Critical decisions in emergency medicine

THE 2020 LLSA LITERATURE REVIEW

The LLSA Literature Review

Synopses of articles from ABEM's 2020 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.

Since November 2019, each monthly issue of *Critical Decisions* has provided a summary of one of the articles from ABEM's 2020 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/cdem**.

Best wishes,

Andrew J. Eyre, MD, Section Editor
Harvard Affiliated Emergency Medicine Residency
Brigham and Women's Hospital

Michael S. Beeson, MD, MBA, FACEP
Northeastern Ohio Universities

IN THIS ISSUE

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Critical decisions in emergency medicine

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Idarucizumab for Dabigatran Reversal

By Rachel Gartland, MD; and Michael E. Abboud, MD
Department of Emergency Medicine,
University of Pennsylvania, Philadelphia
Reviewed by Andrew Eyre, MD, MHPEd

Gottlieb M, Khishfe B. Idarucizumab for the reversal of dabigatran. *Ann Emerg Med.* 2017 May;69(5):554-558.

The emergent reversal of anticoagulation is becoming increasingly complex as more pharmaceutical options become available. Dabigatran (ie, Pradaxa), a relatively newer anticoagulant, is a direct thrombin inhibitor. When dabigatran was released in 2010, however, there was no known reversal agent for the medication. As such, proposed treatments for life-threatening bleeding in patients taking the drug included the administration of fresh frozen plasma and factor VII, hemodialysis, and supportive care. In 2015, the FDA approved idarucizumab (ie, Praxbind), a monoclonal antibody specific to the thrombin-binding site on dabigatran, for the reversal of dabigatran-associated anticoagulation.

Idarucizumab binds to dabigatran with 350 times greater affinity than it does to thrombin, making it an excellent reversal agent. Studies show that the elevations in multiple coagulation parameters (eg, activated partial thromboplastin time and activated clotting time) seen with dabigatran therapy completely normalize with the administration of idarucizumab.

Importantly, these values do not overcorrect, which helps avoid the hypercoagulable states often associated with other medications and products used for anticoagulation reversal. In addition, the adverse effects reported in patients using idarucizumab (ie, headaches and skin and infusion site irritation) are mild and well tolerated.

Idarucizumab also works rapidly and wears off quickly, with a half-life of less than 1 hour in healthy patients. Both dabigatran and idarucizumab are renally excreted; so, while the reversal of dabigatran is just as fast and effective in patients with renal disease, clearance of these medications may be decreased. Nevertheless, restarting dabigatran therapy 24 hours after the administration of idarucizumab leads to the reinitiation of effective anticoagulation in those without renal disease, regardless of age. Subsequent doses of idarucizumab or other hemostatic agents may be required for patients who require prolonged anticoagulation reversal.

While idarucizumab is a promising albeit expensive antidote for patients who require dabigatran reversal, clinically relevant outcomes, such as clinical hemostasis and overall mortality (compared to placebo), have not been clearly described. Although the true utility of the drug has yet to be fully elucidated, its timely use in specific patient populations is advised.

KEY POINTS

- Dabigatran is a direct thrombin inhibitor. Idarucizumab, a monoclonal antibody specific to dabigatran's thrombin-binding site, was developed as a dabigatran reversal agent.
- Idarucizumab has a short half-life and a high affinity for dabigatran, leading to the fast, complete correction of dabigatran-induced anticoagulation. This short half-life also enables the early reinitiation of dabigatran.
- Idarucizumab is generally well tolerated and has few adverse effects. In particular, it does not cause hypercoagulability, a complication often seen with other reversal techniques.
- It is not yet known if the fast and complete normalization of coagulation and laboratory parameters leads to more clinically relevant outcomes.

Opioid-Prescribing Policies

By **Andrew Mittelman, MD;** and **Laura Welsh, MD**
Department of Emergency Medicine,
Boston University, Massachusetts
Reviewed by **Andrew Eyre, MD, MHPEd**

Osborn SR, Yu J, Williams B, Vasilyadis M, Blackmore CC. Changes in provider prescribing patterns after implementation of an emergency department prescription opioid policy. *J Emerg Med.* 2017 Apr;52(4):538-546.

The misuse of opioids, both prescription and illicit, is responsible for an estimated 950,000 emergency department visits every year and has become the leading cause of accidental death in the United States. Many patients who seek treatment for opioid abuse report obtaining their prescriptions from the emergency department. In light of this growing epidemic of addiction and death, the development and implementation of effective opioid-prescribing policies is of paramount importance.

A 7-year, single-center study evaluated the prescribing patterns of 34 urban emergency clinicians who were instructed to adopt the *Washington Emergency Department Opioid Prescribing Guidelines*. The protocol was designed to reduce the number and quantity of opioid prescriptions issued for chronic noncancer-related pain, encourage collaboration between regional emergency departments and a single primary care prescriber, and support routine screening for risk factors that may suggest prescription misuse.

The opioid-prescribing guidelines were universally adopted by all participating emergency department prescribers and presented to patients in the form of fliers and placards. The initiative was reinforced using a series of educational initiatives, including departmental lectures, continuing education, and individual prescriber feedback based on pharmacy data.

The new policy led to a 39.6% decrease in the number of opioid prescriptions issued in the emergency department. The largest decreases were noted for younger patients (18-49 years old) and those diagnosed with musculoskeletal complaints, such as low-back or joint pain. Oxycodone saw the biggest reduction in prescriptions, but the effect also extended to hydrocodone, hydromorphone, and codeine. The number of pills prescribed also decreased 14.8% following the intervention. This decrease in both the number and size of opioid prescriptions was sustained during the 2.5-year follow-up period.

It is important to understand that US laws require emergency clinicians to evaluate patients' pain, but such assessments do not mandate the prescription of opioids. However, it is worth noting that the adoption of a stricter opioid-prescribing policy may lead some patients to seek care at other hospitals. Furthermore, such interventions may lead to inappropriate or inadequate pain management.

Additional studies are needed to determine the benefits and possible complications associated with the adoption of explicit opioid-prescribing guidelines.

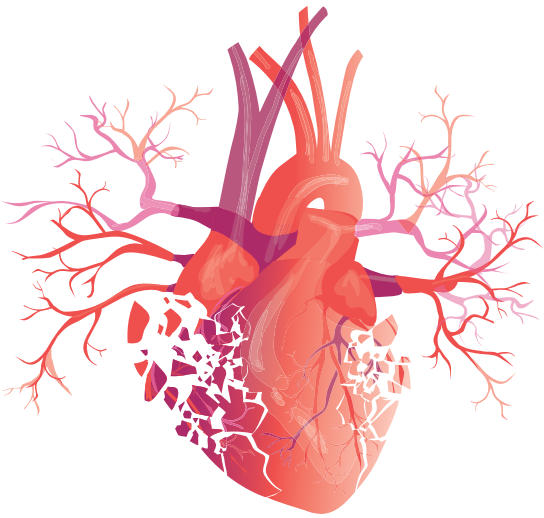
KEY POINTS

- Opioid misuse is responsible for an estimated 950,000 emergency department visits annually.
- With the adoption of formalized opioid-prescribing guidelines, the number of opioid prescriptions in the participating emergency department decreased, as did the total number of pills provided.
- The reduction in opioid prescriptions was sustained during the study's 2.5-year follow-up period.
- It is unknown whether a decreased opioid prescription rate is clinically appropriate.

Acute Myocardial Infarction

By David Schaffer, MD; and Andrew Eyre, MD, MHPEd
Brigham and Women's Hospital, Boston, Massachusetts

Anderson JL, Morrow DA. Acute myocardial infarction.
N Engl J Med. 2017 May 25;376(21):2053-2064.



Despite a decrease in hospitalizations for acute myocardial infarctions (MIs) over the past 40 years, ischemic heart disease remains a leading cause of global disease burden. While MIs can be separated into many different categories based on the underlying pathogenesis of each case, this review article highlights the presentation and current management of type I MIs, which are caused by partial or complete occlusion by coronary artery thrombosis.

For patients who present with symptoms that raise concern for acute coronary syndrome (ACS), the immediate goal is to identify those with ECG evidence of an ST-elevation MI (STEMI). The American Heart Association recommends obtaining an ECG within 10 minutes of the patient's arrival (Class I, Level C recommendation).

Coronary reperfusion is the immediate treatment goal for patients with STEMI. Primary percutaneous coronary intervention (PCI) — angioplasty and stenting — is preferred over IV fibrinolysis with a goal of initiating PCI within 90 minutes from the first medical contact. If PCI cannot be performed within 120 minutes, fibrinolysis should be initiated if not contraindicated (Class I, Level A recommendation). These patients should also be considered for transfer to a PCI-capable facility within 24 hours.

Patients with STEMI should be initiated on dual antiplatelet therapy (DAPT) as early as possible, provided no contraindications exist. In such cases, treatment with ticagrelor or prasugrel is preferred; clopidogrel can be administered to patients undergoing fibrinolysis.

Anticoagulation is also an important part of the early management of STEMI; options include unfractionated heparin (first line), enoxaparin, and bivalirudin.

Serial troponin measurements have long been used to differentiate non-STEMIs (NSTEMIs) from unstable angina and non-ACS chest pain. The more recent introduction of high-sensitivity troponins has enabled clinicians to rule out MIs in a shorter period of time. However, these highly sensitive tests sacrifice specificity; changes in serial troponin levels are commonly used to diagnose NSTEMI, as many patients have elevated troponin at baseline or due to other conditions.

Any patient with an NSTEMI should also be initiated on DAPT and anticoagulation; however, the choice of antiplatelet and anticoagulant agents depends on the anticipated management strategy and bleeding risk factors. For example, enoxaparin sodium may be a good option for patients not undergoing invasive management, but it should be avoided in patients at high risk of bleeding.

Research findings regarding traditional treatments for ACS have been mixed. Supplemental oxygen is only recommended for patients who are hypoxemic (oxygen saturation <90%) or are in respiratory distress. Although debate continues about the use of beta blockers, they are typically initiated within 24 hours and should be avoided in patients with risk factors for cardiogenic shock. Sublingual nitroglycerin, which remains a staple for the treatment of chest pain, can be administered intravenously for persistent chest pain, heart failure, and uncontrolled hypertension.

KEY POINTS

- Patients with symptoms concerning for ACS should receive an ECG within 10 minutes of arrival.
- The goal time for PCI is less than 90 minutes when managing a STEMI. If PCI cannot be performed within 120 minutes, fibrinolysis should be given if not contraindicated.
- Patients with a STEMI or NSTEMI should be initiated on DAPT and anticoagulation; medications should be selected based on several patient-specific factors, including the type of MI, bleeding risk, and potential for PCI or thrombolysis.
- Supplemental oxygen is not recommended for the treatment of MI unless the patient is hypoxemic or in respiratory distress.

Acute Diarrheal Infections

By Kara Yeung, MD; and Andrew Eyre, MD, MHPEd
Harvard Medical School, Boston, Massachusetts

Acree M, Davis AM. Acute diarrheal infections in adults. *JAMA*. 2017 Sep;318(10):957-958.

Acute diarrhea is defined as persistent, loose, watery stools that can last for up to 14 days. Approximately 179 million cases of acute gastroenteritis are diagnosed each year in the United States alone, including 47.8 million cases of foodborne illness. The most common cause of gastroenteritis is norovirus, which accounts for 26% of diarrhea-related emergency department complaints; 90% of deaths due to norovirus occur in patients aged 65 years or older.

Published in 2016 by the American College of Gastroenterology, the following guidelines provide an empirical, algorithmic approach to gastroenteritis. The protocol is based on:

- Presence of grossly bloody stools (dysentery)
- Severity of illness (moderate: forced change in activities; severe: total disability due to diarrhea)
- Presence of fever (≥38.3°C [101°F])
- History of traveling abroad

The initial treatment of acute diarrhea includes oral rehydration, especially when managing high-risk populations, such as the elderly, and travelers who present with severe, watery diarrhea. Antibiotics should be reserved for travelers with moderate-to-severe diarrhea and those in whom a bacterial infection (ie, dysentery and/or fever) is likely. Antibiotics should not be offered to patients with suspected community-acquired or mild travel-associated diarrhea, given that the etiology of these diseases is usually viral in nature. The use of loperamide in conjunction with antibiotics can rapidly reduce the number of diarrheal stools more effectively than bismuth subsalicylate alone.

The prophylactic use of antibiotics should also be reserved for patients with recent travel outside of the US and Europe, those who are at high risk of travel-associated diarrhea, and patients whose illness may cause potentially serious complications or critically affect the intended purpose of travel. Antibiotic administration has its own risks and benefits, including an increased risk of *Clostridium difficile* colitis, particularly with the use of fluoroquinolone. The improper use of antibiotics may lead to more severe and resistant infections.

The net benefits of polymerase chain reaction (PCR) testing have yet to be established in patients with acute diarrhea; however, PCR studies may be faster and more sensitive than traditional stool tests. Dysentery, moderate-to-severe disease, and symptoms that last longer than 7 days should be evaluated using culture-independent methods, at least as an adjunct to traditional diagnostic approaches. While PCR is faster and more sensitive than traditional stool testing, it cannot distinguish between live and dead pathogens; additionally, no associated antibiotic sensitivity panel is provided. Therefore, relying on culture-independent diagnostics alone may subject patients to needless medication and prevent clinicians from tailoring antibiotic regimens to causative and resistant strains.

KEY POINTS

- Antibiotic therapy is not recommended for the treatment of routine acute diarrheal infections or mild traveler's diarrhea.
- Febrile patients with disabling traveler's diarrhea should be treated with antibiotics.
- When prescribing antibiotics for traveler's diarrhea, loperamide can be used as an adjunct to decrease the duration of symptoms and improve the likelihood of a cure.
- Further study is needed regarding the effect of PCR-based assays on patient outcomes and the efficacy of antibiotic therapy.

Child Abuse

By Sarah Ford Bogdanowicz, MD, LCDR; and Daphne Morrison Ponce, MD, LCDR
Naval Medical Center, Portsmouth, Virginia;
and the University of Michigan Health System, Ann Arbor, Michigan
Reviewed by Andrew Eyre, MD, MHPEd

Berkowitz CD. Physical abuse of children. *N Engl J Med.* 2017 Apr;376(17):1659-1666.

Approximately 700,000 cases of child abuse and neglect are reported each year in the United States. It is essential for emergency physicians to not only recognize abuse as a possible cause of trauma but also use sentinel injuries as an opportunity to intervene. Care for at-risk children requires coordination between the medical and legal systems. Physicians are legally mandated to report any case in which there is a reasonable suspicion of abuse.

Abusive Head Trauma

The signs and symptoms of abusive head trauma can be vague, particularly in infants and young children, who may present with nonspecific signs and symptoms, including a brief unexplained event, apnea, loss of consciousness, altered mental status, seizures, vomiting, decreased tone, and poor feeding. "Abusive head trauma" has replaced "shaken baby syndrome" as the proper term for intracranial injuries caused by abuse.

In general, short falls (from <1.5 m) are unlikely to cause severe intracranial trauma. In addition to cross-sectional brain imaging (CT or MRI), the evaluation of suspected abusive head trauma should include a fundoscopic examination by a pediatric ophthalmologist, as retinal hemorrhages are reported in approximately 85% of these patients. A study of intracranial injuries in children younger than 3 years old demonstrated a strong positive-predictive value for abusive head trauma in those with apnea, seizures, retinal hemorrhages, head and neck bruising, rib fractures, and long-bone fractures.

Cutaneous and Intraoral Findings

The patient's skin should be carefully evaluated for bruises, especially on the face, ears, neck, and torso; it is also important to assess for intraoral lesions, such as torn frenula. It may be helpful to remember the adage, "Those who don't cruise rarely bruise." In other words, bruising is an uncommon finding in nonambulatory patients. Patterned bruises and burns, such as those that mimic a hand, belt, or hot object or demonstrate immersion in scalding water, may herald child abuse.

Abdominal Trauma

Inflicted abdominal trauma is less common than abusive head trauma and is more likely to occur in older toddlers. These injuries carry a high mortality risk due to delays in diagnosis and treatment. Hepatic and pancreatic enzymes should be measured if physical abuse is suspected. If enzymes are elevated or if GI symptoms are present, a CT with IV contrast is the appropriate imaging study, as it is more sensitive than ultrasound.

Fractures

Fractures can be indicative of physical child abuse, especially rib fractures; classic metaphyseal lesions ("chip" fractures or "bucket handle" fractures); scapula, sternum, or acromion fractures; and any fracture in nonambulatory children. The developmental ability of the child should be considered as it relates to the reported mechanism of injury. Skeletal surveys are recommended for any child younger than 2 years in whom physical abuse is suspected.

Medical Screening

When evaluating a patient for suspected abuse, it is important to screen for medical diagnoses that may predispose the child to easy bruising or fractures, such as coagulopathies, metabolic bone diseases, and genetic conditions.

KEY POINTS

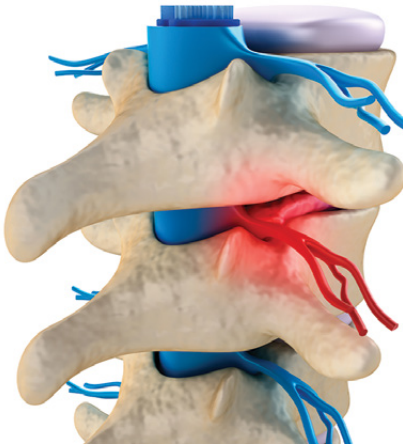
- Consider abusive head trauma in infants and young children with nonspecific symptoms, such as apnea, loss of consciousness, or vomiting.
- When evaluating a child for suspected abuse, it is vital to conduct a thorough history, physical examination, and social evaluation. Additional diagnostic testing may include fundoscopy, brain imaging, a skeletal survey, and measurements of pancreatic and hepatic enzymes.
- Physicians are legally mandated to report any case in which there is a reasonable suspicion of abuse to their local child protective services agency.

DISCLOSURES

The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Dept. of the Navy, Dept. of Defense, or the US Government. We are military service members.

This work was prepared as part of our official duties. Title 17 U.S.C. 105 provides that "Copyright protection under this title is not available for any work of the US Government." Title 17 U.S.C. 101 defines a US Government work as a work prepared by a military service member or employee of the US Government as part of that person's official duties.

Acute Spinal Cord Compression



By Kathryn A. Dasburg, MD; and Nicholas G. Maldonado, MD, FACEP
University of Florida College of Medicine, Gainesville

Reviewed by Andrew Eyre, MD, MHPEd

Ropper AE, Ropper AH. Acute spinal cord compression. *N Engl J Med.* 2017 Apr;376(14):1358-1369.

Acute spinal cord compression is a rare, limb-threatening process that is often heralded by a common, often-benign chief complaint: back pain. The potentially devastating neurological disorder typically arises from diseases that originate in the spinal column and eventually leads to narrowing of the spinal canal. In addition, such etiologies can affect spinal-stabilizing structures (ie, vertebrae, intervertebral discs, ligaments, and facet joints), predisposing patients to subluxation of the vertebrae (ie, spondylolisthesis).

Instability that narrows the spinal canal enough to risk cord damage is generally an indication for spinal fixation via fusion of adjacent vertebrae. Common conditions that result in spinal cord compression through space-occupying lesions or damage to spinal-stabilizing structures include trauma, tumors, epidural abscesses, and epidural hematomas.

Acute spinal cord compression should be suspected in patients who present with localized neck or back pain in association with limb weakness, urinary incontinence or retention, fecal incontinence, or a loss of sensation. The physical examination should focus on spinal palpation and percussion, strength and reflex assessments, and an evaluation of sensory function.

Signs suggestive of acute spinal compression include localized tenderness to percussion, flaccid paralysis, a loss of reflexes, and an abnormal sensory level. Hyperreflexia and a positive Babinski sign, which suggest intrinsic spinal cord disease, may be absent in patients with cord compression syndromes.

Treatments differ based on the etiology of each case, but a neurosurgical consultation is paramount, as early surgical decompression can preserve neurologic function. Cardinal features of common spinal cord syndromes and the main characteristics of conditions that result in spinal cord compression are summarized in Tables 1 and 2.

KEY POINTS

- Acute spinal cord compression should be suspected in patients with localized neck or back pain plus limb weakness, urinary retention or incontinence, or fecal incontinence, with varying signs of midline tenderness to percussion, extremity weakness or paralysis, loss of reflexes, and sensory deficits.
- Four etiologies that commonly result in acute spinal cord compression are spinal trauma, tumors, epidural abscesses, and epidural hematomas.
- A history of trauma, known or increased risk of malignancy, fever or other systemic symptoms (particularly in association with IV drug use or immunosuppression), or anticoagulation in patients with back pain should increase suspicion for spinal cord compression.
- CT is recommended for the evaluation of patients with trauma, and MRI with contrast is the preferred diagnostic test for other causes of back pain. Given the high incidence of multilevel involvement in all etiologies, multiple spinal segments should be imaged.
- The cervical spine is most commonly implicated in cases of trauma; however, the thoracic spine is most commonly to blame for neoplastic or infectious spinal cord compression.
- Treatments differ based on the etiology of each case, but an early neurosurgical consultation is paramount, as early surgical decompression can preserve neurologic function.

TABLE 1. Clinical Syndromes of Acute Spinal Cord Compression

Clinical Syndrome	Spinal Level	Key Features	Motor	Sensory	Reflexes	Babinski Sign	Sphincter Tone
Complete transverse myelopathy	Any	Affects both sides and anterior/posterior spinal cord	Absent below	Absent below	Hyper-	Upgoing	Absent
Hemicord (Brown-Séquard) syndrome	Any	Unilateral on affected side	Absent below	Absent vibration, fine touch, proprioception; contralateral loss of pain and temperature.	Hyper-	Upgoing	Present
Central cord syndrome	Cervical	Hyperesthesia; spared vibration and proprioception	Arm > leg weakness	↓pain and temperature in arms	Variable	Downgoing	Present
Spinal shock	Cervical thoracic	Systemic hypotension	Absent below	Absent below	Areflexia	Absent	Absent
Conus medullaris syndrome	L1-L2	Cord compression at L1-L2	Feet and leg weakness	↓at sacral and lower-lumbar dermatomes	Absent Achilles	Variable	Absent
Cauda equina syndrome	L2-S1	Cord compression at L2-S1; sciatica or radicular pain; urinary retention/ incontinence	Feet and leg weakness	Saddle anesthesia and legs up to groin	Areflexia (absent Achilles ***AND*** patellar)	Diminished or absent	Absent

TABLE 2. Characteristics of Common Etiologies of Acute Spinal Cord Compression

Etiology	Pathologic Mechanism	Assessment	Treatments	Pearls
Traumatic spinal injury	Results from fractured and retropulsed bone fragments, disk herniation, sub-luxation of vertebral bodies, or cord impingement (trauma-associated)	1. Establish anatomic level (lowest segment with normal motor/ sensory function) 2. Grade severity of neurologic deficit† 3. Imaging: CT preferred; MRI for ligamentous injury, disk herniation, or cord edema	• Spine precautions • Maintain goal MAP 85-90 mmHg (may require IVFs and vasopressors) • High-dose steroids no longer recommended • Surgical decompression with open reduction, internal fixation, and spinal fusion	• Up to 20% of traumatic spinal injuries affect more than one level • Injury to cervical segments results in the most significant morbidity, with tetraplegia and respiratory failure
Neoplastic epidural lesion	Results from spinal metastases that extend the from bone into epidural space as well as pathologic fractures with associated retropulsion and cord compression	1. Suspect in patients with back pain and known cancer/risk factors 2. Spinal percussion: midline tenderness warrants workup 3. Imaging: Entire spine should be imaged. MRI with contrast is preferred, but noncontrast MRI is still sensitive in those with contraindications. Use CT myelography if unable to do MRI	• Steroids (eg, dexamethasone 10 mg IV, followed by oral dose every 4-6 hours) • Radiotherapy, radiosurgery (high-dose radiation to a specific area under imaging guidance and contoured to shape of tumor) or palliative radiation • Surgical removal with decompression, internal fixation, and spinal fusion	• Cancers with high incidence of spine metastasis: breast, lung, prostate, renal cell, multiple myeloma, non-Hodgkin's lymphoma • Sarcoma, lymphoma, and neuroblastoma are most common in children • Most common segments: T (60%) > L (25%) > C-spine (15%)
Spinal epidural abscess	Results from mass effect of the expanding abscess within the epidural space, with myelopathy that can be acute, subacute, or chronic	1. Suspect in severe localized back pain with fever or other infectious symptoms 2. Risk factors: diabetes, cancer, immunosuppression, IV drug use, surgical complications 3. Labs: leukocytosis and elevated ESR/C-reactive protein support the diagnosis; obtain blood culture prior to giving antibiotics 4. Imaging: MRI with contrast preferred, with enhancing lesion on T2-weighted sequence	• Antibiotics: MRSA, *Streptococcus*, and Gram-negative coverage: vancomycin ***PLUS*** ceftriaxone ***OR*** cefepime; ceftazidime, meropenem if pseudomonas suspected. Nafcillin ***OR*** oxacillin can be considered for optimal methicillin-susceptible *Staphylococcus aureus* (MSSA) coverage • Surgical evacuation with irrigation, biopsy, and culture	• Hematogenous spread, but only half have identified bacterial infection at a distant site • *S. aureus* most common among a range of bacteria (MSSA = MRSA) • T-spine most often affected; multiple contiguous or noncontiguous levels of the spine can be affected
Spinal epidural hematoma	Results from mass effect of the expanding hematoma within the epidural space	1. Suspect in back pain and anticoagulation/bleeding diathesis or trauma 2. Labs: thrombocytopenia, prolonged PT/INR 3. Imaging: MRI reveals clot isointense on T1 and enhancing on T2-weighted sequence	• Anticoagulation reversal • Platelets for severe thrombocytopenia • Surgical evacuation	• Often spontaneous without any report of trauma

† Most common neurologic injury severity assessment is the American Spinal Injury Association Impairment Scale. **Grade A: *Complete***, no sensory or motor function preserved in segments S4-S5; **Grade B: *Sensory incomplete***, sensory but no motor function below level of injury, including S4-S5; **Grade C: *Motor incomplete***, motor function is preserved below injury level, and more than half of key muscles below the neurologic level have muscle power ≤3; **Grade D: *Motor incomplete***, motor function is incomplete as in Grade C, with muscle power ≥3 for at least half the key muscle functions below the level of injury; **Grade E: *Normal***, sensory and motor function are normal.

Nonpenetrating Eye Injuries in Children

By Robert Grzybowski, MD, LCDR; and Daphne Morrison Ponce, MD, LCDR
Naval Medical Center, Portsmouth, Virginia; and the University of Michigan in Ann Arbor
Reviewed by Andrew Eyre, MD, MHPEd

Root JM, Gupta S, Jamal N. Nonpenetrating eye injuries in children. *Clin Pediatr Emerg Med.* 2017 Mar;18(1):74-86.

There are four guiding principles for managing pediatric ocular trauma: (1) evaluate for coexisting trauma, (2) assess for a ruptured globe, (3) obtain visual acuity measurements, and (4) seek a specialty consultation when appropriate. Consider using adjuncts (eg, a child life specialist, papoose restraints) to facilitate the evaluation. When managing nonverbal patients, clinicians should look for the reflexive contraction of the eyelid, a finding that confirms light perception. If a globe rupture is suspected, shield the eye and consider transferring the child to specialty care; do not instill eye drops.

Corneal Abrasions

Symptoms of corneal abrasions include eye pain, photophobia, foreign body sensations, and tearing. The diagnosis is made by direct visualization during a fluorescein examination. If a vertical pattern is noted, evert the eyelids to evaluate for foreign bodies. Eye patching is no longer recommended. Prophylactic antibiotics are indicated: erythromycin ophthalmic for simple abrasions and fluoroquinolone drops for contact lens users. Ophthalmology follow-up is indicated for large abrasions (>4 mm), contact lens wearers, and symptoms that persist longer than 48 hours. Contact lens wearers should discontinue use until after follow-up.

Ocular Burns

Children are more likely to present with ocular burns from household chemicals. Immediate management consists of pain control and irrigation for at least 30 minutes until pH normalizes (6.8 and 7.4) and remains stable at recheck 30 minutes later. Ophthalmology should be consulted early for severe burns.

Traumatic Hyphema

Hyphema (blood in the anterior chamber) is a sign of severe, blunt ocular trauma. These cases pose a risk of rebleeding. Clinical signs include decreased visual acuity, eye pain with pupillary constriction, and damage to the surrounding ocular structures. Hyphema is graded based on the percentage of anterior chamber filling: grade I (<33%), grade II (33%-50%), grade III (>50%), and grade IV (near or complete filling). Initial management includes measuring intraocular pressure (IOP), elevating the head of the bed, using topical anesthetics, and shielding the eye. Prescribe oral analgesia and antiemetics as needed.

Special consideration should be given to those at risk of secondary hemorrhage, including patients with sickle cell disease (SCD) or trait, hemophilia, and von Willebrand disease. Low-grade hyphema can be managed on an outpatient basis with an ophthalmology referral. Consider an urgent referral for patients with grade III or IV hyphema, risk factors, elevated IOP, early corneal blood staining, visual deterioration, or active bleeding. Acute hospitalization is indicated for secondary hemorrhage, suspected child abuse, hyphema greater than 50%, SCD or trait, and patients at risk of noncompliance.

Orbital Fractures

Carefully examine extraocular movements and surrounding orbital structures for dystopia, enophthalmos, flattening of the nasal complex, and telecanthus. Most orbital fractures are managed conservatively with antibiotics, sinus precautions, nasal decongestants, and outpatient follow-up. Consider deferring CT unless there are signs of severe injury or muscle entrapment.

Subacute Presentations

Unilateral symptoms of aching eye pain, redness, and light sensitivity 24 to 72 hours after injury suggest traumatic iritis or traumatic uveitis. A slit-lamp examination will show cells and flare, and an opthalmology consultation is needed. Traumatic injuries that can result in vision loss include retrobulbar neuritis, choroidal rupture, retinal detachment, and commotion retinae. Any loss of vision requires an ophthalmologic consultation.

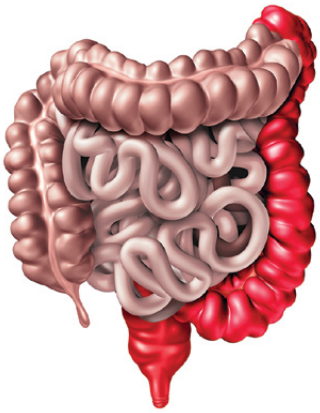
KEY POINTS

- A comprehensive eye examination is essential for evaluating traumatic injuries. Clinical adjuncts can help facilitate the examination in children.
- Visual acuity is the vital sign of the eye. It is imperative to confirm light perception and rule out a ruptured globe and coexisting injuries early.
- An urgent consultation should be sought for any child with a globe rupture, an increase in IOP, changes in visual acuity, or coexisting trauma.
- Consider hospitalization for patients with hyphema greater than 50%, SCD or trait, and when child abuse is suspected.

DISCLOSURES

The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, or the United States Government.

Acute Lower Gastrointestinal Bleeding



By Tareyn N. Morris, MD; and Nicholas G. Maldonado MD, FACEP
University of Florida College of Medicine, Gainesville, Florida
Reviewed by Andrew Eyre, MD, MHPEd

Gralnek IM, Neeman Z, Strate LL. Acute lower gastrointestinal bleeding. *N Engl J Med*. 2017 Mar;376(11):1054-1063.

GI bleeding (GIB) represents the most common cause of hospitalization from GI diseases. Lower GIB (LGIB) accounts for approximately 30% to 40% of these cases. Diverticulosis is the most common culprit (30%-65%), followed by ischemic colitis and hemorrhoids. Although most cases resolve spontaneously without intervention or complications, acute LGIB can be severe and life-threatening.

LGIB is typically evidenced by hematochezia (maroon- or red-colored stool), but it can also manifest as melena (dark, tarry stool) or red blood in cases of brisk upper GI bleeding (UGIB). LGIB is defined as a bleed originating from the colon or rectum. Bleeding that originates between the ligament of Treitz and the ileocecal valve is considered a middle GIB.

A hemodynamic evaluation and focused history are essential when assessing any acute LGIB. Initial questions should focus on the color, amount, frequency, and duration of the bleed. Associated symptoms may elucidate the etiology; for example, unexplained weight loss and changes in stool caliber may suggest colorectal cancer. It is also important to inquire about upper GI symptoms, such as a history of peptic ulcer disease and previous UGIB events. Factors like underlying cardiac, renal, or hepatic disease should also be noted, as they are associated with poorer outcomes. Lastly, the use of NSAIDs, anticoagulants, and antiplatelets should be considered, as anticoagulation may require reversal. A digital rectal examination should be performed, and bedside anoscopy should be considered to assess for hemorrhoidal bleeding.

The initial management of any patient with LGIB begins with IV fluid resuscitation, ideally with crystalloids. The initial workup should include, at minimum, a CBC, coagulation studies, and a type and screen. If an upper GI source is suspected, BUN and creatinine measurements can be helpful. A ratio of BUN to creatinine greater than 30:1 suggests UGIB. A transfusion of packed RBCs should be initiated when managing unstable patients and those with significant active bleeding or a Hgb level less than 7 g/dL. For patients with ischemic cardiovascular disease, a transfusion should be initiated to maintain an Hgb level greater than 9 g/dL.

Colonoscopy is the initial procedure of choice for most patients with acute LGIB. Generally, the test should be performed within 24 hours of presentation and after hemodynamic resuscitation and colon cleansing. Sufficient colon preparation is essential for proper visualization and treatment of the bleed. Hematochezia in the context of hemodynamic instability may represent UGIB; therefore, endoscopy should be considered before colonoscopy.

CT angiography (CTA) and radionuclide technetium 99m-labeled RBC scintigraphy may useful if brisk bleeding and hemodynamic instability persist despite resuscitation efforts, the colonoscopy is nondiagnostic, or endoscopic hemostasis was unsuccessful. If either of these tests yields a positive result, CTA should be performed as soon as possible, as it can provide the location of bleeding and guide therapy.

Methods for endoscopic hemostasis include injections of dilute epinephrine, contact and noncontact thermal devices, and mechanical therapies (clips and band ligation). Surgery may be indicated if endoscopic and radiographic treatment fails. It is important to rule out UGIB as the potential source of an LGIB.

The use of low-dose aspirin before endoscopy has been shown to increase the risk of death. Patients who continue aspirin after the initial bleeding event appear to have a higher rate of rebleeding but a lower risk of serious cardiovascular events and death. Those who take aspirin daily as a secondary cardiovascular prophylaxis should continue taking it. In patients who use dual antiplatelet therapy, guidelines recommend holding the nonaspirin antiplatelet therapy for 1 to 7 days while continuing aspirin without interruption.

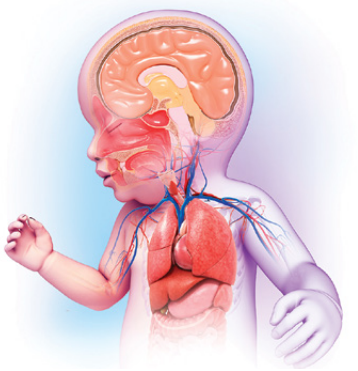
KEY POINTS

- LGIB is defined as bleeding that originates from the colon and rectum; diverticulosis is the most common cause.
- Colonoscopy (ideally <24 hours after arrival) is the initial procedure of choice for most patients with acute LGIB.
- Hematochezia in the context of hemodynamic instability may represent UGIB; therefore, endoscopy should be considered before colonoscopy.
- CTA should be considered for patients with brisk bleeding and hemodynamic instability despite resuscitation.
- Endoscopic therapy includes diluted epinephrine injections, thermal devices, and mechanical therapies (clips and band ligation).
- Low-dose aspirin should be continued in patients who use it as a secondary cardiovascular prophylaxis.

Pediatric Airway and Rapid Sequence Intubation

By LT Chad Thompson, MD; and LCDR Daphne Morrison Ponce, MD
Naval Medical Center Portsmouth, Portsmouth, Virginia, and the University of Michigan, Ann Arbor
Reviewed by Andrew Eyre, MD, MHPEd

Sulton CD, Taylor TR. The pediatric airway and rapid sequence intubation in trauma. *Trauma Reports.* 2017 Nov;18(6).



As with adult patients, the management of pediatric trauma follows a stepwise approach starting with the airway; however, key anatomic differences must be considered when treating children to avoid potentially life-threatening complications.

Airway Anatomy

The pediatric airway consists of three segments: the *supraglottic*, *glottic*, and *intrathoracic* segments. The structure is shorter in length and smaller in diameter that an adult airway, making it harder to visualize; it is also more collapsible, making cricoid pressure less useful. Additionally, the pediatric larynx is anatomically more anterior. These differences, along with a relatively large tongue and occiput, make visualization more difficult in children. It is helpful to note that the cricoid cartilage is the narrowest part of the airway in children younger than 10 years.

Rapid Sequence Intubation

Tracheal intubation is indicated for patients with inadequate oxygenation, inadequate ventilation, a lack of respiratory drive, or a lack of airway protective reflexes. Rapid-sequence intubation (RSI), the simultaneous administration of induction and neuromuscular-blocking agents, enables better airway control. Trauma and respiratory failure are the most common reasons for pediatric airway compromise. All patients should be preoxygenated prior to attempting intubation; when left untreated, hypoxia is a precursor to cardiac arrest. Succinylcholine and rocuronium are appropriate paralytic agents; given its safety profile, rocuronium is preferred. Pretreatment with atropine can be considered for the prevention of bradycardia, but it is controversial.

Airway Equipment

When managing children, remember to use oropharyngeal (OPA) and nasopharyngeal (NPA) airways. OPAs can be inserted directly; the 180-degree rotation is unnecessary and may cause soft-tissue trauma. Avoid NPAs if a basilar skull fracture is suspected. Fit the mask appropriately by measuring from the bridge of the patient's nose to the cleft of the chin. Cuffed endotracheal tubes (ETTs) are preferred over uncuffed ETTs; the familiar ETT size formula for cuffed tubes is **(age ÷ 4) + 3.5**. Length-based tape measurements are useful for appropriate equipment selection. SOAPME is a common mnemonic for remembering the needed supplies: **s**uction, **o**xygen, **a**irway, **p**harmacy, **m**onitors (end-tidal CO_2, ECG, pulse oximetry), and **e**xtra equipment (laryngeal mask airway).

KEY POINTS

- Key anatomic differences make direct visualization harder in pediatric patients.
- Preoxygenation is essential because children are more prone to hypoxia, which can lead to cardiac arrest.
- Appropriately sized equipment is crucial; equations or length-based tapes can be helpful.
- Rocuronium is the preferred paralytic agent for managing pediatric patients.

Avoiding Complications

The Mallampati classification system is helpful for assessing children, but the approach can be limited in uncooperative patients. Patients less than 1 year old and those with facial trauma, blood, vomitus, loose teeth, and secretions are at highest risk for complications. Craniofacial abnormalities (eg, Down syndrome) can hinder visualization. Lastly, cervical spine immobilization disrupts optimal airway alignment.

The first-attempt success rate for emergency physicians in training is about 77%, with mainstem intubation being the most common complication. About 30% of pediatric intubations are in the right mainstem. Subtle head movements can still dislodge the ETT or cause a right-mainstem intubation. The formula for the ETT depth is **tube size x 3**.

Children with blood loss remain normotensive much longer than adults. As when managing any airway in the emergency department, the patient is assumed to have a full stomach, making vomiting and aspiration a higher risk.

DISCLOSURES

The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, or the United States Government.

Necrotizing Soft-Tissue Infections

By Hazar Khidir, MD; and Andrew Eyre, MD, MHPEd
Massachusetts General Hospital, Boston, Massachusetts

Stevens DL, Bryant AE. Necrotizing soft-tissue infections. *N Engl J Med.* 2017 Dec;377(23):2253-2265.

Although necrotizing soft-tissue infections are associated with a spectrum of bacterial etiologies, pathophysiologies, and clinical presentations, they are universally characterized by widespread tissue destruction that can extend from the epidermis to the deep musculature. These pathologies are typically categorized based on the causative bacteria: polymicrobial (type I necrotizing fasciitis), monomicrobial (type II necrotizing fasciitis), or clostridial myonecrosis (gas gangrene).

Classification

Type I necrotizing fasciitis occurs more commonly in elderly patients, those with predisposing conditions like diabetes, and patients who sustain mucosal breaches (eg, colonic, urologic, or gynecological conditions). Type II necrotizing fasciitis is most commonly caused by group A *Streptococcus* or MRSA and may occur in patients of any age group and without underlying illness.

Clostridial myonecrosis is most commonly caused by penetrating skin injuries that compromise the blood supply to soft tissue, creating an anaerobic environment that is ideal for spore germination and bacterial proliferation. Although necrotizing soft-tissue infections are often associated with breaches of the skin (eg, traumatic injuries, surgical procedures, or insect bites), they can also occur after spontaneous, nonpenetrating trauma (eg, muscle strains, contusions).

The pathophysiologic mechanism of tissue necrosis in these patients is thought to be secondary to bacterial seeding in the muscle caused by transient bacteremia from the nasopharynx. Both clostridial and group A streptococcal infections inflict tissue injury through the production of potent bacterial exotoxins.

Diagnosis

The diagnosis of necrotizing soft-tissue infections is often delayed due to a failure to detect early symptoms (*Figure 1*). Classic manifestations of necrotizing fasciitis include soft-tissue edema (75%), erythema (72%), severe pain (72%), tenderness (68%), fever (60%), and skin bullae or necrosis (38%). Factors that differentiate necrotizing fasciitis from cellulitis include recent surgery, hypotension, skin necrosis, hemorrhagic bullae, and pain that is out of proportion to the patient's clinical signs.

Patients with group A streptococcal necrotizing fasciitis often present with fever, rapidly escalating pain, and GI symptoms, including anorexia and diarrhea. Systemic signs of clostridial myonecrosis include profound hypotension, diffuse capillary leaks resulting in hemoconcentration (Hct 50%-80%), and a marked leukemoid reaction (WBC count 50,000-150,000 per mm^3).

Although surgical inspection is the ultimate strategy for diagnosing necrotizing soft-tissue infections, tests that are useful for detecting these pathologies include surrogate laboratory markers, imaging, and Gram stain and culture.

The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) scoring system uses the patient's total WBC count (>15,000), Hgb (<13.5), sodium (<135), glucose (>180), creatinine (>1.6), and C-reactive protein (>140) levels to distinguish between mild infections and necrotizing fasciitis. The LRINEC has a high negative-predictive value, making it more useful for ruling out serious pathologies.

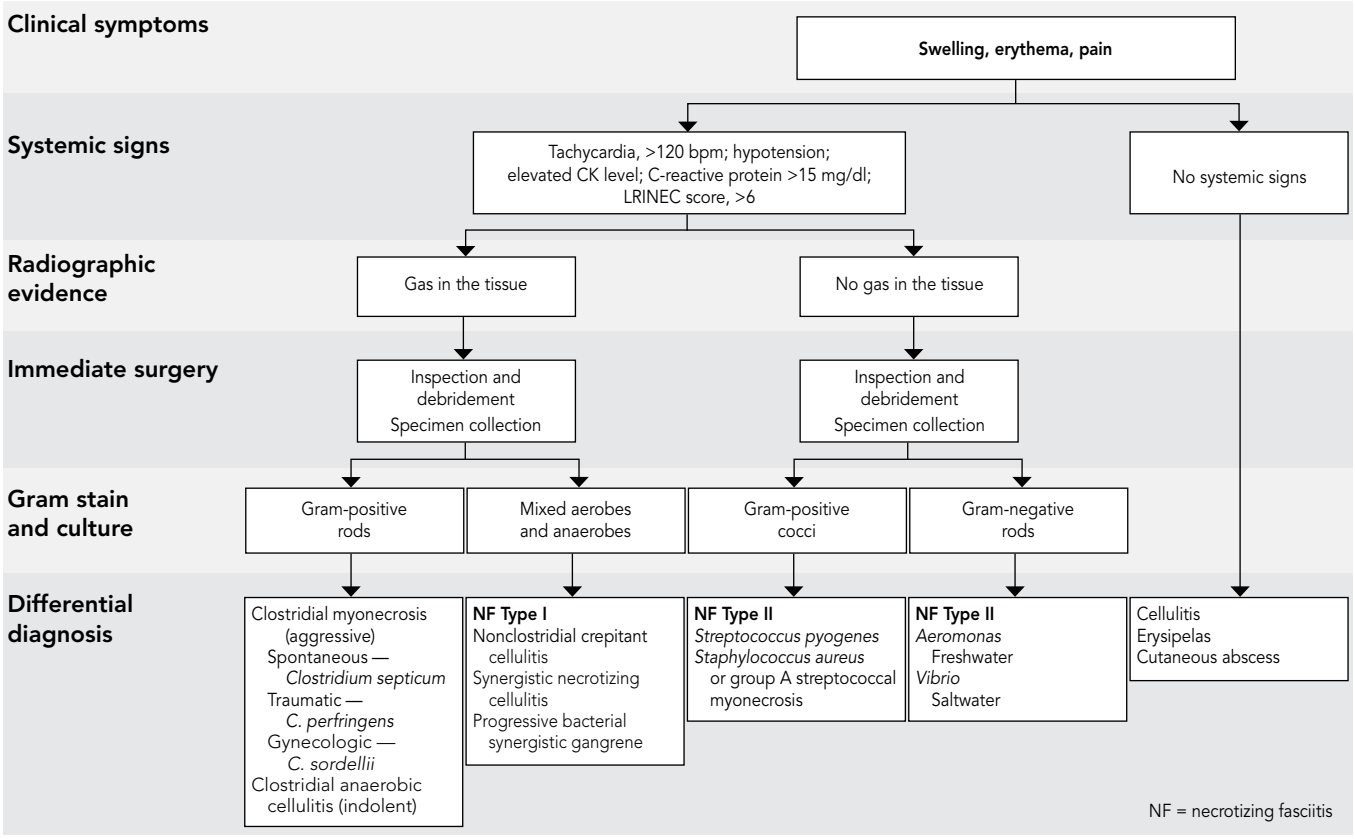
Gas gangrene and type I necrotizing fasciitis are usually evidenced by the presence of gas on imaging studies; however, soft-tissue swelling is typically seen in cases of type II necrotizing fasciitis. A surgical biopsy and Gram stain are critical to determine the bacterial cause of infection and guide antibiotic treatment.

Pitfalls

The most common pitfalls in the diagnosis of necrotizing soft-tissue infections include:

- **Absence of fever:** Patients with clostridial myonecrosis are typically afebrile. In other instances, fever may be masked by the use of NSAIDs.
- **Absence of cutaneous manifestations:** Necrotizing soft-tissue infections can occur secondary to nonpenetrating

FIGURE 1. Algorithm for the Diagnosis of Necrotizing Infections



injuries, including muscular strain. These patients do not present with visible skin findings until they reach severe stages of illness.

- **Absence of pain or attributing pain to another etiology:** Pain may not be detected in patients with altered mental status or diabetes-related neuropathy. Necrotizing soft-tissue infections can occur after traumatic penetrating injuries or routine surgical procedures. In these patients, severe pain is often misattributed to the initial trauma or procedure.
- **Attributing systemic symptoms to other causes:** Early symptoms of some necrotizing soft-tissue infections are often nonspecific and include GI symptoms, myalgias, and fatigue.
- **Specificity of imaging findings:** Although swelling is a sensitive finding on imaging, it is not always specific and can occur in overlapping clinical processes.

Treatment

Necrotizing soft-tissue infections require prompt (≤24 hours) surgical intervention and early empiric broad-spectrum antibiotic coverage. Timely surgical exploration of the site of infection is critical for determining the extent of the infection, obtaining specimens for histology and Gram stain, and debriding necrotic tissue. The earlier patients undergo surgery, the higher their likelihood of survival. After initial surgical exploration, reinspection and debridement should be repeated within 24 hours and every 1 to 2 days until there is no longer any evidence of diseased tissue. Definitive antibiotic treatment should ultimately be based on the patient's laboratory tests and local antibiogram data.

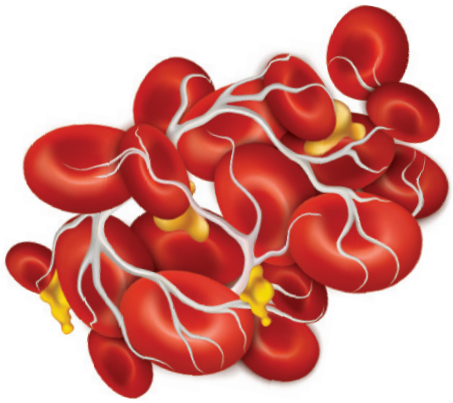
Adjunctive therapies may be considered for the treatment of necrotizing soft-tissue infections, particularly in critically ill patients. Some patients may require high-volume crystalloids or colloid fluid repletion. Patients who develop anemia due to bacterial hemolysin may require a blood transfusion. Some patients with type II necrotizing fasciitis develop toxic shock syndrome-induced cardiomyopathy, requiring vasopressors or cardiac-assist devices.

KEY POINTS

- Necrotizing soft-tissue infections are characterized by widespread tissue destruction that can extend from the epidermis to the deep musculature.
- Surgical intervention is important for both the diagnosis and treatment of necrotizing soft-tissue infections.
- Early surgical intervention is critical for reducing mortality and improving outcomes.
- Empiric broad-spectrum antibiotics should be administered early if a necrotizing soft-tissue infection is suspected.

Transient Ischemic Attack

By Tuyen Kiet, MD; and Laura Welsh, MD
Department of Emergency Medicine, Boston University, Massachusetts
Reviewed by Andrew Eyre, MD, MHPEd



Lo BM, Carpenter CR, Hatten BW, Wright BJ, Brown MD; American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Suspected Transient Ischemic Attack. Clinical policy: critical issues in the evaluation of adult patients with suspected transient ischemic attack in the emergency department. *Ann Emerg Med*. 2016;68(3):354-370.

The evaluation of suspected transient ischemic attacks (TIAs) is neither simple nor straightforward. Highly variable management strategies and a lack of risk-stratification tools can further muddy the clinical picture. In 2016, the American College of Emergency Physicians issued a clinical policy to address these discrepancies.

Although TIAs are not known to cause lasting morbidity, patient outcomes are complicated by the increased, short-term risk of an ischemic stroke. It is vital to differentiate patients who require hospital admission from those who can be managed in an outpatient setting. While several diagnostic instruments exist, their applicability in the emergency department is limited by a variety of factors, including the population and exclusion criteria originally studied.

The ABCD^2 score is the most researched and widely used risk-stratification tool for patients with TIAs; however, this approach may not be generalizable to all institutions. Unfortunately, existing clinical decision guidelines, including the ABCD^2 score, do not appear to reliably predict a patient's risk of an early, recurrent stroke.

It is imperative to identify intracranial masses and bleeds that can mimic stroke; however, very few of these pathologies can be identified with noncontrast head CT (NCHCT). CT scans also appear to have limited utility for identifying TIA patients at risk of having an ischemic stroke within the ensuing month. On the other hand, MRI with diffusion-weighted imaging (DWI) can be used to reliably gauge this risk. When MRI is not readily available, NCHCT is a reasonable option; however, NCHCT should not be used for risk stratification.

The imaging of intracranial and cervical vessels is another important part of the diagnostic algorithm. Patients with stenosis of a vascular supply are at a higher risk of ischemic stroke. Carotid stenosis greater than 50% appears to be an independent predictor of such events. An early carotid endarterectomy in TIA patients with carotid stenosis (>70%) can significantly reduce the future likelihood of stroke and disability.

While DWI is superior to CT for examining the brain, the cervical vasculature can be evaluated with either Doppler ultrasonography (DUS) or magnetic resonance angiography (MRA). DUS has a similar specificity but slightly lower sensitivity than MRA for the detection of clinically significant carotid stenosis. Carotid ultrasound is another acceptable modality for identifying carotid stenosis.

Observation protocols and rapid outpatient TIA clinics have been developed to limit unnecessary hospital admissions. In most emergency department-based protocols, low-risk patients can be admitted to the observation unit for telemetry, serial examinations, brain and vascular imaging, echocardiography, and a neurology consultation as needed. Inpatient admission should be reserved for cases with high-risk features (eg, atrial fibrillation, valvular disease, carotid stenosis, abnormal brain imaging).

Of note, observation unit protocols yield shorter lengths of stay and lower costs. More importantly, the rate of stroke in patients who receive observation care is similar in those who are risk stratified using the ABCD^2 score.

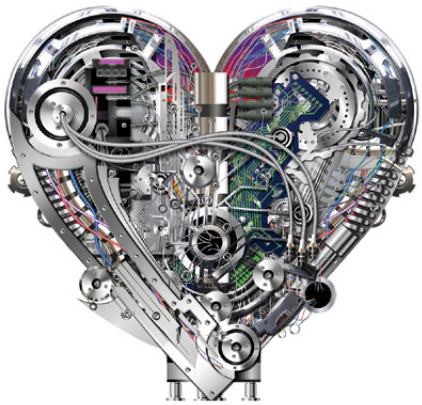
KEY POINTS

- Do not use existing diagnostic instruments to guide the disposition of any patient with a suspected TIA.
- Head CT is not useful for identifying patients at high risk of a subsequent stroke; MRI with DWI is preferred.
- Neck imaging should be obtained when feasible, as the early identification of carotid stenosis has a significant impact on patient management and outcomes.
- DUS is a safe substitute for CT and MRA.
- A rapid emergency department-based protocol can be safely employed to predict a patient's short-term risk of stroke.

MRI Safety in Patients With Cardiac Devices

By Andrew R. Albert, MD; and Michael E. Abboud, MD
University of Pennsylvania, Philadelphia
Reviewed by Andrew Eyre, MD, MHPEd

Nazarian S, Hansford R, Rahsepar AA, et al. Safety of magnetic resonance imaging in patients with cardiac devices. *N Engl J Med.* 2017 Dec 28;377(26):2555-2564.



Many patients who undergo placement of a permanent pacemaker (PPM) or implantable cardioverter-defibrillator (ICD) subsequently develop an indication for MRI. Unfortunately, some are denied the opportunity to undergo the test due to safety concerns. Despite this common trepidation, MRI poses no additional risks to patients with contemporary PPM and ICD systems, most of which are labeled by the FDA as "MRI conditional." Furthermore, the Centers for Medicare & Medicaid Services has determined that access to MRI improves outcomes in patients with modern PPMs and ICDs. Devices that do not carry the MRI-conditional designation are termed "legacy" systems.

A variety of adverse events in patients with ICDs can be attributed to the electromagnetic interference of MRI magnets. For instance, a power-on reset event occurs when a device is briefly turned off and then back on, a process that can cause the system to revert to a backup mode that may fail to appropriately manage a patient's condition. Additionally, MRI can cause changes in sensing and pacing thresholds that require device reprogramming to achieve adequate capture.

Worrisome potential adverse events, such as generator failure and battery depletion, are theoretical risks. PPM and ICD systems may also misinterpret the electromagnetic waves delivered to the patient as atrial or ventricular activity, resulting in unnecessary antitachycardia pacing or shocks. Side effects such as pain, a warm sensation in the area of the device, and palpitations have also been reported.

A recent single-center trial examined the rate of adverse events in patients with a variety of legacy PPM and ICD systems who underwent MRI. The examinations, which were conducted using a predetermined safety protocol, were monitored by an experienced cardiology nurse who had immediate access to an electrophysiologist. The devices were reprogrammed to asynchronous pacing for all patients with inadequate intrinsic heart rates (<40 bpm); inhibited pacing modes were used for all nonpacing-dependent patients.

A variety of standard device parameters were measured immediately prior to and within minutes of MRI completion. A total of 1,509 patients with legacy devices underwent 2,103 thoracic and nonthoracic MRI examinations at a magnetic field strength of 1.5 Tesla.

During the testing, 9 power-on reset episodes were recorded; however, none of these events resulted in device dysfunction or long-term clinical sequelae. Although 6 of the MRI examinations were aborted for a variety of reasons, no unfavorable effects were noted. The changes in device parameters were never large enough to require device reprogramming, either immediately following the examination or at long-term follow-up. There did not appear to be any association between detrimental changes in device parameters and the region that was imaged. Notably, 137 pacing-dependent patients underwent MRI without safety issues.

This evidence supports research by the MagnaSafe Registry, which endorses the safety of MRI for patients with legacy devices, assuming that appropriate protective measures and monitoring are in place.[1]

1. Russo RJ, Costa HS, Silva PD, et al. Assessing the risks associated with MRI in patients with a pacemaker or defibrillator. *N Engl J Med.* 2017 Feb 23;376:755-764.

KEY POINTS

- Access to MRI can improve outcomes in patients with ICDs.
- Patients with legacy PPM or ICD systems can safely undergo both thoracic and nonthoracic MRI examinations; however clinicians should adhere to an appropriate safety and monitoring protocol.
- The changes in device parameters were not detrimental enough to warrant immediate or long-term reprogramming in patients with legacy devices who underwent MRI.

Intramuscular Sedation for Agitation

By Michael Char, MD; and Laura Welsh, MD
Department of Emergency Medicine — Boston Medical Center/Boston University, Massachusetts
Reviewed by Andrew Eyre, MD, MHPEd

Klein LR, Driver BE, Miner JR, Martel ML, Hessel M, Collins JD, et al. Intramuscular midazolam, olanzapine, ziprasidone, or haloperidol for treating acute agitation in the emergency department. *Ann Emerg Med.* 2018 Oct;72(4)374-385.

Agitation is a common emergency department presentation, with severity ranging from restlessness to aggressive and violent behavior. Agitation is a symptom with a broad differential, including drug and alcohol intoxication, psychiatric ailments, and medical illness. Unfortunately, this symptom often impedes the evaluation of these cases and places staff and other patients at risk. First-line approaches include early identification, treatment of reversible causes, and verbal de-escalation, but not all of these strategies may be successful. The administration of parenteral medication is often necessary.

Researchers designed a single-center, prospective, observational study over a 15-week period, examining the efficacy of five medication regimens for the treatment of agitation in adults. The study site was a large, urban, teaching emergency department in Minneapolis, Minnesota, with more than 100,000 annual visits, more than 7,000 of which are for alcohol and illicit substance intoxication. Patients received initial treatment with a prespecified regimen, which changed every 3 weeks. The treatments tested were haloperidol 5 mg and 10 mg, ziprasidone 20 mg, olanzapine 10 mg, and midazolam 5 mg; all medications were administered intramuscularly (IM).

The primary outcome was the patient's altered mental status score (AMSS) 15 minutes after administration, evaluated as a proportion of patients adequately sedated (defined as AMSS <1). Secondary outcomes included the time to adequate sedation, rescue medications administered, and adverse effects.

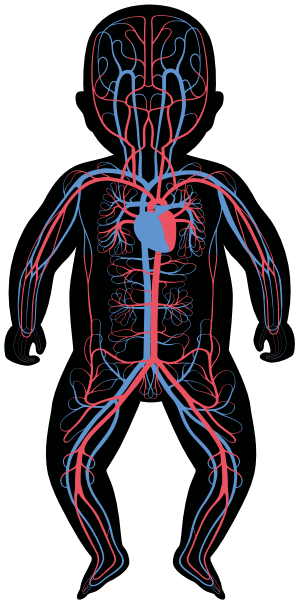
Midazolam, the most effective medication at 15 minutes, achieved a significantly greater proportion of adequately sedated patients compared to ziprasidone and both haloperidol doses. Olanzapine was the second-most effective agent, with statistical significance when compared to both haloperidol doses. The comparison between midazolam and olanzapine was insignificant.

Although midazolam achieved the fastest time to adequate sedation, it was also associated with the highest proportion of patients who required rescue medications. The authors attributed these findings to midazolam's short time to peak effect, half-life, and duration of action. The dose (5 mg vs 10 mg) of haloperidol did not make a significant difference in efficacy. Adverse effects were rare, and there were no notable differences in complications between the drug regimens.

The authors note that their sample is homogeneous; 72% of patients were male, and 88% were agitated due to alcohol intoxication. Additionally, the study participants presented with only mild-to-moderate agitation; the median baseline AMSS was 2. Finally, the study examined monotherapies rather than drug combinations, which are frequently employed in the emergency department. Further study is needed to compare IM monotherapies and drug combinations for the treatment of agitation.

KEY POINTS

- Parenteral medication is often necessary for the timely management of agitation in the emergency department.
- Midazolam 5 mg IM achieved the highest proportion of adequately sedated patients at 15 minutes with the fastest time to adequate sedation.
- Olanzapine 10 mg IM was significantly more effective at achieving adequate sedation at 15 minutes than haloperidol.



Pediatric Shock

By Laura Welsh, MD
Boston Medical Center/Boston University, Massachusetts
Reviewed by Andrew Eyre, MD, MHPEd

Mendelson J. Emergency department management of pediatric shock. *Emerg Med Clin North Am*. 2018 May;36(2):427-440.

Pediatric shock is a significant cause of morbidity and mortality worldwide, but early recognition and resuscitation can improve outcomes. In many cases of pediatric shock, the patient's clinical history may initially be vague and include concerns regarding lethargy, fussiness, and poor urine output.

Tachycardia is the most common vital sign abnormality in cases of pediatric shock, and delayed capillary refill time can be a useful marker of inadequate tissue perfusion. Other signs of hypoperfusion include decreased mental status, reduced urine output, and tachypnea. Hypotension, defined as a blood pressure below the 5th percentile for age, is a late and ominous finding.

The rapid assessment of historical and physical examination clues, targeted at identifying the classification of shock, can help guide therapeutic interventions. *Hypovolemic* shock, often due to GI losses, is the most common cause of shock in children and the leading cause of pediatric death worldwide. It is caused by intravascular volume depletion due to hemorrhage or fluid losses. *Distributive* shock, characterized by vasodilation, is most commonly caused by sepsis and anaphylaxis.

Cardiogenic shock in pediatric patients is commonly due to congenital heart disease, cardiomyopathies, or myocarditis. Finally, *obstructive* shock results from impaired pulmonary or systemic blood flow. In infants, congenital causes of obstructive shock may become apparent when the ductus arteriosus closes within the first few days or weeks after birth.

The initial treatment of these patients should focus on resuscitation. In cases of suspected shock, an initial isotonic fluid bolus of 20 mL/kg should be given, even in the absence of hypotension. Two additional boluses can be administered if systemic perfusion fails to improve. In neonates and patients with suspected cardiogenic shock, boluses of 10 mL/kg should be given instead, with frequent reassessments for signs of volume overload.

Vasopressors should be initiated after adequate volume resuscitation. Epinephrine is the vasopressor of choice followed by dopamine. Central venous line access is generally not needed; intraosseous access should be used for critically ill children in whom peripheral access cannot be achieved.

In pediatric patients, intubation can be considered for hemodynamic instability alone. Increased work of breathing consumes a significant portion of a child's oxygen consumption; however, support with mechanical ventilation can reduce this. Antibiotics should be given to all patients in whom there is concern for sepsis. Any child under 3 months of age with shock should be considered to have septic shock until proven otherwise. Steroids are indicated for those at risk of adrenal insufficiency, and prostaglandins should be used to manage ductal-dependent lesions with resulting obstructive shock.

Frequent reassessments and therapies that target the etiology of the patient's shock are critical. Resuscitation endpoints include the restoration of a capillary refill time under 2 seconds, normal blood pressure for age, normal urine output, and normal mental status. Unlike in adults, lactate clearance is generally not a recommended endpoint in the resuscitation of pediatric patients, as many children in shock have normal lactate levels.

KEY POINTS

- Attention to age-appropriate vital signs is imperative. Tachycardia is the most common vital sign abnormality, while hypotension is a late and ominous finding.
- Sepsis should be considered in all patients under the age of 3 months who present in shock.
- Rapid fluid resuscitation with up to three 20-mL/kg isotonic fluid boluses should be administered within the first hour.
- Epinephrine, the first-line vasopressor for pediatric patients in shock, can be administered through a peripheral IV.