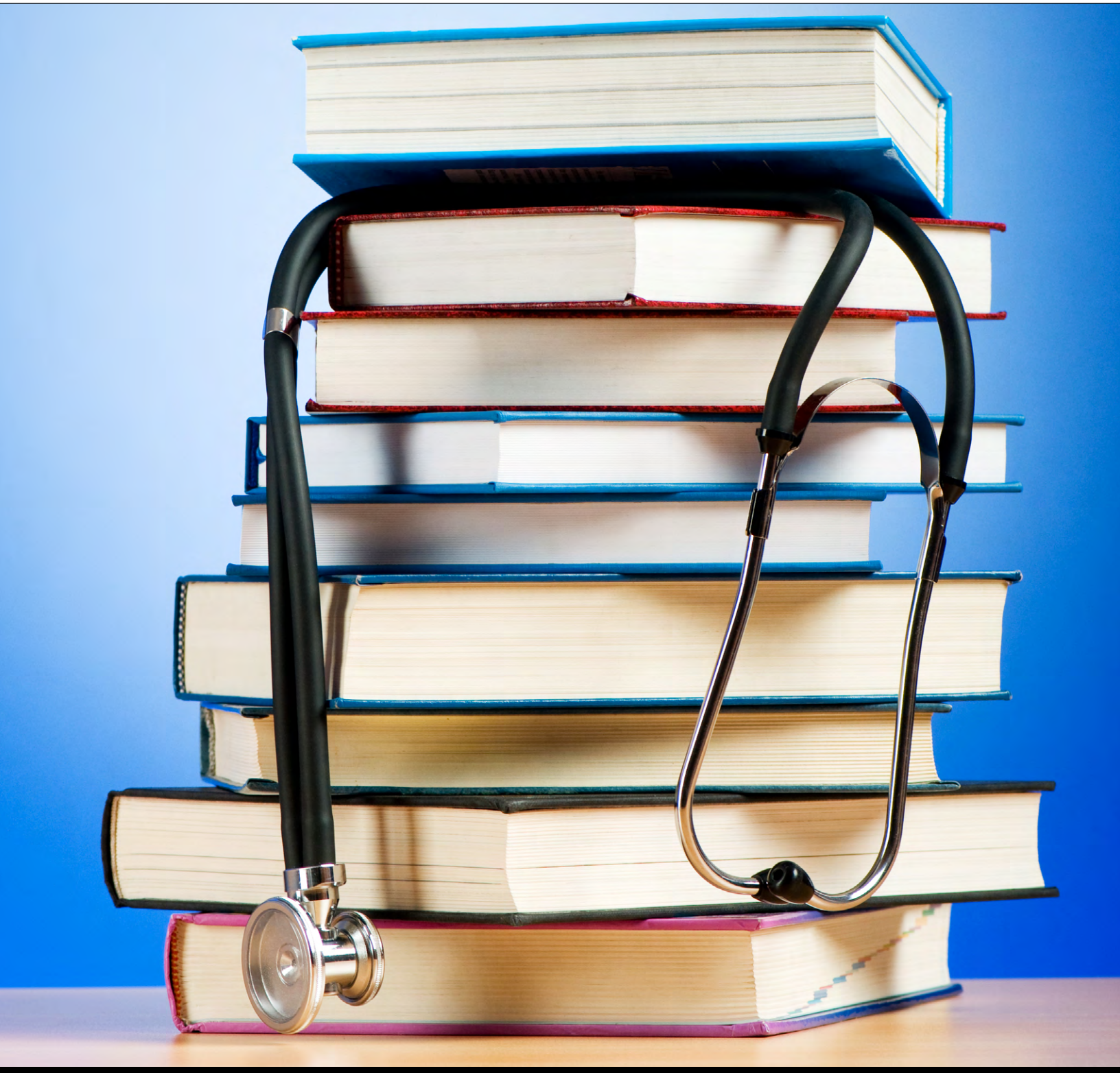
Critical decisions in emergency medicine



THE 2016 LLSA LITERATURE REVIEW

THE OFFICIAL CME PUBLICATION OF THE AMERICAN COLLEGE OF EMERGENCY PHYSICIANS

The LLSA Literature Review

Synopses of articles from ABEM's 2016 Lifelong Learning and Self-Assessment Reading List

FROM THE EDITORS

Since April 2003, *Critical Decisions in Emergency Medicine* has included the bonus feature "The LLSA Literature Review." The impetus for this section was our desire to provide ACEP members with yet another tool to use when preparing for the continuous certification initiative of the American Board of Emergency Medicine (ABEM), specifically, the Lifelong Learning and Self-Assessment (LLSA) tests. Each year, as part of this program, ABEM publishes a list of articles focused on selected portions of the emergency medicine core content. These articles become the LLSA reading list for that year, and the questions for the tests are drawn from these articles.

From August 2015 through August 2016, each monthly issue of *Critical Decisions* has provided a summary of one of the articles from ABEM's 2016 reading list, with bullets highlighting the elements relevant to emergency medicine practice. This online supplemental issue includes a full collection of those summaries, which are intended to highlight the important concepts of each article. We are pleased to offer this benefit free to ACEP members, and hope you find it useful. ACEP members also can download full versions of the articles by logging in at acep.org/llsa.

If you would like to see what else *Critical Decisions* has to offer (clinical lessons, ECG and imaging reviews, drug reviews, and more), we invite you to explore a sample issue online at www.acep.org/criticaldecisions.

Best wishes,

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IN THIS ISSUE

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Critical decisions in emergency medicine

Critical Decisions in Emergency Medicine is the official CME publication of the American College of Emergency Physicians. Additional volumes are available to keep emergency medicine professionals up to date on relevant clinical issues.

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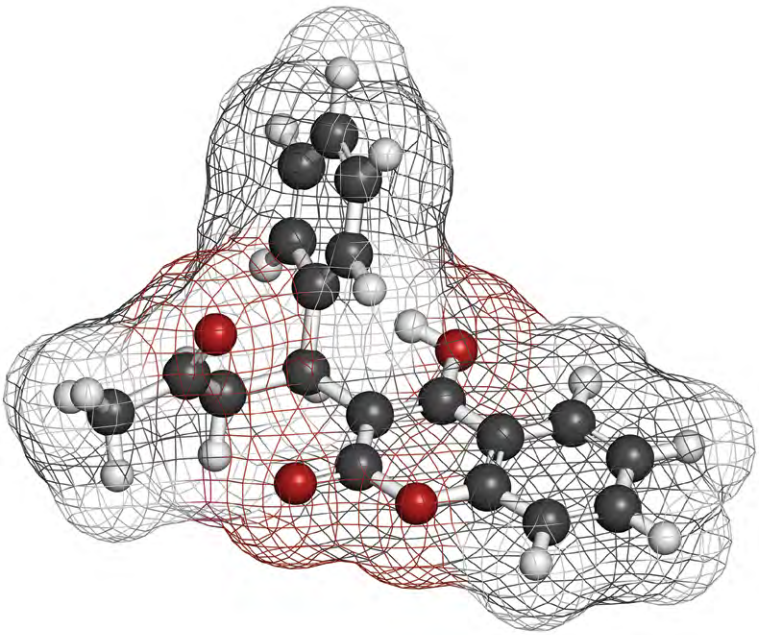
Rapid Reversal of Warfarin-Associated Hemorrhage in the Emergency Department by Prothrombin Complex Concentrates



By **David Yamane, MD**
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Reviewed by **J. Stephen Bohan, MS, MD, FACEP**

Frumkin K. *Ann Emerg Med.* 2013;62(6):616–26.

Warfarin inhibits the synthesis of vitamin K-dependent coagulation factors II, VII, IX, and X. Reversal of warfarin in the setting of life-threatening hemorrhage traditionally is done with vitamin K and fresh frozen plasma (FFP). However, this method can be time consuming and requires substantial fluid administration.

Vitamin K replacement restores the production of intrinsic clotting factors necessary for sustained reversal of anticoagulation. The recommended dose is 5 to 10 mg intravenously; however, it can take up to 4 hours to achieve the desired effects. The rate of anaphylaxis is only 3/10,000.

Fresh frozen plasma requires ABO blood group compatibility testing and 30 to 60 minutes to thaw. The initial recommended minimal dose for warfarin anticoagulation reversal is 15 ml/kg (approximately 4 units for the average 70-kg person); it typically takes 13 to 48 hours to achieve full effect.

Recombinant-activated factor VIIa (rFVIIa) has been used as an off-label treatment for hemorrhage in non-hemophiliac patients. In healthy patients with an international normalized ratio (INR) less than 2, the agent has been proven to reverse the INR in less than 1 hour; however, the clinical impact of this reversal is unknown. rFVIIa carries a risk of thrombotic events (10% to 20%), but should remain a consideration in patients with religious objections to blood products.

Prothrombin complex concentrates (PCCs) are derived from pooled human plasma. Three-factor PCCs contain factors II, IX, and X, proteins C and S, and a small amount of heparin; 4-factor PCCs also contain factor VII. These agents require a small volume of administration (<100 mL) and eliminate the need for ABO-compatibility testing. Reversal of INR can occur in 10 to 30 minutes and lasts for about 6 hours. The use of PCCs in brain hemorrhage has been associated with improved neurologic outcomes and reduced hematoma growth, compared to FFP. Risks include thrombotic events (occurring in 0.9% to 3.8% of patients) and the potential for transmitting infectious agents.

KEY POINTS

- For warfarin reversal of life-threatening bleeding, vitamin K should be administered early through an IV route.
- FFP should be given when other agents are unavailable.
- PCC, preferably 4-factor, is an attractive option for wafarin reversal — especially in brain hemorrhage, due to its low volume, faster INR reversal, and growing evidence of clinical superiority.
- When using 3-factor PCC, consider using FFP or rFVIIa, given the lack of factor VII. If used alone, check the INR 15 minutes post-administration.

Fever in the Postoperative Patient

By **Kathleen Wittels, MD**
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Reviewed by **J. Stephen Bohan, MS, MD, FACEP**

Narayan M, Medinilla SP. *Emerg Med Clin North Am.* 2013;31(4):1045-58.

Postoperative fever is defined as a temperature greater than 38°C (100.4°F); it may be infectious or noninfectious in etiology. Infectious causes typically present later than 48 hours postoperatively, and the likelihood of infection rises as the time from the procedure increases.

Immediate fever occurs during the procedure or within 1 hour following it, and most often is related to inflammatory changes from cytokines. Other causes of immediate postprocedure fever include reactions to medications or blood transfusions, preprocedure infections, fulminant surgical site infections, trauma, and adrenal insufficiency.

There are several potentially life-threatening causes of postoperative fever. Necrotizing soft-tissue infections can occur at any time and are characterized by "dishwater drainage," erythema, edema, induration, bullae, and pain out of proportion to the examination. Treatment includes urgent surgical consultation and the initiation of broad-spectrum antibiotics and intravenous fluids.

Pulmonary embolism (PE) is another dangerous culprit. Fever precipitated by PE usually is low grade (<38.3°C [101°F]) and short-lived. The exception is a septic PE, which causes a high temperature.

Fever and abdominal pain in a patient who has had intra-abdominal surgery should raise concerns for an anastomotic leak and abscess formation. Time to presentation can vary from 1 week to several months following the procedure. Other infectious causes of fever include urinary tract infections (usually from indwelling catheters), pneumonia (risks include exposure to mechanical ventilation), and catheter-related bloodstream infections (CRBIs). If a CRBI is suspected, maintain a low threshold for catheter removal. Postoperative patients also are at high risk for *Clostridium difficile* infections, which can lead to toxic megacolon, a surgical emergency.

Noninfectious causes of postoperative fever include alcohol withdrawal/delirium tremens and adrenal insufficiency. These diagnoses should be treated supportively while evaluating for other triggers. Malignant hyperthermia is a rare cause of postoperative fever. Most often associated with inhaled anesthetics, it requires ventilatory and circulatory support, as well as treatment with dantrolene.

In summary, the degree of fever and time of onset can help direct management strategies for postoperative fever. Laboratory studies, including blood and urine cultures, and imaging should be tailored to each individual case. The definitive treatment of infectious causes of fever includes source control and antibiotic therapy.

KEY POINTS

- Postoperative fever can be infectious or noninfectious in etiology, with the former occurring later in the postoperative period.
- Infectious causes of postoperative fever include necrotizing soft-tissue infections, intra-abdominal anastomotic leak/abscess, urinary tract infections, pneumonia, *C. difficile*, infected prosthetic, and catheter-related bloodstream infections. Evaluation, including cultures, imaging studies, and antibiotic therapy, should be tailored to the individual patient, with close hemodynamic monitoring for signs of sepsis.
- Noninfectious causes of postoperative fever include pulmonary embolism, alcohol withdrawal, and adrenal insufficiency. The latter occurs most commonly due to steroid withdrawal, which readily responds to replacement hydrocortisone. All require aggressive supportive therapy with additional treatments tailored to the underlying diagnosis.

Conjunctivitis: A Systematic Review of Diagnosis and Treatment

By **William Bianchi DO, MSc, LT (MC) USN**; and **Daphne Morrison Ponce, MD, LT (MC) USN**
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Reviewed by **J. Stephen Bohan, MS, MD, FACEP**

Azari A, Barney N. *JAMA*. 2013;310:1721-1729.

Conjunctivitis is defined as inflammation of the thin translucent membrane (conjunctiva) that lines the anterior part of the eye and the inside of the eyelids. It has many etiologies that require different management strategies.

Viral conjunctivitis is the most common etiology of infectious inflammation (responsible for up to 80% of acute cases), the majority of which are caused by adenovirus. The two most common clinical etiologies are pharyngoconjunctival fever (pharyngitis, fever, and bilateral conjunctivitis), and epidemic keratoconjunctivitis (more severe conjunctivitis, often with associated lymphadenopathy). Transmission occurs through direct contact, and communicability is estimated at 10 to 14 days. Treatment largely is supportive; artificial tears, antihistamines, and cold compresses are helpful in alleviating symptoms.

Two viruses with specific treatment regimens are herpes simplex virus (HSV) and herpes zoster (HZV). HSV usually is characterized by unilateral thin/watery discharge with vesicular eyelid lesions. Treatment of HSV consists of topical and oral retrovirals (acyclovir). Topical steroids can potentiate infection and should be avoided. Herpes zoster virus can have a propensity for V1 and V2 of the trigeminal nerve, affecting the eyelids and conjunctiva. Suspected HZV infections that present with vesicles involving the eye or tip of the nose (Hutchinson sign) require referral to ophthalmology for further evaluation.

Bacteria is the second most common cause of infectious conjunctivitis. The combination of bilateral matting of

KEY POINTS

- The most common etiology of conjunctivitis is viral; treatment is supportive and patients should be encouraged to wash their hands to control transmission. Over-the-counter remedies may provide symptomatic relief.
- Bacterial conjunctivitis is characterized by bilateral mattering of the eyelids, lack of itching, and no history of conjunctivitis.
- Any broad-spectrum antibiotic is reasonable for treating bacterial conjunctivitis and can decrease the duration of symptoms; steroids are not recommended.
- Treatment of gonorrheal conjunctivitis should include intramuscular ceftriaxone; oral azithromycin and doxycycline should be utilized for management of possible concomitant chlamydial infection.
- Topical antihistamines and mast-cell stabilizers are recommended for treatment of allergic conjunctivitis.
- Always consider systemic diseases when evaluating conjunctivitis.
- One-week follow up with ophthalmology is recommended for patients who wear contact lenses, require steroids, have photophobia, or who show no improvement after 1 week.
- Steroid drops must be used judiciously as they can increase risk of complications.

Conjunctivitis: A Systematic Review of Diagnosis and Treatment

continued

the eyelids, lack of itching, and no history of conjunctivitis are strongly predictive for bacterial infection. Most cases are self-limiting, thus clinical observation is a reasonable treatment option. However, topical antibiotics are shown to reduce the duration of the disease and should be considered when patients have severe mucopurulent discharge, wear contact lenses, or are immunocompromised. There are no significant differences between any of the broad-spectrum topical antibiotics; all achieve clinical cure. There is no role for topical steroids. Cases of MRSA conjunctivitis are becoming increasingly common, and require referral to ophthalmology for treatment with fortified vancomycin.

There are several clinical findings of high clinical concern. First, severe purulent discharge can indicate infection caused by *neisseria gonorrhoeae,* and carries a high risk of corneal involvement and subsequent perforation. Cultures should be taken, and treatment should consist of intramuscular ceftriaxone with empiric treatment for possible concomitant chlamydial infection. Second, unilateral conjunctivitis with hyperemia, mucopurulent discharge, and lymphoid follicle formation are concerning for chlamydial infection. These cases should be treated with oral azithromycin and doxycycline; the use of topical antibiotic therapy is not supported. In both cases, patient's sexual partners also must be treated.

Finally, chlamydia trachomatis subtypes A-C (the leading cause of blindness worldwide) lead to a roughening of the inner surface of the eyelids. Treatment consists of a single dose of azithromycin or 6 weeks of topical antibiotic ointments.

Allergic reactions are the most frequent noninfectious culprits of conjunctivitis, affecting 15% to 40% of the population. Treatment consists of artificial tears, topical decongestants, NSAIDs, antihistamines, and mast-cell stabilizers. Long-term use of antazoline and naphazoline should be avoided due to rebound hyperemia. Steroids are associated with cataract formation and should be used judiciously.

Always consider the following systemic diseases when evaluating conjunctivitis: mucous membrane pemphigoid, Sjögren syndrome, Kawasaki disease, Stevens-Johnson syndrome, and carotid cavernous fistula.

In addition to the above indications for ophthalmology referral, 1-week follow up is recommended for patients who wear contact lenses, require steroids, have photophobia, or have no improvement after 7 days.

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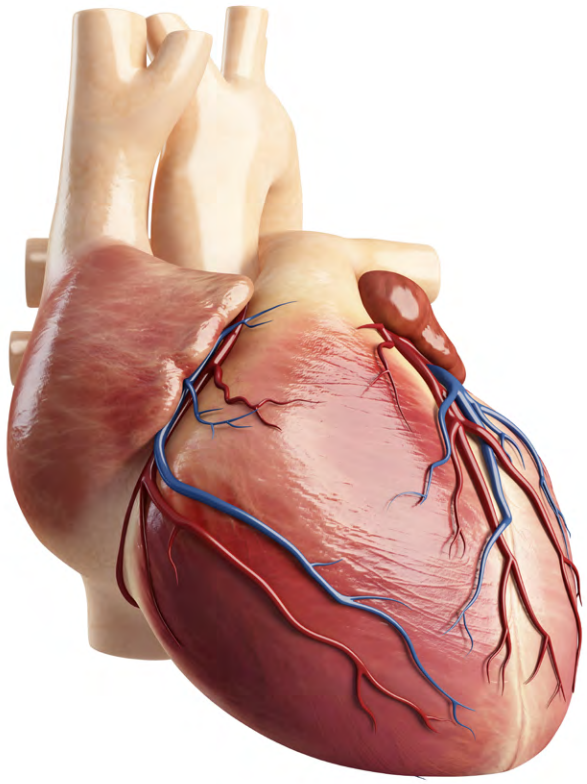

A Randomized Trial of Colchicine for Acute Pericarditis

By Katie E. Golden, MD
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Reviewed by J. Stephen Bohan, MS, MD, FACEP

Imazio M, Brucato A, Cemin R, et al; ICAP Investigators. *N Engl J Med.* 2013;369(16):1522-1528.

Colchicine traditionally has been recommended as the first-line treatment for recurrent pericarditis. The referenced study, conducted in Italy from 2005 to 2010, was designed to examine the medication's role in the treatment of acute cases.

The 240 patients in the multicenter, double-blind trial were randomized to receive either colchicine (.5 mg twice a day for 3 months) or placebo. Both groups, which were demographically and clinically similar, received concurrent anti-inflammatory medication (aspirin or ibuprofen).

Patients over the age of 18 with new-onset pericarditis were invited to enroll. The diagnosis was made using at least two of the following criteria: chest pain typical of pericarditis, a friction rub, suggestive electrocardiographic findings, or a new pericardial effusion. The diagnosis of recurrent pericarditis required a symptom-free interval of 6 weeks; otherwise, patients were diagnosed with incessant pericarditis. Remission was defined as a full resolution of symptoms, as well as electro- and echocardiographic signs of disease.

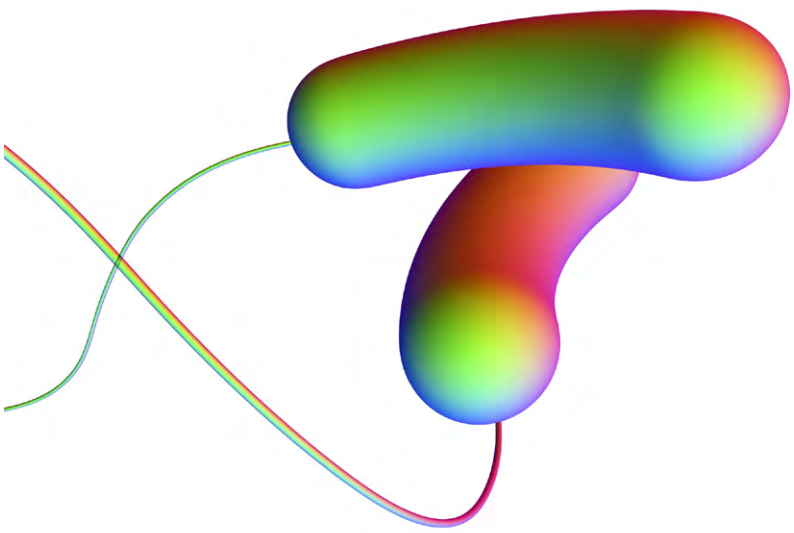
Patients were followed for almost 2 years with frequent follow-up visits, during which investigators collected laboratory data, an electrocardiogram, and an echocardiogram. No patients were lost to follow up, and adherence was over 95%.

The primary outcome was incessant or recurrent pericarditis, which occurred less frequently in the colchicine group versus the placebo group (16.7% versus 37.5%, p <.001), with a relative risk reduction in the colchicine subset of .56 (95% confidence interval .3 to .72). The colchicine group also demonstrated a statistically significant decrease in symptoms at 72 hours (19.2 % versus 40.0%, p = 0.001), the number of recurrences per patient (.21 versus .52, p = 0.001), and hospitalization related to pericarditis (5.0% versus 14.2%, p = 0.02). The medication also improved the rate of remission within 1 week of treatment (85.0% versus 58.3%, p <0.001). The overall number of adverse events was similar in the two study groups. Diarrhea was the most common side effect, which occurred in less than 10% of patients.

In summary, this is the first study to investigate the role of colchicine in the treatment of acute pericarditis. This research suggests that the drug can reduce the rate of recurrent or incessant pericarditis, as well as the duration of symptoms and hospitalization associated with the diagnosis. Furthermore, these effects were seen without a loading dose, which often is associated with the unpleasant gastrointestinal side effects of the medication. There was no significant increase in adverse events or side effects in the patients treated with colchicine.

KEY POINTS

- Colchicine historically has been recommended for the treatment of *chronic* pericarditis. This is the first prospective trial to study the drug in *acute* pericarditis diagnosed by symptoms such as chest pain, ST-segment changes, and new effusions.
- Colchicine was shown to reduce rates of both recurrent and incessant pericarditis as compared to placebo. It also reduced the length of symptoms, number of recurrences, and hospitalization associated with pericarditis.
- These results were seen with low-dose colchicine without a loading dose. This regimen may help reduce the rate of diarrhea, the drug's most common adverse side effect.



A Randomized Trial of Protocol-Based Care for Early Septic Shock

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Reviewed by **J. Stephen Bohan, MS, MD, FACEP**

The ProCESS Investigators. *NEJM.* 2014;370(18):1683-1693.

Ten years ago, a landmark single-center study of sepsis management changed the way emergency physicians cared for sepsis patients by creating a novel protocol, early goal-directed therapy (EGDT). In the decade since that research was published, strategies for sepsis management have underdone many changes that call into question the necessity for every element of EGDT.

The 2014 multicenter ProCESS trial randomly assigned patients to one of three groups for the initial 6 hours of treatment: protocol-based EGDT; protocol-based therapy not requiring placement of a central venous catheter, inotrope administration, or blood transfusions; and usual care. The primary endpoint for the study was 60-day in-hospital mortality among the three groups. Secondary endpoints included all-cause mortality at 90 days and 1 year, as well as duration of any cardiovascular, respiratory, or renal failure, hospital and ICU length of stay, and hospital disposition.

The research was conducted in the emergency departments of 31 academic facilities that used lactate levels as a shock screening method and adhered to *Surviving Sepsis* guidelines, but had no routine resuscitation protocols for septic shock and did not routinely use central venous oxygen saturation ($ScvO_2$) catheters. All 1,341 patients enrolled in the study were at least 18 years old, met two SIRS criteria, and had either refractory hypotension or a serum lactate greater than 4mmol/L. The EGDT protocol employed a central venous catheter for pressure, $ScvO_2$ monitoring, the administration of intravenous fluids, vasopressors, or packed red blood cells.

The "standard" protocol used a peripheral IV or central line (if no other access was available), and a systolic blood pressure and shock index as goals for therapy, with packed red blood cells only administered if hemoglobin levels dropped below 7.5 g/dL. No specific agents were required for either protocol arm. Finally, the usual care arm relied on the bedside provider to direct all care.

Both protocol-based groups saw an increase in the use of central venous catheters, fluid administration, vasoactive agents, and red blood cell transfusions. Patients in these groups also demonstrated an increased need for intensive care and renal replacement therapy. There were no advantages in mortality for the protocol groups over usual care. There was no benefit observed in the EGDT group from the mandated central venous catheterization and central hemodynamic monitoring, and there was no significant difference between 90-day mortality, 1-year mortality, and the need for organ support.

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KEY POINTS

- There was no difference in primary or secondary outcomes between the EGDT protocol, standard protocol, and usual care in the management of septic shock.
- Both protocol-based care groups had an increased use of central venous catheters, intravenous fluids, vasoactive agents, and blood transfusions, with no advantage in morbidity or mortality.

Physician Orders for Life-Sustaining Treatment and Emergency Medicine: Ethical Considerations, Legal Issues, and Emerging Trends

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Reviewed by **J. Stephen Bohan, MS, MD, FACEP**

Jesus J, Geiderman J, Venkat A, et al. *Ann Emerg Med.* 2014;64(2):140-144.

Emergency physicians, who frequently care for patients at the end of life, are required to make time-sensitive decisions regarding interventions — often without the ability to communicate with these vulnerable patients. Advanced directives, do not resuscitate (DNR), and do not intubate (DNI) orders were designed to address patient preferences in end-of-life care, but a number of issues limit their usefulness.

Although DNR/DNI orders direct clinicians to avoid CPR and intubation, they offer little guidance related to other interventions and procedures. Clinicians frequently interpret these directives to mean a refusal of *all* aggressive treatments or procedures, despite the fact that many patients desire aggressive treatment for *acute* conditions. In addition, patients with DNR/DNI orders are less likely to be admitted to the ICU regardless of age and functional status, less likely to be treated according to quality assurance measures, and less likely to be hospitalized for pneumonia.

Advance directives, while aimed to improve clarity and flexibility over DNR/DNI orders, have poor prevalence (18% of population) and even poorer portability. Only 5.6% of patients who report having an advance directive actually bring it with them to the hospital.

Physician orders for life-sustaining treatment (POLST) address standardized portable medical orders, including resuscitation, intubation, antibiotics, and artificial nutrition and hydration. These orders serve as a middle ground for translating patient goals and values into medical orders that can be easily understood and are specific enough to apply to most clinical encounters.

POLST documents appear to increase the effectiveness of care delivered according to a patient's wishes. A multicenter study examining the consistency between treatments provided to nursing home patients and their POLST documents reports that 94% of the interventions provided were consistent with the patients' orders.

The POLST paradigm has the potential to improve the quality of end-of-life care and more accurately relay patient preferences. However, many challenges remain, including inadequate paperwork completion, availability to the treating clinician, and inadequate education on applicable statute and legal protections. It is important for clinicians to verify the accuracy of each POLST form with the patient or proxy. As the penetration and adoption of these documents continue to rise, a secure "cloud-based," centralized federal system has been suggested to help overcome the barriers of availability and portability.

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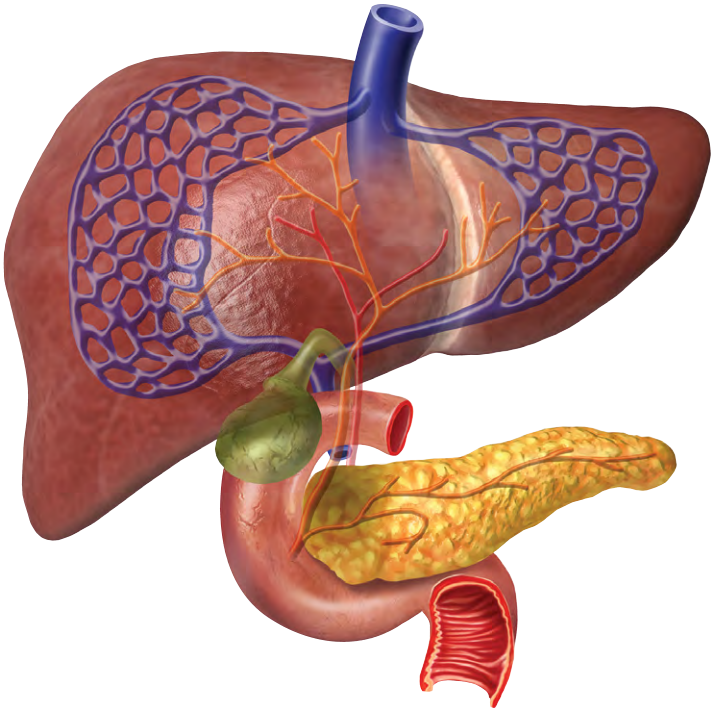
KEY POINTS

- POLST documents allow patients to communicate their specific care goals more clearly than the traditional DNR or DNI orders, and can be implemented more easily and widely than standard advance directives.
- Emergency physicians should familiarize themselves with POLST forms to minimize potential errors in interpretation and execution.
- Regardless of the documentation patients bring to their visit, all care decisions should be discussed with the patient and/or their proxy if available, especially when a patient with intact decision-making capacity chooses to override a prior document.



Acute Liver Failure

By **Laura A. Welsh, MD,** and **Kathleen Wittels, MD**
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Reviewed by **J. Stephen Bohan, MS, MD, FACEP**

Bernal W, Wendon, J. *N Engl J Med.*2013;369:2525-34.

Acute liver failure, the rapid onset of liver injury over 8 to 12 weeks, often is seen in patients without preexisting liver disease. It may be further characterized based on the time from the onset of jaundice to the development of encephalopathy. Mortality approaches 50%, and the development of encephalopathy is a poor prognostic indicator. Early recognition and treatment are paramount to survival.

Globally, the disease's most common etiology is viral hepatitis. In the United States, half of all acute liver failure cases are the result of drug-induced hepatotoxicity, most commonly caused by acetaminophen. Other etiologies include sepsis, neoplastic disease, and Budd-Chiari syndrome.

Early discussion with and possible referral to a liver transplant center are critical to the survival of these patients. Initial management principles are similar to those for other critically ill patients, with a focus on early restoration of systemic perfusion. Endotracheal intubation may be required for airway protection in those with altered mentation; many patients will require renal replacement therapy. Early treatment with acetylcysteine improves outcomes in cases of acetaminophen toxicity, and also can be beneficial to patients with other etiologies of acute liver failure.

The loss of hepatic function provides several specific treatment issues in these patients, many of whom are functionally immunosuppressed. Hypoglycemia is common due to the loss of glycogen stores. Though the international normalized ratio is elevated, the synthesis of both pro- and anticoagulants is affected; bleeding is uncommon.

Additionally, the detoxification of ammonia is impaired in this population. The increased circulating levels can result in cerebral edema and resultant encephalopathy. Intracranial hypertension from severe cerebral edema is a leading cause of death.

Traditional hyperammonemia treatments, including nonabsorbable antibiotics, are inadequate in acute liver failure. Instead, patient management should focus on the prevention of encephalopathy and reduction of its severity through infection control and fever prevention. Both side effects lead to increased ammonia levels, and fever increases cerebral uptake. Patients with established encephalopathy may benefit from prophylactic treatment with osmotic agents such as hypertonic saline or mannitol.

KEY POINTS

- Acetaminophen toxicity is the leading cause of acute liver failure in the US, while viral etiologies are most common in the developing world. The latter may be ameliorated by the administration of acetylcysteine. Although confirming the etiology of the disease may aide in treatment, it should not delay early discussion with and referral to a liver transplant center.
- The initial treatment is similar to that of other critically ill patients, and should focus on improved systemic perfusion and airway protection. Special attention must be paid to infection control; these patients are functionally immuno-suppressed, and infection exacerbates encephalopathy.
- The development of encephalopathy is a poor prognostic indicator, and the resultant cerebral edema and intracranial hypertension is the leading cause of death in these patients.

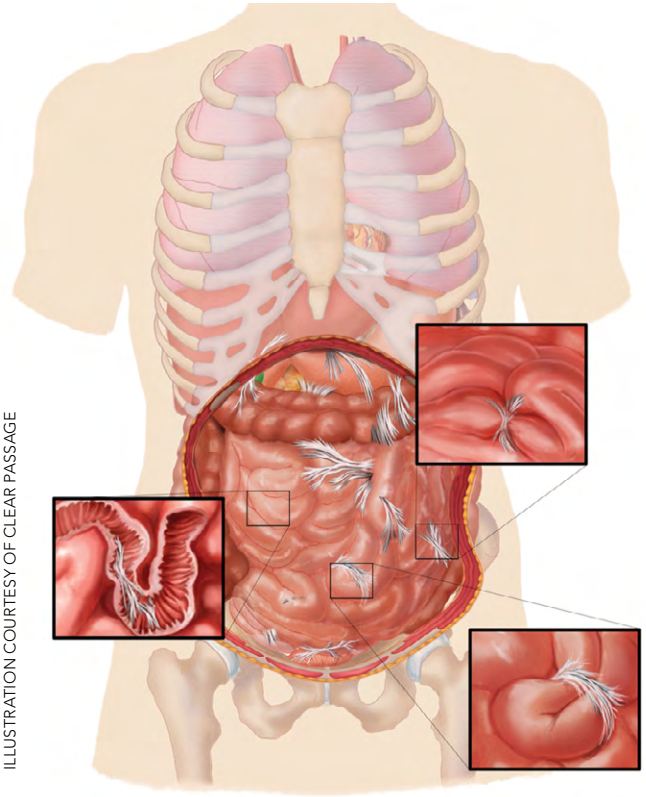

ILLUSTRATION COURTESY OF CLEAR PASSAGE

Adult Small Bowel Obstruction

By **Erika Phindile Chowa, MD,** and **Kathleen Wittels, MD**
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Reviewed by **J. Stephen Bohan, MS, MD, FACEP**

Taylor M, Lalani, N. *Acad Emerg Med.* 2013;20(6):528-544

Small bowel obstruction (SBO) is a common clinical diagnosis in the emergency department. Two percent of patients who present with abdominal pain will have an SBO, as will 15% of those admitted to a surgical unit.

The diagnosis most frequently is secondary to adhesions from prior abdominal surgery. Other causes include neoplasms, hernias, and Crohn's disease. The risk of serious complications such as strangulation and bowel necrosis is high; it is essential to diagnose SBO early.

Although abdominal pain is present in most cases of SBO, no other components of a patient's history can reliably and accurately predict the diagnosis. However, some studies indicate that a history of prior SBO coupled with abdominal pain might serve as a reliable predictor for a subsequent obstruction. Abnormal bowel sounds and abdominal pain with constipation also may indicate SBO. Abdominal distension is the most reliable physical examination finding for predicting the disorder.

Diagnostic modalities that can be used to evaluate for SBO include radiography, computerized tomography (CT), magnetic resonance imaging (MRI), and ultrasound (US). Plain radiography is the least reliable modality; an SBO on x-ray is characterized by dilated loops of bowel and air fluid levels.

CT has high sensitivity and specificity for small bowel obstruction, particularly when using 64-slice cuts. An SBO on CT is characterized by dilated loops of bowel proximal to decompressed loops (also known as a transition point).

The diagnostic findings on US are similar to those found on CT; however, absent or decreased peristalsis also needs to be present to confirm SBO on ultrasound. Although ultrasonography provides diagnostic accuracy, its effectiveness relies, in part, on the training and skill level of the operator. Of note, US is unable to pinpoint the transition point or etiology of an SBO.

MRI offers diagnostic accuracy, but is less optimal than other imaging modalities given its limited availability and the increased time required to perform the test. The diagnostic criteria of MRI are similar to those of CT.

KEY POINTS

- Causes of small bowel obstruction include adhesions, neoplasms, hernias, and Crohn's disease; adhesions are the most common.
- Abdominal pain plus constipation or a history of prior SBO increases the likelihood of obstruction.
- Physical examination findings suggestive of SBO include abdominal distension and decreased bowel sounds.
- CT, the most sensitive and specific diagnostic modality for making this diagnosis, will reveal dilated loops of bowel proximal to decompressed loops (also known as a transition point). With appropriate training, however, an emergency medicine practitioner can accurately diagnose an SBO with high likelihood using ultrasound, but will not be able to identify the transition point.

Bacterial Meningitis Post-PCV7: Declining Incidence and Treatment

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Reviewed by **J. Stephen Bohan, MS, MD, FACEP**

Kowalsky R, Jaffe D. *Pediatr Emerg Care*. 2013 Jun;29(6):758-766.

Since the introduction of the *Haemophilus influenza type b* vaccine in 1988, the incidence of *H. flu type B* meningitis has radically declined. *Streptococcus pneumoniae* now is the most common pathogen for bacterial meningitis in children outside the neonatal period, despite the development of the 7-valent pneumococcal conjugate vaccine (PCV7). *Neisseria meningitides* is the second most common pathogen. Although the rate has decreased in older age groups, the incidence of bacterial meningitis has remained steady in infants younger than 2 months.

Classic signs and symptoms of the disease are less common in younger age groups. Older children may have fever, headache, photophobia, seizure, or changes in mental status. Infants, however, can present with less specific signs such as hypothermia, vomiting, poor feeding, or paradoxical irritability. Physicians should evaluate for shock, volume status, neurologic deficits, and signs of increased intracranial pressure. Neck stiffness is an unreliable symptom in children younger than 2 years.

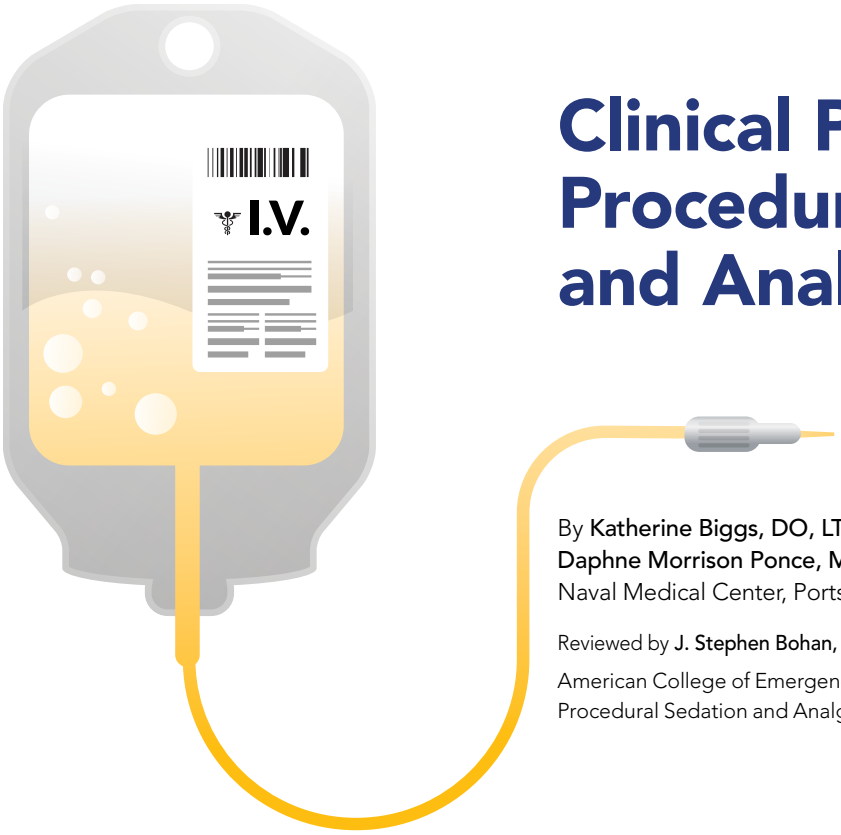
Preferably, cerebral spinal fluid (CSF) laboratory studies and a complete blood count should be acquired prior to the initiation of antibiotics. If the child is hemodynamically unstable or shows signs of increased intracranial pressure requiring computed tomography prior to the performance of a lumbar puncture, antibiotics can be administered after a blood culture has been obtained. No single laboratory test is completely diagnostic, and interpretation is especially difficult in pretreated patients. Notably, most children with CSF pleocytosis are negative for bacterial meningitis.

An enteroviral polymerase chain reaction test also may be helpful in sorting out the cause of the pleocytosis. The bacterial meningitis scoring system can be used in children older than 2 months to identify low-risk patients for whom outpatient management may be appropriate.

Empiric antibiotic therapy with parental antibiotics should be started expeditiously in any child with suspected bacterial meningitis. In infants younger than 1 month, ampicillin plus either cefotaxime or an aminoglycoside should be administered. Acyclovir therapy for human herpesviruses also should be considered, which will cover group B strep, *L. monocytogenes*, and *E. coli*. In children older than 1 month, vancomycin plus a third-generation cephalosporin will cover *S. pneumoniae and N. meningitides*. Steroids are unlikely to be beneficial and generally are not recommended.

KEY POINTS

- *S. pneumoniae* is still the most common pathogen for bacterial meningitis in children outside the neonatal period.
- In infants younger than 1 month, start ampicillin plus either cefotaxime or an aminoglycoside, and consider acyclovir therapy for herpes simplex virus coverage.
- In children older than 1 month, start vancomycin plus a third-generation cephalosporin.



Clinical Policy: Procedural Sedation and Analgesia in the ED

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American College of Emergency Physicians Clinical Policies Subcommittee on Procedural Sedation and Analgesia. *Ann Emerg Med.* 2014;63(2): 247-255.

Procedural sedation and analgesia are essential clinical tools for improving patient comfort and creating the best conditions possible for interventions. ACEP's clinical policy regarding procedural sedation and analgesia in the emergency department is a revision of the organization's influential 2005 document.

Since the original report, additional literature has become available and the Centers for Medicare and Medicaid Services (CMS) have issued guidelines on the topic, which reinforce the principle that hospital policy should be based on nationally recognized protocols.

CMS also states, "Emergency medicine-trained physicians have very specific skillsets to manage airways and the ventilation necessary to provide patient rescue. Therefore, these practitioners are uniquely qualified to provide all levels of analgesia/sedation and anesthesia (moderate to deep to general)."

Critical Definitions

ACEP's updated guidelines clarify several important definitions:

- **Procedural sedation** is a technique for administering medications to suppress the level of consciousness to enable a patient to tolerate a procedure, while still maintaining cardiorespiratory function. The continuum of sedation ranges from minimal to general anesthesia.
- **Minimal sedation** creates a near-baseline level of alertness; the patient can respond to verbal commands, but cognitive function may be impaired.
- **Moderate sedation** causes a depression of consciousness. Patients may have a delayed response to verbal commands, eyelid ptosis, slurred speech, and event amnesia.
- **Dissociative sedation** induces a trance-like cataleptic state with analgesia and amnesia, while still maintaining a patient's cardiorespiratory system.
- **Deep sedation** occurs when a patient has a depressed level of consciousness and only can be aroused by repeated or painful stimuli; respiratory support may be required.
- **General anesthesia** causes unresponsiveness to all stimuli and the absence of airway protective reflexes. It can lead to cardiovascular compromise; ventilator support is required.

Critical Questions

The policy also aimed to answer four clinical questions:

1. **Does preprocedural fasting reduce the risk of emesis or aspiration?** According to a review of recent literature, procedural sedation should not be delayed based on fasting times. Fasting has not demonstrated a reduction in the risk of emesis or aspiration (*level B recommendation*), and aspiration was a rare event in all studies reviewed.
2. **Does capnography reduce the incidence of adverse respiratory events?** Capnography is an additional tool used to detect

Clinical Policy: Procedural Sedation and Analgesia in the ED

continued

hypoventilation and apnea. It has been shown to reduce hypoxia and identify respiratory depression more consistently than pulse oximetry (*level B recommendation*). However, there is no evidence to support its ability to reduce serious adverse events.

3. **What is the minimum number of personnel needed to perform the task and manage complications?** In addition to the clinician performing the procedure, a nurse or other qualified provider should be available to continuously monitor any patient receiving procedural sedation (*level C recommendation*). Clinicians performing sedation should have an understanding of the medications used, the ability to monitor the patient, and the skills to intervene should complications arise. The literature does not provide clear evidence on the number or type of providers required for safe sedation.
4. **Can ketamine, propofol, etomidate, dexmedetomidine, alfentanil, and remifentanil be safely used?** Multiple medications have been used for sedation and analgesia — all with varying levels of success and risk. The following agents are determined to be safe in the patient populations described below.

LEVEL A

- *ketamine* (children)
- *propofol* (adults and children)

LEVEL B

- *etomidate* (adults)
- *ketamine and propofol combination* (adults and children)

LEVEL C

- *ketamine and alfentanil* (adults)
- *etomidate* (children)

Notes: Patients receiving alfentanil may require more stimulation to maintain respirations during procedures. The combination of propofol and ketamine allows for decreased doses, and reduces the side effect profiles of both medications.

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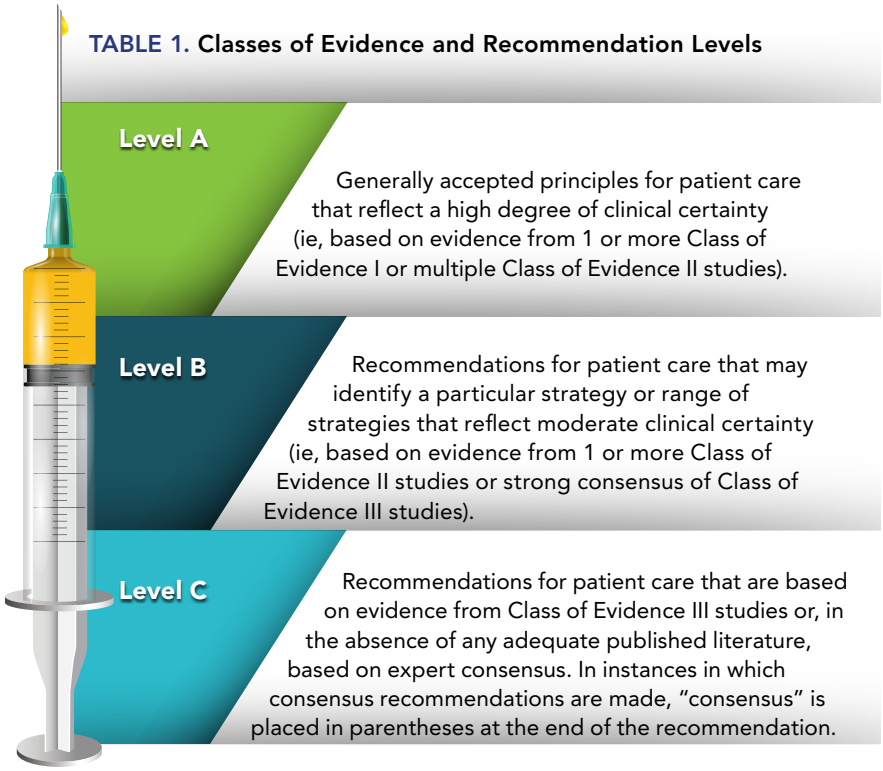
KEY POINTS

- Sedation should never be delayed based on fasting time.
- Capnography can help detect hypoventilation and apnea.
- An additional provider (other than the individual performing the procedure) should be in the room to monitor any patient undergoing sedation; the minimum number of personnel needed is unspecified.
- Multiple agents have been proven safe in procedural sedation and analgesia.

TABLE 1. Classes of Evidence and Recommendation Levels



There are certain circumstances in which the recommendations stemming from a body of evidence should not be rated as highly as the individual studies on which they are based. Factors such as heterogeneity of results, uncertainty about effect magnitude and consequences, and publication bias, among others, might lead to such a downgrading of recommendations.

Clinical Practice: Community-Acquired Pneumonia

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Wunderink RG, Waterer GW. *N Engl J Med.* 2014;370(6):543-551.

Community-acquired pneumonia (CAP) is a leading cause of death in the United States. A combination of infectious symptoms (eg, fever, chills, or leukocytosis), respiratory signs, and a new infiltrate on chest x-ray are indicative of the disease. The diagnosis can be more problematic in elderly patients and those with preexisting cardiopulmonary conditions, whose subtle and confounding symptoms (eg, confusion) easily can be misattributed to other causes.

Streptococcus pneumoniae and atypical bacteria, including mycoplasma, chlamydophila, and legionella, remain the most common pathogenic culprits of CAP. Recommended oral antibiotic regimens for outpatient treatment include macrolides, doxycycline, and fluoroquinolones. When administering these medications, local resistance patterns and a patient's recent antibiotic use should be considered.

The Infectious Diseases Society of America and the American Thoracic Society recommend treatment with a respiratory fluoroquinolone (levofloxacin or moxifloxacin) or a second- or third-generation cephalosporin taken in combination with a macrolide.

Empiric broad-spectrum coverage for *Pseudomonas aeruginosa* and methicillin-resistant Staphylococcus aureus (MRSA) is recommended for patients with health care-associated pneumonia (HCAP). However, specific criteria for managing this disease remain controversial, and researchers caution against the excessive use of broad-spectrum antibiotics.

Antibiotics should be administered as soon as the diagnosis is made, unless the patient is in shock; in such cases, they should be given at the first sign of hypotension. Patients with CAP should be treated for 5 to 7 days; there is no evidence that a prolonged antibiotic course will lead to better outcomes in immunocompetent patients.

Clinical decision tools such as the CURB-65 severity score can help emergency physicians make informed decisions about a patient's disposition. Despite its popularity and ease of use, however, CURB-65 has not been well validated.

Special attention should be paid to abnormal laboratory test results such as blood urea nitrogen, leucopenia, thrombocytopenia, and elevated lactate. These values should be weighed against any preexisting conditions (eg, tachypnea, hypotension, confusion) when determining whether to admit or discharge a patient home. It is important to note that these decisions can have substantial effects on clinical outcomes.

KEY POINTS

- The patient's clinical picture should be assessed in combination with laboratory test results to help guide the disposition (eg, discharge home, or admit to an inpatient floor or ICU).
- Outpatient CAP treatment should include antibiotics with coverage for *Streptococcus pneumoniae* and atypical pathogens (eg, macrolides).
- Inpatient CAP coverage should include a respiratory fluoroquinolone or a second- or third-generation cephalosporin plus a macrolide.
- A diagnosis of health care-associated pneumonia identifies patients at risk for drug-resistant organisms such as *Pseudomonas* or MRSA; however, the protocols for managing HCAP remain controversial.
- Community-acquired MRSA is increasingly common; antibiotics that suppress exotoxins (eg, linezolid or clindamycin + vancomycin) are indicated.

Hyperglycemic Crisis

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Van Ness-Otunnu R, Hack JB. *J Emerg Med.* 2013;45(50):797-805.

Two distinct clinical entities underlie "hyperglycemic crisis" — diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic syndrome (HHS). Although both cause diffuse metabolic derangement and severe dehydration, they have completely different patho-physiologic origins. The incidence of these syndromes will increase concomitantly with the escalating number of patients with type 1 and type 2 diabetes. (In 2010, there were 26 million people in the US living with type 2 diabetes; and the incidence of type 1 diabetes has grown, particularly in children younger than 5 years.)

The diagnostic criteria for DKA include a blood glucose level greater than 250 mg/dL, moderate ketonuria or ketonemia, arterial pH of 7.3, and bicarbonate less than 15 mEq/L. The criteria for HHS are altered sensorium, a glucose level greater than 600 mg/dL, minimal or no ketonemia, serum osmolality greater than 320 MOsm/kg, arterial pH higher than 7.3, and a bicarbonate greater than 15 mEq/L.

DKA, which is characterized by symptoms that have been present for hours to days, often is precipitated by some other pathologic event. HHS, on the other hand, is more insidious and evolves over days to weeks. Ketoacidosis can occur with normal or minimally elevated glucose levels — an entity that comprises about 10% of these cases. The presence of acidemia should always raise suspicion for a toxidrome.

Since severe dehydration is present in all patients with either syndrome, hydration is the first therapeutic intervention; infusions should start at 20 mL/kg/hr, with a goal of achieving urine output of 1 mL/kg/hr.

Electrolyte perturbations are ubiquitous in these syndromes, with both sodium and potassium being elevated or decreased. An ECG is a quick and reliable way to assess for severe hyperkalemia. The patient's potassium status should be confirmed by laboratory testing before the initiation of insulin therapy, which can exacerbate hypokalemia. If hypokalemia is confirmed, potassium should be restored to at least 3.3mEq/L prior to insulin administration.

An IV infusion of insulin is standard therapy (regular insulin at 0.14 units/kg/hr); there appears to be no difference in outcomes between bolus or intravenous therapies. Glucose levels should be measured hourly; if a 10% reduction in serum glucose has not been achieved after the first hour, additional insulin should be given (a bolus of 0.14 units/kg), and the infusion should be continued.

Subsequent care should be rendered according to a hospital-wide protocol, which will guide the continued administration of IV fluids (both amount and constituents) and insulin therapy based on blood glucose levels. The same rule applies to the management of potassium and sodium derangements. These treatment recommendations apply to both DKA and HHS.

KEY POINTS

- Type 1 diabetes, and — consequently — DKA are increasing in young children, and should be included in the differential diagnosis for a newly ill child. A finger stick glucose, an IV infusion of saline, and an ECG are the first important steps in diagnosis and treatment. If severe signs of hyperkalemia are absent on ECG, insulin therapy should be withheld until the patient's serum potassium level is known and significant deficits are corrected.
- An intravenous infusion of insulin is the standard treatment.
- HHS should be considered in any elderly patient with altered mental status.
- Institutional guidelines should be in place for the management of elevated glucose, including protocols for fluid administration, insulin therapy, and electrolyte derangement.

Bleeding and Coagulopathies in Critical Care

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Hunt BJ. *N Engl J Med.* 2014;370(9):847-59.

Effective management of bleeding is complex and requires an accurate diagnosis of the underlying pathology, which can be challenging to confirm in the emergency department. Although there is a paucity of literature to guide these strategies, a peripheral blood smear and panel of tests — when coupled with a thorough clinical examination — can help pinpoint the etiology of a coagulopathy.

The optimal management of **major bleeding** remains under investigation, but current practice includes the use of fresh frozen plasma (FFP) and packed red blood cells (at a 1:1 or 1:2 ratio). Supplementation of fibrinogen to 1.5-2.0 g/L is recommended; however, this guideline has not been explored. Research supports the use of tranexamic acid (TXA) within 3 hours of a trauma; it does not appear to increase the risk of thrombotic events.

The prophylactic use of FFP to correct coagulation abnormalities prior to **invasive procedures** is not recommended. As long as the prothrombin time ratio (PT) is ≤1.5 and direct pressure is applied, a central line or arterial catheter can be placed.

Disseminated intravascular coagulopathy (DIC) is an acquired syndrome that includes diffuse activation of the clotting cascade, which can result in bleeding, clotting, or both. The most common cause of DIC in the critical care setting is sepsis. Because DIC is a consumptive coagulopathy, these patients' platelet counts will be low with a commensurate rise in PT, activated partial thromboplastin time (aPTT), and fibrin degradation products.

Bleeding in a patient with DIC should prompt a platelet (PLT) transfusion to >50,000/mm^3, FFP transfusion until PT and aPTT are <1.5 times normal, and fibrinogen until blood levels are >1.5 g/L. Anti-fibrinolytic agents should not be given; the use of heparin is controversial.

Thrombocytopenia (TCP) has many etiologies, including decreased PLT production, destruction, or sequestration in the spleen. An otherwise stable, thrombocytopenic patient who is not bleeding requires a PLT transfusion when levels are <10,000/mm^3. The threshold can be decreased to 5,000/mm^3 for a non-bleeding patient with chronic PLT production failure.

An actively bleeding, thrombocytopenic patient should be transfused to a threshold of 50,000/mm^3; a patient who has a combined hemostatic deficiency, CNS bleeding, or planned neurosurgery should be transfused to ≥100,000/mm^3. However, there is a lack of data to support these guidelines.

Thrombotic thrombocytopenia purpura (TTP) and drug-induced TCP require urgent intervention. The mainstay treatment for TTP is plasmaphoresis. If unmanaged, the disorder poses a mortality rate of 90%. Drug-induced TCP is transient; removal of the offending agent is required.

Liver disease decreases the production of coagulation factors. This is balanced by a decreased production of anticoagulants; thus, patients are not coagulopathic, despite abnormal laboratory values. Additionally, cholestatic liver disease results in a decreased uptake of lipid-soluble vitamin K; supplementation is recommended.

Renal disease results in anemia and the accumulation of uremic toxins, which contribute to PLT dysfunction. Dialysis, erythropoietin, cryoprecipitate, conjugated estrogen, desmopressin, and TXA can play a role in the treatment of renal disease-associated coagulopathy.

Significant bleeding caused by **antithrombotic therapy** can be hard to treat because many of these drugs cannot be reversed with a specific agent. Bleeding triggered by aspirin, clopidogrel, prasugrel, or ticagrelor can be controlled with PLT transfusions. Protamine can reverse heparin-induced anticoagulation.

Vitamin K and prothrombin complex concentrate can reverse the effects of vitamin K antagonists (warfarin). Many new, oral antithrombotic drugs (eg, dabigatran and rivaroxaban) do not have specific antidotes; prothrombin complex concentrate may be the most effective treatment.

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KEY POINTS

- Many coagulopathies can't be confirmed with a specific diagnostic test; clinicians must rely on a thorough history, physical examination, and laboratory results.
- TXA should be administered when a massive transfusion is expected, ideally within 3 hours of the injury.
- There is a lack of data to support transfusion goals in thrombocytopenia.