski's ulcers.[15] Hepatic impairment also may develop, likely because of decreased cardiac output. The reduced clearance of lactic acid also contributes to acidemia. Pancreatitis also frequently occurs in hypothermia. It is found at autopsy in 20-30% of cases.[16] Although the mechanism is not well understood, it may be that thrombosis in the microcirculation causes ischemia and perilobular necrosis in the pancreas.[11]

Endocrine. Total body metabolism decreases with increasing hypothermia, as measured by a fall in oxygen consumption: 6% for every degree Celsius fall in temperature.[17] The basal metabolic rate is reduced to 50% at a core temperature of 28°C.

Pharmacology. Drugs are generally ineffective below 30°C (86°F)**.**[6] Not only are the enzymatic systems on which they work affected, but protein binding increases. Because of decreased circulation to the skin, muscles, and gastrointestinal tract, only intravenous drugs are reliably absorbed. If they are given to a patient with a core temperature below 30°C (86°F), they are likely to remain inactive until the patent is rewarmed, at which point they become active when their effects may no longer be desirable.

Afterdrop. No discussion of hypothermia would be complete without mention of the dreaded afterdrop. This phenomenon refers to the continuing decrease in core temperature after removal from a cold environment, even when active rewarming is in progress. Although there was once controversy about the relative contribution of countercurrent heat exchange and increased flow of cool peripheral blood as vasoconstriction decreased, it is now clear that afterdrop is primarily a mass effect of continuing conduction of heat from the warmer core to the cooler peripheral tissues.

Several studies have measured peripheral blood flow during external rewarming and have noted minimal flow through skin and muscle while the afterdrop was occurring. Afterdrop also has been observed in fibrillating patients without any circulation.[18] Afterdrop occurs because heat flows from an area of higher temperature to one of lower temperature. The temperature of the skin is usually 15-25°C (59-77°F), cooler than the temperature of the core of a hypothermic patient.[18] No method of warming can transfer heat from the shell to the core against a temperature gradient. Until the temperature of the shell is raised to the temperature of the core, heat will continue to flow from the core to the periphery, and core temperature will fall despite application of external heat. Afterdrop cannot be prevented, but can be minimized. Even with optimal internal warming methods, afterdrop can be as much as 0.5° C (1° F).

Clinical Features

Hypothermia is classified as *mild* from 32°C to 35°C (90-95°F), *moderate* from 28° to 32°C (82-90°F), and *severe* less than 28°C (< 82°F). (*See Table 1.*) The temperature ranges serve mainly as guidelines; there is a large variability in individual response to hypothermia. This classification system is of less use in multisystem trauma patients, in whom mortality rates approach 100% when core temperatures fall below 32°C (90°F).[19]

Mildly hypothermic patients are usually alert and shivering vigorously, but become progressively confused as they cool. Early features of hypothermia include impaired judgment and progressive loss of both fine and gross motor control. Initially

respiratory rate, heart rate, and blood pressure are increased, but with further cooling, they are depressed.

Moderately hypothermic patients are confused and, as they cool, lose the ability to shiver. Although the average temperature at which shivering stops and consciousness is lost is said to be 30°C (86°F); some patients may be able to shiver and to carry on a conversation with a core temperature as low as 28°C (82°F).[6] Respiratory rate, heart rate, and blood pressure continue to decrease in a linear fashion with further cooling.

Table 1. Classification of Level of Hypothermia

CLASSIFICATION	CORE TEMP	PATIENT'S ABILITY TO REWARM WITHOUT EXTERNAL HEAT SOURCE	CLINICAL PRESENTATION	
Normal	Above 95°F (35°C)		Cold sensation, shivering	
Mild	95-90° F (35-32°C)	Good	Physical impairment • Fine motor • Gross motor	Mental impairment • Complex • Simple
Moderate	90-82° F (32-28° C)	Limited	Below 86° F (30°C) shivering stops; loss of consciousness	
Severe	Below 82°F (28° C)	Unable	• Rigidity • Vital signs reduced or absent • Severe risk of mechanically stimulated ventricular fibrillation (VF) (rough handling)	
	Below 77°F	Unable	• Spontaneous ventricular fibrillation (VF)	

Adapted from Section of Community Health and EMS. *Cold Injuries Guidelines.* Alaska Multilevel 2003 Version.

Severely hypothermic patients are unconscious and may feel rigid. Vital signs may be difficult to obtain or may be absent. The presentation of hypothermia may be straightforward or easily overlooked as in an unconscious patient found indoors. The key to diagnosis is to measure the core temperature. Hypothermia frequently presents as a complication of another condition (e.g., trauma or intoxication with alcohol or other drugs). Iatrogenic hypothermia may occur in the ED or radiology suite secondary to failure to prevent heat loss, and therefore, may occur in normothermic patients after a period of time in the ED. Hypothermia also may occur in the settings of heat stroke cooling or emergency delivery.

Diagnosis

Hypothermia is diagnosed by measuring the core temperature, which is the temperature of the internal organs, especially the heart. The diagnosis and treatment of hypothermia depend upon accurate measurement and tracking of core temperature. Esophageal temperatures provide the most accurate measure of core temperature with the probe placed by the same technique used for insertion of nasogastric or orogastric tubes. The technique is now standard in operating rooms, and, contrary to earlier beliefs, readings are not affected by inhalation of heated oxygen in intubated patients.

Epitympanic temperature is measured by a special probe in proximity to the tympanic membrane. Although not readily available in the United States, it provides a reasonable, noninvasive measure of core temperature through measurement of the temperature of blood in the carotid artery.[20] This is a different device than the widely used infrared tympanic temperature devices, which are unreliable in hypothermia.

Rectal temperature is a poor index of core temperature, especially during rewarming. Rectal temperature changes lag behind changes in core temperature, with rectal temperatures still falling while the core temperature is increasing. Bladder catheter temperatures have the same limitations as rectal temperatures. Oral temperatures are affected by changes in ventilation and are useful only in excluding hypothermia. If using rectal or oral thermometers, remember that standard thermometers only read to a minimum of 34°C (93°F); special cold-reading thermometers should be used if esophageal or epitympanic probes are not available.

Differential Diagnosis

Although the diagnosis of hypothermia is based upon core body temperature, conditions other than cold exposure can cause hypothermia and should be considered as either the primary cause or as contributing factors.

Endocrine causes of hypothermia include hypothyroidism, adrenal and pituitary insufficiency, and hypoglycemia. Initial evaluation in hypothermic patients may include bedside glucose testing, empiric administration of steroids (use dexamethasone to preserve the ability to do later stimulation testing), and other laboratory tests as indicated.

Central nervous system problems that can cause or contribute to hypothermia include trauma, stroke, tumor, and other causes of hypothalamic dysfunction (e.g., Wernicke's syndrome). Consider administration of thiamine (100 mg IV) and computed tomography (CT) imaging of the head.

Acute spinal cord transection causes vasodilation below the level of the lesion as well as paralysis. Patients who sustain a spinal cord injury in a cold environment are extremely susceptible to rapid cooling because of the inability to vasoconstrict peripheral vessels and the inability to shiver.

Alcohol and other drugs interfere with judgment and can promote sleep or induce coma. Exposure to carbon monoxide also can cause coma. Specific toxidromes may be present. The empiric use of naloxone (0.4 – 2 mg IV or more) may be diagnostic.

Myocardial infarction may be present in a hypothermia patient. Chest pain, dyspnea, lightheadedness, syncope, or other nonspecific symptoms may provide clues. The ECG and cardiac markers are the keys to diagnosis.

Peritonitis from abdominal injuries or other conditions (e.g., appendicitis) may be masked or may be simulated by a rigid abdomen in hypothermia. Other diagnostic methods (e.g., laboratory studies and CT imaging) may be necessary to make or exclude these diagnoses.

Hypothermia may be the consequence of infection, including meningitis, encephalitis, pneumonia, and sepsis. Especially in infants and immunocompromised patients, hypothermia may be a sign of overwhelming infection. Searching for the source of infection may include chest x-ray, urinalysis, lumbar puncture, and blood cultures.

Management

Assessment and Initial Care in the Prehospital Phase. Safety of the rescuers is the first priority in prehospital care; they should take care not to become victims. The patient should be removed from the cold environment as soon as possible, but moderate and severely hypothermic patients should be removed very gently because the ventricular fibrillation threshold is lowered. Avoid unnecessary movement of the extremities to avoid flow of cooled blood from the periphery to the core. Gently remove wet clothing by cutting it off. Cover the patient, including the head and neck, to avoid heat loss. Heat the inside of ambulances to 28°C (82°F).

Patients who have an altered level of consciousness, who are not shivering vigorously or at all, and who have depressed vital signs are likely to be hypothermic. Measure the core temperature as soon as possible, preferably with an esophageal probe thermometer. Administer heated humidified oxygen, and intubate patients whose depressed level of consciousness presents an aspiration risk. If possible, ventilate moderate or severely hypothermic patients with heated humidified oxygen, which may decrease the fibrillation threshold. Because metabolism is decreased, decrease the ventilatory rate, to about one-half the normal rate, to avoid hyperventilating the patient.

Most patients who are hypothermic are volume depleted and require intravenous fluids. Fluids should be administered to replace estimated deficits rather than for rewarming, and heated when possible to 40-42°C (104-108°F). Give fluid as boluses and titrate to effect, rather than giving continuous infusions.

Hypothermic patients without vital signs or other signs of life present a great challenge. Patients suffering from severe hypothermia have been resuscitated with full neurologic recovery. Dependent lividity, apparent rigor mortis, and fixed, dilated pupils are not contraindications to resuscitation. At 23°C (73°F) all re-flexes are absent, including corneal and oculocephalic reflexes. At 19°C (66°F) electroencephalographic (EEG) activity is absent.[6]

However, in contrast to the adage that "nobody is dead until they are warm and dead," some people are cold and dead. Unfortunately, patients who have been totally submerged in cold water for significantly more than 1 hour, have a core temperature below 10°C (50°F), have obvious lethal injuries (e.g., decapitation, total exsanguination, or tissue decomposition), are frozen (e.g, ice formation in the airway), or have a chest wall so stiff that compressions are impossible will not survive. The lowest recorded temperature for a survivor of accidental hypothermia is 13.7°C.[21] Studies have demonstrated that most survivals after prolonged submersion involved water colder than 5°C (41°F).[22] Submersion for up to 88 minutes in water or cardiopulmonary resuscitation (CPR) for many hours is still compatible with intact survival.[23-25]

Field treatment may be limited by an inability to perform cardiac monitoring. Patients may have vital signs and a perfusing cardiac rhythm, which are undetectable without monitoring. Because hypothermia decreases the metabolic requirements of organs, especially the brain, these patients may be stable – in a so-called metabolic ice box. Starting chest compressions could precipitate ventricular tachycardia (VT) or ventricular fibrillation (VF). An automated electronic defibrillator may be used for initial evaluation of the cardiac rhythm. The signal to shock means that the patient is in VT or VF. Palpation of a weak, bradycardic pulse in a cold, stiff, hypothermic patient, may be difficult. Treat any organized cardiac rhythm as a sign of life, and do not start chest compressions. It is not worth the risk of converting a perfusing rhythm to VT or VF with chest compressions, although the evidence for this is only circumstantial.[26,27] Chest compressions typically are more difficult and require more force. The cardiac output achieved during CPR will be only 50% of that achieved during normothermic CPR.[18] Defibrillation should be attempted at standard energy settings (three attempts) for VF or pulseless VT, but realize that attempts likely will be unsuccessful at core temperatures less than 30-32°C (86-90°F). Defer further attempts until the patient has been rewarmed above 32°C (90°F) according to Advanced Cardiac Life Support (ACLS) guidelines. Do not give drugs, including anti-arrhythmics. Drugs are generally ineffective below 32°C (90°F). However, give naloxone (0.4 – 2 mg IV or more), thiamine (100 mg IV) and glucose (D50W-1 ampoule) empirically.

More complete guidelines for prehospital care, including care involving prolonged transport times and field rewarming can be found in the State of Alaska Cold Injuries Guidelines.[28]

Emergency Department Care

Once the patient arrives in the ED, carefully re-evaluate airway, breathing, and circulation and continue to avoid movement or manipulation of the patient that might precipitate VF. Because cold skin is injured easily, avoid direct application of hot objects or excessive pressure (e.g., uninsulated hot water bottles or BP cuffs). Continue forced air rewarming, and cover the patient's head and neck with warm blankets.

Ventilation often is compromised by associated chest rigidity and decreased diaphragmatic movement. Consider fiberoptic intubation if there is severe jaw rigidity. The oral route is preferred because of risk of coagulopathic bleeding with nasotracheal intubation. However, muscular rigidity may complicate orotracheal intubation. Do not use neuromuscular blockers, which are not effective at temperatures below 30°C (86°F).[29] Intubation does not appear to induce VF.[30] Patients may require frequent tracheal suctioning in the setting of cold-induced bronchorrhea. Pulse oximetry is unlikely to be accurate or even possible in the setting of hypothermia and poor perfusion.

Circulatory status is often difficult to assess in hypothermic patients due to the extreme peripheral vasoconstriction. A Doppler ultrasound may be necessary to detect a pulse. Peripheral venous access is preferred but may be difficult. Pulmonary artery catheters and central venous catheters in the subclavian or jugular veins should be avoided because of the potential for causing VT or VF. If initial defibrillation was unsuccessful, resume CPR, begin rewarming, and attempt defibrillation again only when the core temperature reaches 32°C (90°F). Most hypothermia-induced dysrhythmias convert spontaneously with rewarming. Atrial fibrillation is common but is usually rate-controlled because of slowed ventricular response and resolves spontaneously with rewarming. Antiarrhythmic and vasoactive drugs in the moderate to severely hypothermic patient are usually ineffective at core temperatures less than 30°C,[31] and once the core temperature is above 30°C (86°F), only the lowest effective dose should be used because of potential toxicity with altered metabolism. Bretylium tosylate has been effective in animal studies.[32,33] The optimal dose and infusion rate of bretylium and other vasoactive drugs are unknown and indications are unclear for patients with hypothermia.

An almost universal requirement in hypothermia is aggressive volume resuscitation. The standard fluid is 5% dextrose in normal saline (D5NS). Lactated Ringer's should not be used because of decreased ability of the liver to metabolize lactate. Intravenous fluids should be heated to 40-42°C (104-108°F). If a commercial fluid warmer is not available, heat intravenous fluids in a microwave oven and remember to shake the solution after heating to eliminate hot spots. Because the circulating blood volume is decreased by as much as two-thirds in moderate hypothermia,[10] this fluid can provide significant rewarming. In cases of moderate or severe hypothermia, place a nasogastric or orogastric tube to relieve gastric distention and decrease the risk of aspiration. A urinary catheter is useful in monitoring urine output and volume resuscitation efforts.

Do not use medications prior to rewarming. Medications, if administered in mildly hypothermic patients, should be given only intravenously; oral, rectal, and intramuscular routes are unreliable. Phenothiazines, meperidine (Demerol), buspirone, and other drugs reduce or abolish the ability to shiver.

Physical Examination

Once initial stabilization has been accomplished, a thorough secondary survey should be performed to identify conditions that may have led to or resulted from hypothermia. Patients who are mildly hypothermic should be alert; if they have an altered level of consciousness, search for a cause other than hypothermia.

Vital signs may be helpful for identifying other problems; respiratory rates that are too fast or too slow for a given core temperature suggest metabolic acidosis or a central nervous system lesion; heart rate should be about 50% of normal at a core temperature of 28°C (86°F),[6] if faster than expected, look for other causes (e.g., hypovolemia, hypoglycemia, or drug ingestions).

Diagnostic Testing

Electrocardiogram (ECG). Impaired cardiac electrical activity can be seen on ECG tracings. Repolarization abnormalities, which appear as distinctive Osborn or J waves, are most prominent in the precordial leads V2-V5 and occur in one-third of hypothermic patients, at temperatures below 32°C (90°F).[6] Although the amplitude of the J wave correlates with the degree of hypothermia, J waves are not pathognomonic of hypothermia and may also be seen with subarachnoid hemorrhage, other cerebral injuries, and with myocardial ischemia. Other ECG changes include widened QRS complexes, with ST elevation or depression, and T-wave inversions. QT prolongation and AV block may develop days after normal temperature has been restored.[11]

Arterial Blood Gases. Arterial blood gas measurement may be used to monitor patients. Uncorrected blood gas samples are heated routinely to 37°C prior to analysis. Corrected values, derived from factors applied to account for temperature effects on gas pressure solubility and hemoglobin dissociation curves, should not be used because there will be a spurious increase in pH levels and a decrease in carbon dioxide tension. The arterial blood gas measurement commonly shows a combined metabolic acidosis (decreased tissue perfusion with accumulation of lactate) and respiratory acidosis (decreased respiratory effort and CNS depression).

Hematologic Testing. Because hypothermia has an associated expected rise in hematocrit, a normal or low value suggests acute hemorrhage or preexisting anemia. White blood cell and platelet counts are artificially lowered by sequestration. Expect low counts to normalize with rewarming.

Electrolytes. Monitor serum electrolyte levels during rewarming. Potassium levels do not vary with core temperature, but hypothermia increases the toxicity of potassium. Both hypokalemia and hyperkalemia may complicate the course of resuscitation. Hyperkalemia is a marker of acidosis and cell death. In hypothermic patients who have underlying trauma or asphyxia, potassium levels greater than 10 mEq/L predict failure of attempted resuscitation.[34] The ECG is not helpful because potassium-induced ECG changes are less evident in hypothermia.

Glucose. Blood glucose levels may be elevated in mild hypothermia because of catecholamine-induced glycogenolysis and in moderate to severe hypothermia because endogenous insulin has decreased activity below 30°C (86°F). Subacute and chronic hypothermia cause glycogen depletion with subsequent hypoglycemia. Hypoglycemia is a common cause of hypothermia in the hospitalized patient.[29]

Table 2. Rewarming Methods

Method	Notes	Patient Type
ACTIVE EXTERNAL REWARMING METHODS		
Forced air warming	High flow of warm air across the skin	All
Plumbed (water-filled) blankets	Use only if forced air is not available.	All
Other external devices (e.g., the Charcoal Heatpac [the Norwegian heater])	This device is designed primarily for field use.	All
Warmed blankets	Blankets from a warmer	All
Warm (tub) bath	Water should be up to 100°F (37°C).	Mild hypothermia
AVA rewarming (arm/leg)	The lower arms and hands (distal to the elbow) and the lower legs and feet (distal to the knees) are immersed in water between 107-112°F (42-45°C). This opens the arteriovenous anastomoses (AVA).	Mild hypothermia
ACTIVE CORE REWARMING		
Warm IVs	104°-108°F (40°-42°C)	All
Heated, humidified oxygen ventilation	104°-108°F (40°-42°C)	All
Peritoneal lavage	104°-108°F (40°-42°C)*	Preferred invasive method for moderate to severe hypothermia
Chest lavage via chest tubes ‡	104°-108°F (40°-42°C)*	Used for moderate-to-severe hypothermia; less effective than peritoneal lavage
Continous arteriovenous rewarming (CAVR)	Blood from an arterial catheter runs through a warmer and returns to the body via a venous catheter.	Severe hypothermia with SBP > 60
Extracorporeal circulation (cardiac bypass)		Severe hypothermia with SBP < 60
NOT RECOMMENDED		
Chemical heat packs, gastric lavage, rectal lavage		

* For further details about techniques, solutions, and rates, see Danzl DF. Accidental Hypothermia. In: Auerbach PS, ed. Wilderness Medicine. 4th ed. St. Louis: Mosby; 2001:177.
‡ Chest tube lavage should be used only if placement of a chest tube has been performed already for another indication.
Adapted from Alaska Cold Injuries Guidelines.

Coagulation Studies. Hypothermia causes coagulopathy, but the interpretation of the coagulation profile is difficult. PT and PTT may be deceptively normal unless measured at the patient's temperature instead of being warmed to 37°C. Instead of replacing clotting factors, rewarming is the appropriate treatment for this type of coagulopathy. If rewarming is successful, correct any residual coagulopathy. Disseminated intravascular coagulation (DIC) may occur after rewarming.

Amylase. Hyperamylasemia can be a result of preexisting pancreatitis or of pancreatitis induced by hypothermia. It correlates with the severity of hypothermia and mortality.[29]

Other Tests. If the cause of hypothermia is not known, consider adding thyroid function tests, toxicology screens, ethanol level and serum amylase tests, as well as urinalysis and cultures to rule out infection.

Radiologic Testing. The indications for x-rays are similar to those for normothermic patients.

Rewarming Techniques

The choice of rewarming techniques depends upon the severity of hypothermia, the hemodynamic status of the patient, institutional and physician preference, and the availability of specialized personnel and equipment. (*See Table 2.)*

Passive External Rewarming. Passive external rewarming (PER) requires the patient to produce heat spontaneously, making it useful only for the mildest cases of hypothermia. The patient should have all wet clothing removed, be placed in a warm, dry environment and covered (remember to cover the head and neck) with an insulating material to prevent further heat loss. Rewarming rates with PER in mild hypothermia vary between

0.5-2.0°C per hour.[2]

Active Rewarming. Provide active rewarming — the addition of exogenous heat — for all patients who cannot generate sufficient heat for PER. Indications for active rewarming include cardiovascular instability, poikilothermia (core temperature < 32°C [< 90°F]), inadequate rewarming with other methods, endocrinologic insufficiency, and traumatic peripheral vasodilation due to spinal cord injury.[29]

Active External Rewarming. Active external rewarming (AER) involves transfer of exogenous heat to the patient via the skin. These methods, which include heating blankets, forced heated air systems, or heating lamps, are widely used because they are familiar, inexpensive, and readily available. They quickly rewarm hypothermic patients and often are all that is necessary in mild or even moderate hypothermia.

Theoretically, if the periphery is rewarmed before the core, cold, acidemic blood returns to the heart. In practice, this is not a problem with forced-air rewarming.[35] A number of devices in which heated air is blown into a semiclosed cover over the patient's trunk and extremities are available and forced air rewarms more quickly than blankets alone (2.4°C/h compared with 1.4°C/h).[36,37] Newer devices are small enough to be used in ground or air ambulances. A special portable rigid forced air cover (PORIFAC) has been developed for prehospital use.[38]

Another active external rewarming technique makes use of arteriovenous anastamoses (AVA). These anastamoses, which are found in the forearm and calf vessels, normally close during vasoconstriction in hypothermia. In AVA rewarming, the anastamoses are opened by heat or by negative pressure and heat is applied. In one version, the mildly or moderately hypothermic patient sits on a chair with arms to the elbows and legs to the knees immersed in 44-45° C (111-113°F) water.[39] In a second version, one arm is placed in a negative pressure sleeve, and hot water is circulated in tubing inside the sleeve. Early research gave promising results,[40] but subsequent studies have failed to demonstrate its usefulness,[41] and at least one subject in an unpublished study sustained a burn.[42]

Active Core Rewarming. Active core rewarming (ACR) techniques are the most effective and rapid methods of raising the core temperature; numerous alternatives are available and range from minimally invasive to extracorporeal warming.[6,42]

Humidified oxygen heated to 40-45°C (104-113°F) should be used in all hypothermia resuscitations. Theoretically, airway rewarming has little influence on outcome; it can cause an elevation in core temperature of no more than 1-1.5°C (2-3°F) per hour.[30] However, the small contribution to temperature rise is synergistic with other modalities. Inhalation therapy helps to maintain core temperature by preventing heat loss during expiration, which may be equivalent to approximately 10% of metabolic heat production. It may warm the hypothalamus and reverse the cold-induced depression of respiratory centers, improving the level of consciousness.

A variety of body cavities can be heated by irrigation with warmed fluids. Therapeutic peritoneal lavage seems to be the most effective method. To use the single catheter technique, infuse 2 L of dialysate or other isotonic fluid (e.g., normal saline) at 44°C (111°F) every 20-30 minutes, and then suction as much as possible before the next cycle. Alternatively, place two catheters through the midline into the right and left abdominal gutters and use them as afferent and efferent exchange lines to exchange up to 6 L/h.[43] Advantages of peritoneal lavage include hepatic rewarming, renal support when dialysate is used, and the ability to continue cardiopulmonary resuscitation. Rewarming rates average 1-3 degrees/hr.[29]

Although less effective than peritoneal lavage, closed thoracic lavage may be useful if the patient has another indication for tube thoracostomy (e.g., pneumothorax or hemothorax). Two large thoracostomy tubes should be placed: one in the anterior second or third intercostal space in the midclavicular line and a second tube in the posterior axillary line in the fifth or sixth intercostal space. Infuse sterile normal saline heated to 40-42°C (104-108°F) through the anterior tube and allow it to drain passively into the posterior tube. The left hemithorax is preferred because it exposes the bulk of the myocardium and the great vessels to rewarming. Although rarely employed, mediastinal irrigation and myocardial lavage could be considered in patients who have severe hypothermia and no spontaneous perfusion, but require expertise in thoracotomy.[44]

Irrigation of the stomach, bladder, or colon is not very effective, because the surface area available for heat transfer is minimal. Gastric lavage has the additional risk of aspiration and cannot be continued during chest compressions.

Extracorporeal Methods. Extracorporeal methods include hemodialysis, arteriovenous rewarming, venovenous rewarming, and cardiopulmonary bypass (CPB). Hemodialysis utilizes a two-way flow catheter with percutaneous cannulation of a single vessel. The femoral vein is preferable because of the risk of myocardial irritation with the guide wire if other routes are used. Consider this technique in hemodynamically stable patients, with associated severe renal dysfunction or intoxication with dialyzable substances. Newer techniques of arteriovenous and venovenous rewarming, utilize counter-current fluid rewarming. Arteriovenous techniques depend upon the patient's blood pressure, which must be at least 60 mm Hg systolic.[45] Venovenous techniques require a roller pump and are described in more detail in the literature.[6,29]

Cardiopulmonary bypass (CPB) is the most invasive and labor-intensive technique for rewarming. It utilizes the standard femoral artery-femoral vein access. It has the advantages of complete hemodynamic support during rewarming, and rapid rise in temperature, as quickly as 1-2°C (2-4°F) every 3 to 5 minutes. CPB is the modality of choice in cardiopulmonary arrest. It also is indicated in patients with 1) cardiac arrest or hemodynamic instability with a core temperature of less than 32°C (90°F); 2) no response to less invasive techniques; 3) completely frozen extremities; and 4) rhabdomyolysis with severe hyperkalemia.[9] Disadvantages include the need for considerable set-up time, trained personnel, and access to appropriate resources. Historically, CPB has required systemic anticoagulation, which is contraindicated in patients with known traumatic injuries, but the use

of heparin-bonded tubing allows CPB use even in the setting of trauma.[44,47] Long-term outcome of patients who have severe hypothermia treated with CPB has been favorable.[48]

Future Techniques

New developments in hypothermia treatment continue to emerge. In animal studies, a technique under investigation is administration of intravenous fluids heated to 65°F (149°F). Rewarming rates doubled to 2.9 to 3.7°C (37.22 to 38.66°F) per hour without increasing apparent complications.[49,50] However, this technique is likely to cause skin damage in hypothermic human patients and although animal studies are promising, further investigation is needed.[51]

Indwelling venous heat-exchange catheters, placed in the femoral vein, have been used to produce mild hypothermia in studies of neuroprotective effects after stroke or cardiac arrest.[52,53] Their use in rewarming has not been extensively studied.[54] Because of the restriction of circulating blood volume in hypothermia,[10] they may prove to be quite effective, while being minimally invasive.

Disposition

Discharge is possible in most otherwise healthy patients with mild hypothermia who have been rewarmed, unless they have associated injuries or conditions that necessitate admission. Admit all patients with moderate or severe hypothermia. Patients who require resources not available at the facility may benefit from transfer.

Complications

Severe or prolonged hypothermia may cause noncardiogenic pulmonary edema, gastric submucosal hemorrhage, pancreatic necrosis, cerebrovascular accidents, and myocardial infarction. Pneumonia is very common secondary to loss of protective cough reflexes, cold-induced bronchorrhea, and direct injury to lung tissue. It is the leading cause of death among patients who survive rewarming.[37]

Prognosis

Mortality rates among hypothermic patients are highly variable and attempts to identify prognostic factors have had limited success. Few studies are published, many of which are retrospective chart reviews in selected patient populations that have produced variable results, some of which contradict each other.

Danzl and colleagues conducted a prospective multicenter study in North America involving 428 patients (mostly adults) with no standardized rewarming methods. The overall mortality rate was 17%. They delineated five risk factors for death within 24 hours: blood urea nitrogen greater than 100, systolic blood pressure less than 70 mm Hg, prehospital CPR, tracheal intubation, and nasogastric tube placement. Factors found not to correlate with survival included immersion, major trauma, comorbid illness, age, and sex.[30]

A small retrospective French study of 47 ICU patients during a 19-year period with a mortality rate of 38% found the following factors to be associated with a poor prognosis: delay after the incident, a slow rewarming rate, and the need for vasopressors. In this highly selected group of very sick patients, baseline temperature did not correlate with prognosis.[55]

A small Austrian study of 55 patients, all with core temperatures less than 30°C (86°F), used different methods of rewarming based on the hemodynamic status of the patients.[56] Survival was 100% in the group with stable hemodynamics who were rewarmed with airway rewarming, warm fluids, and insulation; 72% in the group with unstable hemodynamics who were rewarmed by peritoneal dialysis; and 13% in the group with cardiac arrest who were rewarmed by extracorporeal circulation. The prognosis was excellent in patients without hypoxia or serious underlying disease.

A much larger Swiss study of 234 patients during an eight-year period found that the most important negative survival factors included asphyxia, slow rate of cooling, asystole on arrival, elevated serum potassium level, and increased age.[57] Temperature on arrival was not correlated with survival.

Although hypothermia is a favorable prognostic factor after hypoxic events, a fact recognized in pediatric submersion, prolonged submersion with resultant severe hypothermia still carries a grim prognosis.[24,58-60]

Therapeutic Hypothermia. The cerebroprotective effects of hypothermia have potential benefits in patients who have had nontraumatic hypoxic insults (e.g., cardiac arrest and stroke).[61,62] This evolving field is beyond the scope of this article, but is relevant to the use of therapeutic hypothermia in trauma patients, which is reviewed in the next section.

Hypothermia in Trauma Patients

Hypothermia in trauma has the potential to increase morbidity and mortality through its effects on coagulation and the heart and has been identified as one element in a lethal triad of hypothermia, acidosis, and coagulopathy.[64] Recognition of the lethal triad has led to the introduction of abbreviated laparotomy (damage control surgery) in some trauma patients.[64,65] However, hypothermia has potential benefits in trauma as well, by cerebral protective effects in traumatic brain injury. To date, studies have had mixed results.[66-69] Hypothermia also has the potential to permit otherwise impossible salvage surgery,[64] and to allow suspended animation during circulatory arrest. A number of laboratory studies have shown cerebral protection using therapeutic hypothermia in dogs who have been exsanguinated or have had delayed resuscitation from cardiac arrest.[69-74] In addition, the effect of hypothermia in decreasing immunologic response may be beneficial in trauma.[75]

Although ATLS stresses measures to prevent and treat hypothermia in trauma patients, it is not clear whether hypothermia itself causes increased mortality or whether it is associated with metabolic derangements caused by severe trauma. A retrospective review of 173 trauma patients found no increase in mortality in hypothermic trauma patients when patients were stratified by injury severity.[76] An older, retrospective study of 71 patients characterized hypothermia as an "ominous predictor of

survival."[19] The mortality rate was 40% when the core temperature was less than 34°C (93°F), 69% when temperature was less than 33°C (91°F), and 100% when the temperature was less than 32°C (90°F). However, this study looked only at severely injured patients (i.e., those with a Injury Severity Score > 25), did not pursue aggressive rewarming, and all patients underwent surgery for definitive treatment. The authors were careful to stress that their study did not address the issue of whether the hypothermia itself or the severity of the injury that caused the hypothermia was responsible for the increased mortality. A more recent study of 395 trauma patients in an intensive care setting found a few survivors with core temperatures less than 32°C (90°F).[77]

Conclusion

Hypothermia is a potentially life-threatening problem in the ED. Recognition should be straightforward, but diagnosis of associated conditions and management are fraught with difficulties. Mildly hypothermic patients without comorbidities can rewarm themselves with minimal help. Most other hypothermic patients require active rewarming, which can be a difficult process. Early aggressive management may prevent life-threatening complications and avoid the morbidity and mortality associated with hypothermia.

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CE/CME Objectives

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

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

CE/CME Instructions

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

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CE/CME Questions

1. Hematocrit:
   A. increases due to hypothermia.
   B. is unchanged due to hypothermia.
   C. decreases due to hypothermia.
   D. is not consistently increased or decreased due to hypothermia.

2. The most accurate measure of core body temperature is:
   A. rectal temperature.
   B. esophageal temperature.
   C. tympanic temperature.
   D. epitympanic temperature.

3. A patient who is hypothermic, alert, and shivering most likely has which of the following conditions?
   A. Mild hypothermia
   B. Moderate hypothermia
   C. Severe hypothermia

4. Which of the following conditions is a contraindication to resuscitation in a hypothermic patient?
   A. Dependent lividity
   B. Fixed, dilated pupils
   C. Tissue decomposition
   D. Apparent rigor mortis

5. A hypothermic patient with a core temperature of 25° C (77°F) is in ventricular fibrillation. Which of the following describes the recommended method of defibrillation?
   A. Three attempts at half of standard energy settings, then stop if unsuccessful.
   B. Three attempts at half of standard energy settings. If unsuccessful, warm the patient to 32° C (90°F) and attempt defibrillation again with three attempts at standard energy settings.
   C. Three attempts at standard energy settings. If unsuccessful, then stop.
   D. Three attempts at standard energy settings. If unsuccessful, warm the patient to 32° C (90°F) and attempt defibrillation again with three attempts at standard energy settings.

6. When following arterial blood gas results in hypothermic patients, which of the following methods should be used to correct the values?
   A. Use temperature corrected values corrected to 37° C (98.6°F).
   B. Use temperature corrected measures corrected to 35° C (95°F).
   C. Use uncorrected values.
   D. Arterial blood gases cannot be used in hypothermic patients.

7. A moderately or severely hypothermic patient should be handled gently because:
   A. the seizure threshold is decreased.
   B. ventricular fibrillation threshold is decreased.
   C. he is shivering vigorously.
   D. None of the above

8. Which of the following is a method of active external rewarming?
   A. Airway rewarming
   B. Forced air rewarming
   C. Peritoneal lavage
   D. Cardiopulmonary bypass

9. Which of the following has the potential to increase heat loss in ED patients?
   A. Administration of heated IV fluids at 40-42°C (104-108°F)
   B. Heating trauma rooms to 28°C (82°F)
   C. Intubation and administration of heated, humidified oxygen
   D. Administration of meperidine for pain relief

10. A patient whose core temperature is 28°C (82°F) would be expected to have which of the following solely on the basis of hypothermia?
   A. Increased heart rate
   B. Normal heart rate
   C. Decreased heart rate

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

In Future Issues:

Pelvic trauma

Thomson American Health Consultants
Building Six, Suite 400
3525 Piedmont Road NE
Atlanta, Georgia 30305-1515
Tel (404) 262-7436 Fax (404) 262-7837
www.ahcpub.com

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