Critical decisions in emergency medicine

THE 2024 LLSA LITERATURE REVIEW

The LLSA Literature Review

Synopses of articles from ABEM's 2024 Lifelong Learning and Self-Assessment Reading List

FROM THE EDITORS

Since April 2003, *Critical Decisions in Emergency Medicine*, ACEP's monthly CME publication, has included the 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.

This online supplemental issue of *Critical Decisions in Emergency Medicine* includes the 11 summaries of the 2023 LLSA reading list, 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 can also download full versions of the articles by logging in at **acep.org/moccenter/llsa**.

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

Best wishes,

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Critical decisions in emergency medicine

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Managing Opioid Use Disorder

By Aaron D'Amore, MD; and
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Objective

On completion of this article, you should be able to:

- Explain how patients with untreated OUD can be identified and treated in the emergency department.

Hawk K, Hoppe J, Ketcham E, et al. Consensus recommendations on the treatment of opioid use disorder in the emergency department. *Ann Emerg Med*. 2021 Sep;78(3):434-442.

KEY POINTS

- OAT initiation in the emergency department is proven to reduce mortality from opioid overdose.
- Effectively treating OUD in the emergency department aligns with emergency medicine's goal of intervening for high-mortality diseases.
- Emergency departments are often the only access patients with OUD have to health care. Thus, emergency physicians must identify patients with untreated OUD and provide them treatment.

People in the United States are more likely to die from unintentional opioid overdoses than from motor vehicle collisions. There is strong evidence to support that opioid agonist therapy (OAT) significantly reduces mortality from opioid use disorder (OUD). Patients with OUD often access health care only through the emergency department. Even though patients who present for a nonfatal opioid overdose are 100 times more likely to have a fatal overdose in the following year, only one-third receive OAT for this subsequent overdose. Hawk et al present consensus guidelines for the treatment of OUD in the emergency department based on a review of the pertinent literature and recommendations from an expert panel.

Three main pharmacologic agents are used to treat OUD: naltrexone, methadone, and buprenorphine. Naltrexone is a long-acting, competitive μ-opioid receptor antagonist used for both OUD and alcohol use disorder. Naltrexone precipitates opioid withdrawal; patients must abstain from opioids 7 to 10 days prior to using it, which significantly limits its use in emergency departments. Methadone is a synthetic μ-opioid receptor full agonist that can treat both OUD and chronic pain. Although it can potentially cause fatal respiratory depression if doses are rapidly escalated, methadone's benefit is that it does not precipitate opioid withdrawal. Buprenorphine, a synthetic μ-opioid receptor partial agonist, has a ceiling effect on respiratory depression but can precipitate withdrawal if initiated prior to abstinence- or naloxone-related withdrawal.

The guidelines recommend that emergency physicians treat opioid withdrawal, offer buprenorphine initiation, and provide a means for access to ongoing medication. Strong evidence indicates that patients with OUD treated with OAT outside of emergency departments have significant reductions in morbidity and mortality. The evidence also indicates that emergency departments are effective settings to engage patients in formal addiction treatment. A randomized controlled trial demonstrated that 78% of patients with OUD who were initiated on buprenorphine in the emergency department and referred for ongoing buprenorphine treatment participated in formal addiction treatment at 30 days and reduced their opioid use from 5.4 to 0.9 days per week.

Experts also recommend normalizing buprenorphine initiation in the emergency department for patients with untreated OUD and using nonstigmatizing language when discussing patients' drug use and treatment to maintain their trust. This language should be patient centered, professional, and objective, avoiding words such as *abuse*, *addict*, and *being clean*. Recommended person-first language to use would be phrases such as *person who injects drugs* or *patient with an opioid use disorder*. After buprenorphine initiation, patients should be sent home with a prescription for buprenorphine to bridge them to care with a trusted outpatient opioid treatment physician or program, ideally within 1 week.

Evidence-based, institution-specific protocols should be used to identify and treat patients with OUD to ensure buy-in and consistency. Most emergency department protocols include a validated assessment for OUD, an assessment of opioid withdrawal, and a determination of pregnancy status. Many protocols suggest that buprenorphine should be initiated with at least 8 mg for patients with clinical signs of opioid withdrawal and can be increased to 24 mg or more throughout the course of the visit. Emergency departments should engage their community stakeholders in developing treatment protocols to ensure that patients initiated on buprenorphine have direct, reliable, and specific referrals to outpatient programs for continuity. When no such connections exist, emergency physicians can identify programs in their area by using the Substance Abuse and Mental Health Services

Administration (SAMHSA) website. Hospitals can also provide health advocates and navigators in the emergency department who help motivate patients and assist them with getting to their appointments to ensure better outcomes.

Collaboration with hospital leadership and strong advocacy are critical for adopting OUD protocols in a hospital system. Integrating clinical decision support into the electronic health record has been shown to be highly effective in streamlining emergency department OUD treatment and referrals for continued treatment. A pilot study showed that these decision-making tools more than doubled prescription rates for buprenorphine and the number of physicians who adopted the prescribing practice.

Cellulitis and Abscess in the Emergency Department

By Richard Tamirian, MD; and
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Harvard Affiliated Emergency Medicine Residency, Boston, Massachusetts

Objective

On completion of this article, you should be able to:

- Describe the presentation and emergency management of cellulitis and abscesses.

Long B, Gottlieb M. Diagnosis and management of cellulitis and abscess in the emergency department setting: an evidence-based review. *J Emerg Med.* 2022 Jan;62(1):16-27.

KEY POINTS

- MRSA is the most common cause of abscesses, and *S. aureus*, *S. pyogenes*, and other β-hemolytic streptococci are the most common causes of cellulitis.
- POCUS can help differentiate cellulitis from an abscess and can guide abscess incision and drainage.
- Penicillins or cephalosporins are preferred treatments for cellulitis. Incision and drainage is used to treat an abscess, with the preferred technique of loop drainage.
- Adjunctive antibiotics can be considered for abscesses; antibiotics have been shown to reduce failure rates and new lesion formation.

Skin and soft tissue infections (SSTIs), including cellulitis and abscesses, occur when disruption to the skin barrier allows skin flora and other bacteria to enter the dermis and subcutaneous tissues, creating an inflammatory response. Cellulitis and abscesses contribute to approximately 6 million emergency department visits each year.

Nonpurulent cellulitis is most often caused by β-hemolytic streptococcal species, usually group A (*Streptococcus pyogenes*). *Staphylococcus aureus* is the second-most isolated organism, accounting for 14% to 27% of cases; only 4% of nonpurulent cellulitis cases are attributed to methicillin-resistant *S. aureus* (MRSA). By contrast, approximately 70% of isolates from abscesses grow MRSA. In patients who use intravenous drugs, however, abscesses may be polymicrobial.

Emergency physicians should inquire about initial symptoms, symptom progression, prior treatment, and a history of any similar conditions. They should also assess for risk factors for SSTIs or more serious infections, including intravenous drug use, circulatory problems, known ulcers or wounds, and immunocompromised states such as from cancer treatment or diabetes. During physical examination, patients should be evaluated for any possible entry points for infection and any signs that suggest cellulitis, including tenderness, warmth, edema, or erythema. Fluctuance with surrounding erythema may suggest an abscess.

The differential diagnosis for patients with suspected cellulitis or a suspected abscess is broad and includes vascular pathologies, autoimmune conditions, and allergic dermatoses. Cellulitis typically presents acutely and unilaterally, which can help differentiate it from other conditions. Cellulitis is also associated with a prior history of the condition, lower-extremity ulcers or edema, tinea pedis, and an elevated body mass index. Emergency physicians should also evaluate for crepitus, bullae, vesicles, and any pain that is out of proportion to examination — these symptoms may suggest a necrotizing soft tissue infection.

Laboratory testing is of limited use but typically shows a leukocytosis and elevated inflammatory markers. Routine blood cultures are not recommended but can be useful in patients with suspected sepsis or patients at greater risk of infection with atypical organisms (eg, immunocompromised patients and those with animal bites). Routine skin cultures are not recommended for typical cellulitis but are recommended for drained abscesses, although the majority of isolates will be MRSA.

Plain x-rays are of little use in SSTI evaluation. CT may be useful when evaluating abscesses, especially deeper collections, but it has been shown to miss smaller abscesses. Point-of-care ultrasound (POCUS) is the preferred imaging modality: It has a 94.6% sensitivity and 85.4% specificity and may be useful in differentiating cellulitis from an abscess. A cobblestone appearance of subcutaneous tissues is consistent with cellulitis, whereas a hypoechoic fluid collection is more suggestive of an abscess. POCUS can also be used to guide the incision and drainage of an abscess.

Most patients with cellulitis or an abscess can be treated in the outpatient setting after antibiotics are initiated or after incision and drainage in the emergency department. For nonpurulent cellulitis, the recommended antibiotics include penicillin V potassium, a cephalosporin, dicloxacillin, or clindamycin. If purulent drainage or an abscess is present, cellulitis should be treated with an antibiotic with MRSA coverage, such as trimethoprim-sulfamethoxazole or doxycycline. An antibiotic course of 5 to 10 days is typically appropriate, although 7 to 14 days may be required for immunocompromised patients. NSAIDs, when not contraindicated, should be used for symptomatic relief.

Incision and drainage is the treatment of choice for abscesses. The loop drainage technique has been shown to have significantly lower failure rates than a traditional incision and drainage. In the loop drainage technique, two small incisions are made at opposite margins of the abscess. Loculations are then broken up with a hemostat, and a vessel loop is placed through the incisions and tied to create a loop. The loop can be slid back and forth daily to allow patients to drain the abscess and can be cut and removed when the abscess resolves. Irrigation of the abscess pocket has not been shown to improve outcomes and is not recommended. Packing is more controversial, with limited data on its effectiveness; it can be reserved for larger abscess (>5 cm in diameter). The role of antibiotics for uncomplicated abscesses is also unclear, but antibiotics have been shown to significantly reduce rates of treatment failure and new lesion formation. Most patients without systemic signs of infection or who have not failed prior treatment can be safely discharged with strict follow-up and return precautions.

Preventing Post–Lumbar Puncture Headache

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Reviewed by Andrew J. Eyre, MD, MS-HPEd

Objective

On completion of this article you should be able to:

- Discuss best practices for preventing post–lumbar puncture headache.

Cognat E, Koehl B, Lilamand M, et al. Preventing post-lumbar puncture headache. *Ann Emerg Med.* 2021 Sep;78(3):443-450.

KEY POINTS

- Atraumatic needles are associated with a twofold decrease in post–lumbar puncture headache, with similar procedural success rates when compared to cutting needles.
- Older age (≥60 years) and early mobilization may protect against post–lumbar puncture headache. Limited data suggest that smaller needles and placing patients in the lateral decubitus position are also associated with a lower incidence of these headaches.
- Volume of CSF sampled has not been shown to correlate with the incidence of post–lumbar puncture headache.

Post–lumbar puncture headache is the most common complication of lumbar punctures, occurring in 3.5% to 33% of patients. The risk factors associated with post–lumbar puncture headache are not fully understood. Practices aimed to prevent post–lumbar puncture headaches are heterogeneous.

In this literature review on common questions about post–lumbar puncture headaches, only one significant, nonmodifiable risk factor was identified: Age older than 60 years is a protective factor. Early studies showed an increase in incidence of post–lumbar puncture headache among women and patients with a lower body mass index. However, subsequent studies have failed to replicate the correlation between these factors and an increased incidence of post–lumbar puncture headache. Underlying illnesses such as HIV and autoimmune disease with cerebral involvement are unassociated with a significant alteration in headache risk.

Traumatic sharp cutting-point needles, or *Quincke needles*, were the initial needle developed for lumbar puncture. Atraumatic, or noncutting, needles (ie, pencil-point and bullet-point needles) were developed later. In postmortem dura mater studies, atraumatic needles caused decreased CSF leakage and dura mater injury. Clinically, two large meta-analyses identified a nearly twofold increase in post–lumbar puncture headache when cutting needles were used compared to atraumatic needles, and both needle types are associated with similar rates of procedural success. With cutting needles specifically, one study found a lower incidence of post–lumbar puncture headache when the bevel of the needle was oriented parallel to the axis of the spine.

The association between needle diameter and post–lumbar puncture headache is unclear. Needles with smaller calibers are associated with a decreased risk of post–lumbar puncture headache in smaller studies; however, a large meta-analysis failed to demonstrate an association. A single-center randomized controlled trial found a significant decrease in the incidence of post–lumbar puncture headache with the use of a 25-gauge atraumatic needle compared to a 22-gauge needle. Reinserting the stylet into the cannula prior to needle removal theoretically prevents arachnoid strand entrapment during removal. One randomized controlled trial showed a significant relationship between stylet reinsertion and prevention of post–lumbar puncture headache; however, the association did not hold in a subsequent randomized controlled trial.

Performing a lumbar puncture with the patient in the lateral decubitus position potentially offers more protection against post–lumbar puncture headache than with the patient in a seated position; however, data are limited for diagnostic lumbar punctures. The number of lumbar puncture attempts, presence of blood in the CSF, and deformation of the needle (apart from hook-shaped deformities) are not associated with post–lumbar puncture headache, although data are limited.

Although physicians may limit the collection of CSF samples to 10 to 15 mL due to a clinical concern for headache, several studies failed to demonstrate a correlation. In fact, one study found that although larger volumes of diagnostic lumbar puncture sampling up to 30 mL are associated with an increased risk of immediate postprocedural headache, these volumes decrease the risk of post–lumbar puncture headache. Additionally, negative pressure and active suction to expedite CSF collection are not associated with an increased post–lumbar puncture headache risk.

Patients are commonly instructed to lie flat after a lumbar puncture. However, a review of nearly 3,000 patients found that immobilization was actually associated with a low-but-significant increase in post–lumbar puncture headache. Additionally, headache incidence is not reduced following fluid supplementation or caffeine intake. Data on medications to prevent post–lumbar puncture headaches, particularly for diagnostic lumbar punctures, are quite limited. One uncontrolled study showed a potential benefit of frovatriptan use, but this has not yet been replicated.

Overall, this study may change commonly held notions and practices for post–lumbar puncture headache risk factors and prevention. An age older than 60 years has been repeatedly demonstrated to be a nonmodifiable protective factor against post–lumbar puncture headache. During the procedure, atraumatic and smaller gauge needles are protective, while stylet reinsertion, limiting CSF sample volume, and repeated or traumatic lumbar puncture may not influence risk. Post procedure, immobilization may be harmful, and fluid or caffeine supplementation provide no benefit.

Managing Diabetic Ketoacidosis in Children

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Reviewed by Andrew J. Eyre, MD, MS-HPEd

Objective

On completion of this article, you should be able to:

- State how to manage DKA and its complications in pediatric patients.

Tzimenatos L, Nigrovic LE. Managing diabetic ketoacidosis in children. *Ann Emerg Med*. 2021 Sep;78(3):340-345.

KEY POINTS

- Suspect DKA in patients with diabetes who have polyuria, polydipsia, weight loss, abdominal pain, vomiting, headaches, Kussmaul respirations, or an altered mental status. Check glucose, electrolyte, and blood gas levels and order a urinalysis.
- Treatment of DKA includes fluid resuscitation, insulin administration, and monitoring vital signs and glucose and electrolyte levels.
- Frequent neurologic checks help identify cerebral edema early. Physicians should immediately initiate head-of-bed elevation and hyperosmolar therapy.

Introduction

Diabetic ketoacidosis (DKA) is a severe complication that is predominantly seen in children with type 1 diabetes. DKA occurs in 30% of children at the time of their diabetes diagnosis and 6% to 8% annually after initial diagnosis. Diagnosis of DKA hinges on laboratory findings of hyperglycemia and ketosis. Treatment focuses on intravenous fluids and insulin administration. *Close laboratory and clinical monitoring are necessary for cerebral edema, a rare but lethal complication of DKA treatment.* Recent research offers new insights into fluid treatment strategies for pediatric DKA.

Diagnosis

Clinical Presentation

Early signs of DKA include polyuria, polydipsia, fatigue, and weight loss. Younger children can manifest subtle symptoms, and their blood pressure and heart rate may not accurately reflect dehydration levels. If left untreated, the symptoms evolve into abdominal pain, vomiting, and headaches. Advanced DKA reveals compensatory responses to metabolic acidosis: labored, tachypneic Kussmaul respirations and altered mental status.

DKA Definition

Diagnosis of DKA requires the following specific laboratory findings:

- Hyperglycemia (blood glucose level >200 mg/dL);
- Metabolic acidosis (venous pH <7.3; serum bicarbonate level <15 mEq/L); and
- Ketosis (elevated serum or urine ketone levels).

Laboratory Evaluation

When DKA is suspected, critical laboratory tests include a point-of-care glucose reading; venous blood gas (VBG) analysis; serum levels of electrolytes (with magnesium and phosphorus), creatinine, and β-hydroxybutyrate; and a urinalysis. Serum sodium levels should be corrected for the presence of hyperglycemia. CBCs may reveal a nonspecific leukocytosis. Long-term glucose control can be monitored using the hemoglobin A_{1C} test. Notably, acute kidney injury has been observed in a significant portion of children with DKA and is often related to severe dehydration and acidosis.

Treatment

Fluid and Electrolyte Replacement

Most children with DKA are dehydrated by 5% to 10%. Fluid resuscitation should span 36 to 48 hours. *Begin with a normal saline 10 mL/kg intravenous bolus; repeat if persistent poor perfusion or tachycardia is present in patients with a Glasgow Coma Scale (GCS) score ≥14.* When treating DKA, fluid and insulin interventions will gradually correct the observed acidosis, so sodium bicarbonate use should be avoided. Potassium replacement (adding 40 mEq/L) is vital when serum potassium levels are under 5 mEq/L. Although these potassium levels appear elevated, there is usually a total-body potassium deficit. Patients with DKA are often placed on oral intake restrictions because of nausea and a decreased mental status; however, in appropriate cases, small amounts of water or ice chips can be beneficial.

Insulin Infusion

After fluid resuscitation, insulin administration is paramount. For mild cases (pH 7.2-7.3), subcutaneous rapid-acting insulin should be used. For moderate to severe cases (pH <7.2), however, regular intravenous insulin (0.05-0.1 units/kg/hr) is required; improvement of acidosis

indicates the patient can be transitioned away from intravenous insulin.

Prevention of Hypoglycemia

When glucose levels drop below 300 mg/dL, dextrose (5%-10%) should be added to intravenous fluids. The two-bag method — one with and one without dextrose — regulates blood glucose levels by adjusting fluid types.

Airway Management

Unless the patient is in respiratory failure or cannot protect their airway, intubation is avoided in pediatric patients with DKA because it blunts compensatory respiratory alkalosis.

Patient Monitoring

Constant monitoring is essential and includes hourly vital sign, neurologic, and glucose checks and repeat laboratory tests for electrolyte and VBG values every 2 to 3 hours. A second peripheral intravenous line is needed for frequent blood draws and uninterrupted insulin infusion. Invasive procedures like central venous catheter placement are discouraged because of risks like deep vein thrombosis.

Disposition and Transfer Considerations

Patient disposition is contingent on disease severity and available resources. If DKA is mild, acidosis has resolved, and the patient tolerates oral intake, they can be managed in the emergency department with strict outpatient follow-up care. However, patients with severe DKA (pH <7.1), altered mental status, or electrolyte derangements require ICU monitoring. Importantly, transfers for moderate to severe DKA should involve critical care transport teams.

Cerebral Edema

Cerebral edema is an uncommon (<1%) but devastating complication of DKA treatment. Clinical manifestations typically emerge 12 to 24 hours post treatment and include neurologic and vital sign changes. When cerebral edema is detected, rapid treatment with hyperosmolar therapy is paramount. Hypertonic saline solutions are increasingly becoming the first line of treatment for this condition.

Prevention

Although clinically overt cerebral edema is uncommon, the more common subclinical presentations are still linked to long-term neurocognitive deficits. Historically, physicians attributed the onset of cerebral edema to rapid fluid administration or electrolyte correction. However, recent studies have challenged these beliefs. A 10-center study found no direct association between the rate of fluid administration and cerebral edema and suggested that hydration status should guide fluid resuscitation for children with initial GCS scores of 14 or 15.

Treatment

Treating cerebral edema should be swift. It includes elevating the head of the bed and providing hyperosmolar therapy, either intravenous mannitol (0.5-1 mg/kg) or hypertonic 3% saline (5 mL/kg).

Summary

DKA is a pediatric emergency that requires early diagnosis, appropriate fluid resuscitation, intravenous insulin infusion, and vigilant monitoring to detect electrolyte abnormalities or cerebral edema. Recent evidence does not support restricting fluid administration to prevent cerebral edema in these patients. Given the potential complications of DKA, most patients warrant ICU care until DKA resolves. Effective management can ensure better outcomes for pediatric DKA patients.

Disclosure

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.

Palliative Care

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Reviewed by Andrew J. Eyre, MD, MSHP-Ed

Objective

On completion of this article, you should be able to:

- Discuss best practice guidelines for emergency department primary palliative care.

Loffredo AJ, Chan GK, Wang DH, et al. United States best practice guidelines for primary palliative care in the emergency department. *Ann Emerg Med.* 2021 Nov;78(5):658-669.

KEY POINTS

- Patients who have a life expectancy of ≤6 months, have DNR orders, and want to focus on symptom management and quality of life are appropriate for referral to palliative care.
- Emergency department primary palliative care includes engaging interdisciplinary teams, communicating effectively, establishing trust, and utilizing a holistic approach to provide empathetic and effective care for patients with advanced diseases.
- US emergency department primary palliative care guidelines provide recommendations for quality care through four phases: (1) screenings and assessments, (2) patient management interventions, (3) palliative care consultations, and (4) transitions of care.

Approximately 20% of patients on hospice and 75% of Americans older than 65 years visit the emergency department in their final 6 months of life. As the US population continues to age, it is important to address the palliative care needs of patients and educate emergency physicians on the best practices of primary palliative care. Primary palliative care includes basic palliative care skills, patient-centered communication, advance care planning, and refractory symptom management. Emergency department palliative care improves these patients' quality of life while also reducing their utilization costs. Moreover, emergency physicians are in a unique position, given their brief time of care and resource-limited environment, to assess for and implement primary palliative care.

An interdisciplinary emergency department palliative care expert panel and literature review published guidelines for best practices for palliative care in the emergency room. The panel included both academic and community faculty, many of whom had formal hospice and palliative care training. A draft of key points was presented at the American College of Emergency Physicians Scientific Assembly Palliative Medicine Section meetings in 2017 and 2018 for additional comments, which were then implemented into the drafts and peer reviewed.

The Four Pillars of Primary Palliative Care in the Emergency Department Guidelines

Screening and Assessing Patients for Palliative Care Needs

Emergency physicians should utilize the available screening tools to assess for a spectrum of palliative care needs including physical, emotional, social, psychological, and spiritual needs, as well as those associated with grief. Important steps in screening include identifying the presence of life-limiting conditions, considering functional decline, and using clinical judgment to consider the risk of death. Support tools in electronic health records can be helpful in prompting physicians to ask patients about advance care planning or lack thereof, previous hospice discharges, prior do not resuscitate (DNR) orders, long-term care facility stays, and palliative care consultations. Categories of primary palliative care needs for emergency department patients include a clarification of code status, symptom management, an introduction to palliative care, and caring for a current or prior hospice patient.

Patient Management and Palliative Care Interventions

Emergency physicians should provide medication to palliate symptoms of pain, nausea and emesis, dyspnea, delirium, terminal secretions, and constipation. They should also ensure effective communication during goals-of-care discussions. The four integral components of effective goals-of-care conversations include assessing the patient's and family's understanding of the active illness, understanding how the illness has changed the patient's quality of life, asking about the patient's and family's fears, and providing clinical recommendations after synthesizing patient and family priorities, prognosis, and clinical trajectory. Although definitive decisions are difficult for patients and families to make in the emergency department, time-limited trials of life-prolonging care with plans for revisiting goals-of-care conversations after an initial stabilization can help by allowing time for family discussions about explicit milestones of improvement, alternative considerations, and progress definitions.

Palliative Care Consultations in the Emergency Department

Referrals can be made from the emergency department for patients who have a life expectancy of ≤6 months, have DNR orders, and want to focus on symptom management and quality of life. Hospice agencies, social work, and case management will assist with patient logistics, including hospice education, signing over insurance benefits, establishing home visits or inpatient hospice, and engaging caregivers, such as nurses, chaplains, and other palliative care professionals.

Transitions of Care

In contrast to traditional dispositions, hospice care–eligible emergency department patients have a myriad of options, including admission while awaiting an inpatient palliative consultation, admission for pain control, referral to an outpatient palliative clinic, transition to comfort care, and transfer to an observation unit.

Peripheral Nerve Block for Hip Fracture

By Abigail Raynor, MD, MPH; and
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Objective

On completion of this article, you should be able to:

- Discuss the use of PNBs as a tool for pain management in patients with hip fractures.

Gottlieb M, Long B. Peripheral nerve block for hip fracture. *Acad Emerg Med.* 2021 Oct;28(10):1198-1199.

KEY POINTS

- PNBs are an effective tool for pain management in patients with acute hip fractures, resulting in a decreased incidence of delirium and chest infections as well as a shorter time to mobilization.
- Adverse events associated with PNBs are rare; poor outcomes like nerve damage occur in just 0.03% of all cases.
- The American Academy of Orthopedic Surgeons now recommends the use of regional analgesia for preoperative pain control in all patients with hip fracture.

A peripheral nerve block (PNB) is one of the many tools emergency physicians can employ to manage acute pain. The procedure typically involves injecting local anesthetic adjacent to a nerve or into a fascial plane surrounding a nerve. PNBs can be performed using anatomic landmarks but are increasingly being performed using point-of-care ultrasound to guide needle placement with more accuracy. PNBs are commonly used to address pain secondary to bony injuries, such as hip or rib fractures; they can also be used to manage a wide variety of presentations ranging from sciatica to soft tissue injuries.

From 2000 to 2010, hip fractures accounted for more hospitalizations in women older than 55 years than all other fractures combined. The fascia iliaca block is considered a highly effective means for managing discomfort secondary to this injury because it typically offers up to 12 hours of regional pain relief. When performed correctly, PNBs not only offer significant symptomatic improvement, but they also reduce the number of additional agents required to manage patients' pain.

Although effective, opioid-based regimens are associated with adverse effects such as delirium and respiratory depression, both of which disproportionately impact the geriatric population. Given the prevalence of bony injuries in older adults, there is a demonstrated interest in selecting pain management plans that offer symptomatic relief while avoiding the potentially deleterious effects of opioids.

The Peripheral Nerve Block for Hip Fracture study was a meta-analysis of 43 randomized controlled trials that sought to assess the impact of PNBs on pain and other critical outcomes like delirium, time to mobilization, and chest infections in adults with hip fractures. The study reviewed data from 2,750 adults aged 59 to 89 years who were admitted to the hospital with acute hip fracture and were awaiting operative repair.

The authors found that the administration of PNB offered reduced pain and decreased the incidence of delirium and chest infections, with a shorter time to mobilization when compared to no block or a placebo. Specifically, this study found that PNBs reduced pain by 2.5 points on a 1- to 10-point scale compared to no block or a placebo, were associated with a 7.3% lower risk of acute delirium during hospitalization, and showed a 15.9% lower risk of chest infection. The administration of PNBs was found to be protective against delirium for 1 in 14 patients and chest infections for 1 in 7.

These findings suggest that PNBs are a highly effective means of managing pain in adults with hip fractures because they reduce the likelihood of poor outcomes. The American Academy of Orthopedic Surgeons now recommends the use of PNBs in all patients with hip fractures; they do so in agreement with the Society for Academic Emergency Medicine, who has assigned a color recommendation of green (meaning the benefits exceed the harm) for this intervention.

The limitations of this study include the lack of assessment of harm or poor outcomes associated with the administration of PNB. Although this intervention is thought to be a safe procedure, adverse events include nerve damage, local toxicity, and infections. As this paper reports, such events are rare, with other studies documenting that permanent nerve injury occurred in just 0.03% of nerve blocks and local toxicity in 1.3 per 10,000 cases. In summary, PNBs are an effective option for controlling pain in the preoperative setting for adults who sustain hip fractures. They are associated with not only improvement in pain but also reduction of poor outcomes during hospitalization. It is, therefore, recommended that nerve blocks be considered a key intervention in the emergency department management of patients with hip fracture.

Multisystem Inflammatory Syndrome in Children

By David W. Spivey, MD, LT, MC, USN; and Daphne P. Morrison Ponce, MD, CDR, MC, USN
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Reviewed by Andrew J. Eyre, MD, MS-HPEd

Objective

On completion of this article, you should be able to:

- Describe the presentation and management of MIS-C.

Waseem M, Shariff MA, Tay ET, et al. Multisystem inflammatory syndrome in children. *J Emerg Med.* 2022 Jan;62(1):28-37.

KEY POINTS

- The presence of GI symptoms, lymphopenia, and thrombocytopenia are helpful in distinguishing MIS-C from KD.
- MIS-C is a diagnosis of exclusion.
- Management of shock in MIS-C is based on warm vs cold phenotypes, similar to managing shock in other conditions.
- First-line treatment of MIS-C is IVIG and corticosteroids.

Multisystem inflammatory syndrome (MIS-C) is a newly identified condition that affects children who have had a recent COVID-19 infection. The symptoms of this condition affect multiple organ systems and resemble other severe syndromes such as Kawasaki disease (KD) and septic shock.

Clinical Presentation

MIS-C is defined as illness in an individual younger than 21 years that presents with fever, laboratory evidence of inflammation, and pathology of two or more organ systems. The illness cannot be explained by an alternative diagnosis and is associated with a recent or current COVID-19 infection or exposure; it requires hospitalization. Presenting symptoms can include headaches, altered mental status, conjunctivitis, oral mucosa changes, sore throat, cough, abdominal pain, vomiting, diarrhea, rash, lymphadenitis, and extremity edema. *The presence of GI symptoms can be helpful in differentiating MIS-C from KD.* Untreated, MIS-C can lead to the complications of myocarditis, coronary artery aneurysm, shock, serositis, acute respiratory distress syndrome, acute kidney injury, or hepatic failure. Laboratory findings in patients with MIS-C often include elevated levels of inflammatory markers: C-reactive protein (CRP), procalcitonin, erythrocyte sedimentation rate (ESR), D-dimer, lactate dehydrogenase, and ferritin. *An elevated WBC count with lymphopenia and thrombocytopenia can also help distinguish MIS-C from KD because KD typically presents with lympho- and thrombocytosis.*

Emergency Department Management

In stable children who present with symptoms concerning for MIS-C, consider obtaining initial screening laboratory tests. These tests consist of a CBC, complete metabolic panel, CRP, BNP, D-dimer, and COVID-19 test. Other tests to consider (depending on the clinical picture) include blood cultures, a urinalysis and urine culture, a respiratory viral panel, a throat culture, HIV testing, and tuberculosis testing. *Because MIS-C is a diagnosis of exclusion, other bacterial and viral causes of symptoms should be ruled out.* Well-appearing children with reassuring laboratory studies can reasonably be discharged home if close primary care follow-up is available within 24 to 48 hours. If initial inflammatory markers are elevated, further investigation is warranted, including tests for ESR, ferritin levels, troponin levels, and coagulopathies. Stable patients with MIS-C should be admitted to a monitored floor bed.

If children with suspected MIS-C are ill appearing and have signs of shock, the extremities and capillary refill time should be evaluated to distinguish between warm and cold shock. A bedside echocardiogram should also be performed to evaluate ejection fraction and volume status to guide fluid resuscitation. Septic patients (warm shock) should receive norepinephrine as the first-line vasopressor. Patients with cardiac dysfunction (cold shock) should receive epinephrine as their first-line vasopressor. Unstable MIS-C patients require admission to the pediatric ICU (PICU); transfer to a center capable of extracorporeal membrane oxygenation should be discussed with a cardiology specialist and the PICU team if a child is exhibiting signs of cardiac dysfunction.

Pharmacologic Management

First-line treatments for MIS-C include intravenous immune globulin (IVIG) and corticosteroids. IVIG should

be given as a single dose of 2 g/kg to patients who meet criteria for KD. Methylprednisolone 1 to 2 mg/kg/d can be given in addition to IVIG in ill-appearing patients and patients with shock or end-organ dysfunction. Additionally, low-dose aspirin (3-5 mg/kg/d) should be given to all MIS-C patients who do not have the contraindications of bleeding or severe thrombocytopenia; therapeutic anticoagulation should be strongly considered. After consultation with rheumatology or infectious disease specialists, anakinra and tocilizumab can be considered for cases refractory to IVIG and steroids. Remdesivir should be considered for patients with an active COVID-19 infection.

Disclosure

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.

Alcoholic Ketoacidosis

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Reviewed by Andrew J. Eyre, MD, MS-HPEd

Objective

On completion of this article, you should be able to:

- Diagnose and treat patients with AKA.

Long B, Lentz S, Gottlieb M. Alcoholic ketoacidosis: etiologies, evaluation, and management. *J Emerg Med.* 2021 Dec;61(6):658-665.

KEY POINTS

- AKA is a frequently encountered but an often-missed diagnosis in the emergency department, accounting for up to 7% of deaths in patients who drink alcohol.
- The pathophysiology of AKA is primarily driven by volume depletion, reduced glycogen and nutritional stores, and NADH/NAD+ mismatch, resulting in a starvation state and ketosis similar to diabetic ketoacidosis.
- AKA is diagnosed after taking a careful history, conducting a physical examination, and obtaining a serum metabolic panel with electrolyte measurements (including levels of magnesium and phosphorus) and serum ketone levels (such as β-hydroxybutyrate).
- Treatment primarily includes volume resuscitation, correction of electrolyte and glucose abnormalities, and thiamine repletion.
- Similar to diabetic ketoacidosis, physicians should rule out common precipitants of AKA, such as infection, pancreatitis, and toxic alcohol ingestion.

Alcoholic ketoacidosis (AKA) (previously referred to as alcoholic ketosis or alcoholic acidosis) is a clinical condition long recognized but likely still underdiagnosed in emergency departments. It is usually encountered in patients with chronic heavy alcohol use, especially those who have recently stopped drinking. According to the NIH, approximately 29.5 million people aged 12 years and older in the United States — representing 10.5% of all Americans in that age group — have alcohol use disorder.[1] AKA accounts for 7% of all deaths in patients who drink alcohol. Early detection and treatment in the emergency department are imperative.

AKA is characterized by ketoacidosis related to alcohol use and is primarily driven by reduced glycogen and nutritional stores, volume depletion, and elevation of the reduced nicotinamide adenine dinucleotide to nicotinamide adenine dinucleotide ratio (NADH:NAD+). Chronic alcohol users who get most of their daily calories from alcohol have poor nutritional reserves. This lack of nutrition leads to a starvation state with increased production of glucagon, growth hormones, cortisol, and catecholamines, resulting in depressed gluconeogenesis and glycogenolysis. In turn, lipolysis is upregulated, and fatty acids are oxidized into ketone bodies that cause the primary ketosis seen in AKA. The metabolism of alcohol increases the NADH:NAD+ ratio, interfering with mitochondrial activity and increasing conversion of pyruvate to lactate. In patients with cirrhosis, this metabolic storm may be further compounded by the damaged liver's decreased ability to perform gluconeogenesis and by resultant increases in gluconeogenic precursors such as lactic acid.[2] Taken together, these patients can easily develop severe metabolic derangements and significant lactic acidosis.

Patients with AKA will present with a history of alcohol use, poor nutrition, and usually, a recent halt in their alcohol intake. Their most common complaints are nausea, vomiting, and abdominal pain, which often precede the development of ketoacidosis — decreased nutritional intake and increased fluid and electrolyte losses from vomiting exacerbate their underlying metabolic derangements. Diagnosis is made through laboratory evaluation, including serum measurements of ketones (β-hydroxybutyrate) and a metabolic panel that includes evaluation of renal function and electrolyte levels (including magnesium and phosphorus). A blood gas analysis should also be considered because one study found that only 23% of patients had a typical anion gap metabolic acidosis alone.[3] Liver function testing is useful because it can help delineate underlying alcohol-induced liver disease. Patients with frequent alcohol use are at risk of other comorbidities; tests that measure serum osmolality and acetaminophen or salicylate levels can help narrow the differential diagnosis. In addition to acidosis and ketosis, patients with AKA frequently have abnormal BUN and creatinine levels and other electrolyte derangements. Importantly, serum markers of magnesium, potassium, and phosphorus are not reliable markers of total body stores in these patients.

Management of AKA follows good resuscitative principles and is, fortunately, similar to management

of diabetic ketoacidosis, which emergency physicians are used to managing. Volume resuscitation should be done with a pH-balanced crystalloid solution such as lactated Ringer to avoid exacerbating acidosis; electrolyte and glucose levels should also be replenished. Thiamine deficiency is common in patients with AKA — supplementation with thiamine 200 mg IV is recommended (500 mg IV instead if Wernicke encephalopathy is suspected). In clinically stable patients who are tolerating oral intake, oral repletion and nutrition are preferred, but in patients with severe or ongoing derangements, longer intravenous infusions may be necessary. If ketosis is persistent, consider supplemental insulin and dextrose.

The disposition of these patients depends on the severity of their metabolic derangements, as well as their clinical status. Patients with mild acidemia and electrolyte derangements who can tolerate oral intake can be discharged with outpatient follow-up care and addiction support services, while those with severe electrolyte derangements, ketoacidosis, or an inability to tolerate oral intake should be admitted. Patients with cardiac arrhythmias secondary to electrolyte imbalance require telemetry and ICU admission.

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Chronic Drug Use and Abdominal Pain

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Reviewed by Andrew J. Eyre, MD, MS-HPEd

Objective

On completion of this article, you should be able to:

- Explain the association between abdominal pain and substance use disorder and how to manage abdominal pain induced by substances.

Cates AL, Farmer B. Chronic drug use and abdominal pain. *Emerg Med Clin North Am*. 2021 Nov;39(4):821-837.

KEY POINTS

- The differential diagnosis for abdominal pain in patients with concomitant substance use disorder is broad. A thorough history and physical examination are crucial to guiding diagnostic testing and treatment. Drug screening tests may not capture synthetic substances, differentiate between current or remote use, or indicate acute withdrawal.
- Alcohol use can cause pathologic changes throughout the entire GI and hepatic systems, ranging from mild gastritis to life-threatening hemorrhage and organ failure.
- Opioid use decreases gastric motility, resulting in constipation, while acute opioid withdrawal causes diarrhea, nausea, and vomiting.
- Cocaine and amphetamines have vasoconstrictive properties that can contribute to bowel and hepatic ischemia.
- In addition to treating the specific etiology of abdominal pain, physicians should also provide treatment and resources for substance use disorder.

Abdominal pain is a frequent chief complaint of patients who present to the emergency department. Some of these patients also have a comorbid substance use disorder. The differential diagnosis for abdominal pain in patients with substance use disorder is broad, ranging from mild to life-threatening and acute to chronic conditions. A thorough history — including details of substance use, pain chronicity, and pain severity — and a physical examination are essential for appropriate diagnostic testing and subsequent treatment. Drug screening tests may not capture synthetic substances, differentiate between current or remote substance use, or indicate acute withdrawal. Establishing good rapport and avoiding stigma are essential to providing care to this patient population.

Ethanol

Alcohol is one of the most-used substances and can cause pathologic changes throughout the entire GI and hepatic systems. Acutely, alcohol can cause gastroesophageal reflux disease and, over time, can lead to portal venous hypertension and cause esophageal varices. Vomiting secondary to alcohol consumption can lead to esophageal tears (Mallory-Weiss syndrome), variceal bleeding, or esophageal rupture (Boerhaave syndrome). Alcohol use can also cause gastritis, which can be associated with *Helicobacter pylori* infection and peptic ulceration. Chronic alcohol use is often implicated in pancreatic disease including mild to severe pancreatitis, pancreatic abscess, pseudocyst, necrosis, and failure; most cases of chronic pancreatitis are associated with excessive alcohol use.

Within the hepatic system, alcoholic liver disease is a spectrum of disorders that includes steatosis, hepatitis, and cirrhosis. Alcoholic liver disease often leads to derangements such as anemia, thrombocytopenia, hypoalbuminemia, hyperbilirubinemia, a prolonged INR, and elevated liver transaminases. Intestinal dysfunction that results in diarrhea, impaired nutritional absorption, and folate deficiency can also occur from alcohol use. Binge drinking without proper nutritional intake can lead to alcoholic ketoacidosis. Alcohol cessation after prolonged consumption and dependency can cause withdrawal, including life-threatening seizures. Chronic alcohol use is associated with GI and hepatic carcinogenesis. Treatment of alcohol use disorder includes resuscitation, symptomatic management (eg, fluids, electrolyte repletion, antiemetics, withdrawal treatment), and controlled cessation.

Opioid Use

Opioid use disorder decreases gastric motility, resulting in constipation, adynamic ileus, and pseudo-obstruction. Opioid-induced constipation is common and can occur with both short-term and chronic opioid use, including with methadone and buprenorphine therapies. Opioids inhibit gastric emptying and peristalsis, which increases fluid absorption and hardens stool. Opioid-induced constipation can subsequently lead to bloating, nausea, and vomiting. An aggressive bowel regimen with fluids, dietary fiber, and

laxatives should be considered to help alleviate symptoms. Chronic opioid use can lead to narcotic bowel syndrome, which manifests as colicky abdominal pain that is likely secondary to hyperalgesia.

Combination opioids (typically an opioid paired with acetaminophen) can cause hepatotoxicity. Injection use poses additional risks that can lead to infection. Acute opioid withdrawal causes diarrhea, nausea, and vomiting and is managed with symptomatic and supportive care. Short-acting opioids may be appropriate for managing severe pain and withdrawal symptoms. Many options exist for medication-assisted treatment of opioid use disorder, including methadone, buprenorphine, and naltrexone.

Cannabinoids

Cannabinoid use is common in the United States, especially with increased legalization and medical marijuana prescribing. Chronic cannabinoid use can lead to cannabinoid hyperemesis syndrome, which causes patients to experience cyclic abdominal pain, nausea, and vomiting. Symptoms are often relieved by taking a hot shower. Supportive care with antiemetics, capsaicin, and haloperidol can also alleviate symptoms. Cessation of cannabis use resolves the syndrome.

Cocaine, Amphetamine, and Ketamine

Cocaine and amphetamines have vasoconstrictive properties that can contribute to bowel and hepatic ischemia. Cocaine can, rarely, cause pancreatitis. Treatment of these complications from cocaine and amphetamine use includes benzodiazepines. Chronic ketamine use can cause urologic dysfunction that leads to abdominal pain. Ketamine is also sometimes implicated in GI or biliary pathology. Treatment of abdominal pain from ketamine use includes drug cessation and supportive care.

In addition to treating abdominal pain in patients with substance use disorder, physicians should provide treatment and resources for their substance use disorder.

Antimicrobial Therapy for Pediatric Community-Acquired Pneumonia

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Reviewed by Andrew J. Eyre, MD, MS-HPEd

Objective

On completion of this article, you should be able to:

- Explain treatment recommendations for pediatric CAP based on results from the SAFER randomized clinical trial.

Pernica JM, Harman S, Kam AJ, et al. Short-course antimicrobial therapy for pediatric community-acquired pneumonia: the SAFER randomized clinical trial. *JAMA Pediatr.* 2021 May 1;175(5):475-482.

KEY POINTS

- Short-course antibiotic therapy appears comparable to standard care for previously healthy children with CAP who do not require hospitalization.
- Clinical practice guidelines should consider recommending 5 days of amoxicillin for pediatric pneumonia management in accordance with antimicrobial stewardship principles.

Community-acquired pneumonia (CAP) is a common occurrence in children, many of whom are brought to the emergency department for evaluation and treatment. Data to support optimal treatment duration for pediatric patients with CAP are limited, prompting further investigation in the Short-Course Antimicrobial Therapy for Pediatric Respiratory Infections (SAFER) study. The study sought to determine if 5 days of high-dose amoxicillin would lead to noninferior rates of clinical cure compared to the current standard of 10 days of high-dose amoxicillin.

The SAFER study was a blinded, noninferiority, randomized controlled trial conducted in the emergency departments of two children's hospitals in Canada. It included children with CAP aged 6 months to 10 years who were well enough to be treated as outpatients. The definition of CAP included all these criteria:

- Fever (>38°C [100.4°F] rectal) in the 48 hours before presentation;
- Tachypnea (for age), increased work of breathing, or findings on auscultation consistent with CAP;
- Chest x-ray findings consistent with CAP; and
- Primary diagnosis of CAP by an emergency physician.

Patients were excluded from the study for a variety of reasons including a history of conditions that predispose to severe disease or pneumonia with atypical microbiology (ie, preexisting pulmonary, heart, or kidney disease; neoplasms; immunodeficiency; or a history of aspiration events), antibiotic treatment prior to presentation, and the use of warfarin or tetracyclines. Children with a greater than 48-hour admission to the hospital in the preceding 2 months, a CAP diagnosis in the previous month, or a lung abscess in the previous 6 months were also excluded to avoid enrolling children with hospital-acquired or complicated pneumonia.

Participants were randomly assigned to receive either 5 or 10 days of amoxicillin therapy. The primary outcome was clinical cure, defined as initial improvement during the first 4 days after enrollment, significant improvement in dyspnea, no more than one fever spike, and lack of additional antibiotics or hospital admission. Secondary outcomes included the number of days the participant or caregiver missed work or school, number of days of mild adverse drug reactions, incidence of serious adverse drug reactions, participant adherence to the study's medications, and recurrence of presumed bacterial respiratory illness after the primary outcome visit in the month after enrollment. The study's investigators added another secondary cure outcome post hoc — a clinical cure that does not require additional intervention. It was added to account for patients who had more than one fever spike but no other worrisome symptoms, a situation that would have made them clinical failures within the trial but not necessarily in practice.

Results indicated that both the primary outcome (overall clinical cure at 14 to 21 days after enrollment) and secondary cure outcome (clinical cure that does not require additional intervention) were similar between the two groups. Caregiver work absenteeism was significantly lower in the intervention group than in the control group; all other secondary outcomes were similar between groups. In an intention-to-treat analysis, short-course treatment and clinical cure that does not require additional intervention were found to be statistically noninferior in all analyses. *The results of the trial suggest that short-course antibiotic therapy for CAP in children who do not require hospitalization is comparable to the current standard of care.*

Disclosure

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. This work was prepared as part of our official duties as military service members. Title 17 USC §105 provides that "copyright protection under this title is not available for any work of the United States Government." A United States Government work is defined in 17 USC §101 as a work prepared by a military service member or employee of the United States Government as part of that person's official duties.

Acute Atrial Fibrillation and Atrial Flutter Best Practices

By Nicolas Du Fayet De La Tour, DO; and Nicholas G. Maldonado MD, FACEP

University of Florida College of Medicine, Gainesville

Reviewed by Andrew J. Eyre, MD, MS-HPEd

Objective

On completion of this article, you should be able to:

- Explain the CAEP's recommendations for managing acute atrial fibrillation or flutter in the emergency department.

Stiell IG, de Wit K, Scheuermeyer FX, et al. 2021 CAEP acute atrial fibrillation/flutter best practices checklist. *CJEM*. 2021 Sep;23(5):604-610.

KEY POINTS

- The approach to acute atrial fibrillation and atrial flutter in the emergency department is similar: assessment, risk stratification, rate or rhythm control, stroke prevention, disposition, and follow-up care.
- For atrial fibrillation or flutter with rapid ventricular response secondary to an underlying medical cause, evaluate and treat the underlying cause, avoid aggressive rate control, and cardiovert only if unstable.
- For primary atrial fibrillation and atrial flutter, either rhythm or rate control strategies are acceptable.
- When indicated, OACs, preferably DOACs, should be considered and prescribed at discharge to reduce stroke risk.
- Patients with uncomplicated atrial fibrillation or flutter rarely require admission, but timely follow-up care should be arranged.

Atrial fibrillation and atrial flutter are common arrhythmias encountered in the emergency department. Although these conditions are distinct in their etiology, pathophysiology, ECG manifestations, and definitive treatments, their acute presentations are approached and managed similarly. In the featured LLSA article, an advisory committee of the Canadian Association of Emergency Physicians (CAEP) shares best practices for managing patients with symptomatic acute atrial fibrillation or flutter in the emergency department. The committee's primary focus is recent-onset atrial fibrillation and atrial flutter — defined as first-onset recurrent paroxysmal or recurrent persistent episodes — with a time of onset to emergency department presentation of less than 48 hours and up to 7 days in length.

Assessment and Risk Stratification

In patients with atrial fibrillation or flutter who have rapid ventricular response, the authors of the LLSA article recommend three key considerations in the initial assessment: (1) whether this presentation is due to a primary arrythmia or is secondary to an underlying medical cause (eg, sepsis or hypovolemia), (2) the degree of patient stability, and (3) the safety of cardioversion. If the cause of atrial fibrillation or flutter with rapid ventricular response is suspected to be secondary to an underlying medical cause, evaluate and treat the underlying cause, avoid aggressive rate control, and use caution in deciding to cardiovert. The degree of patient stability is determined by assessing for end-organ dysfunction, including signs and symptoms of shock, hypotension, myocardial ischemia, and pulmonary edema. For unstable patients, urgent electric cardioversion is advised if the onset of symptoms is less than 48 hours or if atrial fibrillation occurs with Wolff-Parkinson-White syndrome (rapid ventricular preexcitation). Otherwise, consider rate control first. The safety of cardioversion in patients with primary atrial fibrillation or flutter depends on several factors. The authors stated that it is safe to cardiovert if a patient has been adequately anticoagulated for a minimum of 3 weeks; adequacy is determined by physician judgment, confirmed compliance, or current or recent therapeutic medication levels (in the case of warfarin). Cardioversion may still be safe for patients who have not been adequately anticoagulated for more than 3 weeks if they have not had a stroke or transient ischemic attack, do not have valvular heart disease, *and* either symptom onset is less than 12 hours, symptom onset is between 12 and 48 hours, and their CHADS-65 criteria score is less than 2 or the evaluation for thrombus on transesophageal echocardiography is negative.

Rate and Rhythm Control

For patients with primary atrial fibrillation or flutter, either rhythm or rate control strategies with shared decision-making are acceptable. When deemed safe, the authors prefer rhythm control. If a rate control strategy is chosen,

Medication	Initial Intravenous Doses	Subsequent Doses	Discharge Oral Doses	Pearls and Pitfalls
Diltiazem (calcium channel blocker)	0.25 mg/kg IV over 10 min Repeat every 15-20 min at 0.35 mg/kg up to three doses	Start 30-60 mg PO within 30 min of effective intravenous rate control	Discharge on 30-60 mg four times daily or extended release 120-240 mg daily	Avoid in acute heart failure or known left ventricular dysfunction
Metoprolol (β-blocker)	2.5-5 mg IV over 2 min Repeat every 15-20 min up to three doses	Start 25-50 mg PO within 30 min of effective IV rate control	Discharge on 25-50 mg twice daily	Caution if hypotensive
Digoxin	0.25-0.5-mg loading dose	0.25 mg IV every 4-6 hr to max of 1.5 mg over 24 hr	N/A	Caution in renal insufficiency or failure

TABLE 1. Rate control strategies for acute atrial fibrillation or flutter in the emergency department

calcium channel and β-blockers are considered first line; which drug is selected depends on the drug classes the patient takes at home, other patient factors, and physician preference. Initial doses should be intravenous until rate control is achieved, followed by oral therapy within 30 minutes of effective intravenous rate control. The goal heart rate for effective control is less than 100 bpm at rest or less than 110 bpm when walking. Calcium channel blockers should be avoided in patients with acute heart failure or known left ventricular dysfunction. Digoxin is second line but becomes first line for acute heart failure or hypotension; however, it should be used with caution in patients with renal insufficiency or failure. Table 1 summarizes rate control strategies for acute atrial fibrillation or flutter.

If a rhythm control strategy is chosen, either pharmacologic or electric cardioversion is acceptable, and the alternative can be used if the initial approach with one is unsuccessful. For pharmacologic cardioversion, procainamide is preferred over amiodarone. Electric cardioversion can be attempted if proper staffing, equipment, and procedural sedation are available. Table 2 summarizes rhythm control strategies.

For patients with atrial fibrillation or flutter and Wolff-Parkinson-White syndrome, the authors note additional key considerations. Atrioventricular nodal blocking agents are contraindicated in these patients because these agents can significantly worsen their condition. Urgent electric cardioversion can be used in these patients if they are unstable; intravenous procainamide can be used if they are stable.

Stroke Prevention

Patients with acute atrial fibrillation or flutter are at risk of embolic stroke — the authors provide guidance on oral anticoagulants (OACs) for stroke prevention. In general, OACs should be prescribed at discharge when indicated. Key considerations include the patient's risk of stroke based on validated assessment tools, the risk of bleeding balanced with the benefit of stroke prevention, and the choice, dose, and duration of OACs based on patient factors.

To assess stroke risk, the authors used the CHADS-65 score. To assess bleeding risk, they recommend reviewing OAC contraindications on the McMaster checklist, along with shared decision-making that includes patient preferences after they receive education on the risks and benefits of OACs. Commonly prescribed OACs and doses are summarized in Table 3. The authors recommend long-term OAC therapy for atrial fibrillation and atrial flutter patients who meet any positive CHADS-65 criteria and

Pharmacologic Cardioversion
Medication: Intravenous procainamide — 15 mg/kg in 500 mL normal saline over 60 min, maximum 1,500 mg **Pearls and pitfalls:** Avoid if systolic blood pressure <100 mm Hg or QTc >500 msec Pause infusion if hypotensive or QRS lengthens >30% Check QTc after cardioversion.
Electric Cardioversion
Procedure: Direct current cardioversion — apply pads in either anterolateral or anteroposterior position, avoiding sternum and breast tissue. Synchronize and deliver 150-200 J. **Pearls and pitfalls:** Ensure appropriate equipment, staffing, and procedural sedation per local protocol. Avoid starting low energy. If failure, try alternative pad positioning

TABLE 2. Rhythm control strategies for acute atrial fibrillation or flutter in the emergency department

Class	Medication	Dose	Dose Adjustments and Comments
Factor Xa inhibitors	Apixaban	5 mg PO twice daily	Use 2.5 mg PO twice daily if at least two factors are present: (1) serum creatinine >133 µmol/L (2) age >80 yr, (3) weight <60 kg
	Rivaroxaban	20 mg PO daily	Use 15 mg PO daily if creatinine clearance is 30-49 mL/min
	Edoxaban	60 mg PO daily	Use 30 mg PO daily if creatinine clearance is 30-50 mL/min or <60 kg Consider drug-drug interactions
Direct thrombin inhibitor	Dabigatran	150 mg PO twice daily	Use 110 mg PO twice daily if age >80 yr, or >75 yr with bleeding risk
Vitamin K antagonist	Warfarin	5 mg PO daily	1-2 mg PO daily if patient is frail, has a low weight, or is of Asian descent Arrange for INR testing after two to four doses, with outpatient dose adjustment to therapeutic level

TABLE 3. OAC classes, medications, and dosing for stroke prevention in atrial fibrillation or flutter in the emergency department

recommend discontinuing aspirin in patients who have stable coronary or peripheral artery disease while on OACs. They also recommend short-term (4 wk) OAC therapy for all transesophageal echocardiography–guided cardioversion and consideration of short-term OAC therapy for patients negative for the CHADS-65 criteria. Patients should take 81 mg of aspirin daily if they are negative for the CHADS-65 criteria and have stable coronary, aortic, or peripheral vascular disease. Patients who spontaneously cardiovert in the emergency department should receive short- or long-term OAC therapy according to the CHADS-65 criteria.

In general, direct OACs (DOACs) are preferred over warfarin. Warfarin is the preferred OAC for patients with mechanical valves, moderate to severe mitral stenosis, or severe renal impairment (creatinine clearance <30 mL/min). According to the authors, nephrology and hematology should be consulted when initiating OAC therapy in patients with severe renal impairment.

Disposition and Follow-up Care

The authors provide several recommendations on discharge and follow-up care. For uncomplicated atrial fibrillation or flutter, admission is rarely needed. If patients remain significantly symptomatic or have acute coronary syndrome or acute heart failure despite treatment, they should be admitted. When sinus rhythm is achieved in a patient, prescriptions for calcium channel blockers and β-blockers are not needed at discharge. Close follow-up care within 7 days is recommended if warfarin or rate control medications are initiated at discharge. For all other patients, follow-up care with cardiology or internal medicine within 4 to 6 weeks should be arranged.

High-Risk Airway Management

By Jai Krish Gill, MD; and Nicholas G. Maldonado MD, FACEP

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Reviewed by Andrew J. Eyre, MD, MS-HPEd

Objective

On completion of this article, you should be able to:

- Discuss recommendations for emergent airway management in high-risk patient populations.

Lentz S, Grossman A, Koyfman A, Long B. High-risk airway management in the emergency department: diseases and approaches, part II. *J Emerg Med.* 2020 Oct;59(4):573-585.

KEY POINTS

- Prepare for anatomic and physiologic challenges associated with emergency airway management for high-risk clinical conditions to ensure success and minimize risk.
- Resuscitating and stabilizing, optimizing hemodynamics, and maintaining physiologic parameters are critical while performing airway management to avoid deterioration.
- Alternative approaches to patient positioning, preoxygenation, and initial device selection can prevent rapid desaturation, improve glottic view, and mitigate harm in certain conditions.

Airway management is a core component of emergency medicine, with a structured algorithm engrained in emergency medicine education and training. This algorithm stresses adequate preparation for anatomic and physiologic challenges that may arise during emergency airway management. First-attempt success and minimizing the risk of adverse outcomes are ultimate goals. Evidence is mounting that identifies certain clinical conditions that pose an increased risk of harm during or immediately after airway management; each of these conditions needs its own nuanced approach. The authors of the featured LLSA article discuss their key considerations and recommendations when performing airway management for seven high-risk clinical conditions: trauma and hemorrhagic shock, elevated intracranial pressure, upper GI bleeding, cardiac tamponade, aortic stenosis, morbid obesity, and pregnancy.

Trauma and Hemorrhagic Shock

In critically ill trauma patients with hemorrhagic shock, three key considerations are (1) ensuring adequate resuscitation and preoxygenation prior to an intubation attempt, (2) maintaining cervical spine immobilization when injury is suspected, and (3) anticipating the risk of anatomic distortion and debris in cases of head, face, or neck trauma. The authors highlight evidence showing that preintubation hypotension and a shock index (heart rate or systolic blood pressure) ≥0.8 to 0.9 are predictors of postintubation decompensation and cardiac arrest. When performing airway management in these patients, they recommend rapid sequence intubation (RSI) as the preferred method, aggressively resuscitating patients with hypotension or a shock index ≥0.8 to 0.9, and using a hemodynamically neutral or appropriately dosed induction agent. In cases of suspected cervical spine injury, physicians should use manual in-line cervical spine immobilization, anticipate a difficult view, and consider video laryngoscopy or direct laryngoscopy with a bougie as the initial approach. In cases of head, face, or neck trauma, the risk of airway distortion and obstruction should be anticipated and, thus, adequate suction, advanced airway equipment (ie, fiberoptic scope), and a surgical airway for a "can't oxygenate, can't ventilate" scenario should be made available.

Elevated Intracranial Pressure

The authors highlight the risk of secondary brain injury and poor neurologic outcomes associated with hypoxia, hypercapnia, and hypotension in patients with known or suspected elevated intracranial pressure. When elevated intracranial pressure is suspected, efforts should be made to maintain oxygen saturation above 94%, $PaCO_2$ between 35 and 45 mm Hg, and MAP above 80 mm Hg prior to, during, and after intubation. The authors recommend achieving these levels by using RSI, ensuring adequate preoxygenation, and performing bag-valve-mask ventilation if indicated.

They also recommend aggressively avoiding hypotension, avoiding further elevation of intracranial pressure, and avoiding decreasing cerebral perfusion pressure. Choose a hemodynamically neutral induction agent such as etomidate. Pretreatment with fentanyl 2 to 3 µg/kg should also be considered, particularly for hypertensive patients. Pretreatment with lidocaine is no longer universally recommended. Ketamine is an alternative induction agent that is shown to be safe in this patient population despite prior concerns that it may elevate intracranial pressure. Propofol should be used with caution given its risks of hypotension and decreased

cerebral perfusion. In addition to considering a paralytic agent's routine contraindications, physicians should also consider whether serial neurologic examinations are required with an agent. Succinylcholine has the benefit of rapid onset and short duration. If rocuronium or another nondepolarizing agent with a longer duration is used, the reversal agent sugammadex can be considered after intubation to facilitate neurologic examination. After intubation, the head of the bed should be elevated ≥30° and ventilator settings should be closely monitored to maintain $PaCO_2$ between 35 and 45 mm Hg and oxygen saturation above 94%, while avoiding hyperoxia.

Upper GI Bleeding

For patients with upper GI bleeding, the authors' key considerations are the risk of shock, aspiration, and a blood-obscured glottic view. They caution against routinely intubating these patients prior to endoscopy and, instead, recommend considering intubation for specific cases like massive hematemesis or altered mental status that prevents patients from protecting their airway. When airway management is needed, the preparations for shock are the same as those for trauma patients: Aggressively resuscitate patients with hypotension or a shock index ≥0.8 to 0.9 prior to intubation. Aspiration of blood is anticipated in patients with upper GI bleeds; to reduce their risk of aspiration, these patients should be positioned more upright during intubation. The risk of a blood-obscured glottic view can be reduced by placing a nasogastric tube prior to intubation in select cases. Readily available dual suction and direct laryngoscopy for visualization are other considerations when anticipating a blood-obscured glottic view. The authors discuss the suction-assisted laryngoscopy and airway decontamination (SALAD) technique, which involves placing suction within the esophagus during intubation and is particularly helpful when there is a large amount of active bleeding.

Cardiac Tamponade

The authors note that there is a paucity of literature on emergency airway management in patients with cardiac tamponade. They discuss the hemodynamic effects of cardiac tamponade and how the various stages of airway management can lead to decompensation in these patients. They state that induction agents can blunt sympathetic tone, diminish compensatory tachycardia and vasoconstriction, and depress myocardial function. They also state that positive pressure ventilation can increase intrathoracic pressure, thereby reducing preload and cardiac output, which risks hemodynamic collapse and cardiac arrest. For these reasons, intubation in these patients should be delayed until definitive treatment is available, or emergent pericardiocentesis should be performed with local anesthesia prior to intubation if possible. If intubation is unavoidable, the authors recommend optimizing preload with small intravenous fluid boluses (250-500 mL), considering awake intubation with ketamine to maintain spontaneous respirations, and using ventilator settings with low tidal volumes and positive end-expiratory pressures.

Aortic Stenosis

The authors also discuss the hemodynamic effects of aortic stenosis and the goals of maintaining adequate intravascular volume, preload, systemic vascular resistance, and normal sinus rhythm and avoiding tachy- and bradycardia during airway management. Like with cardiac tamponade patients, the phases of airway management risk reducing preload and cardiac output and can precipitate hemodynamic collapse in patients with aortic stenosis. Three key recommendations for airway management in patients with aortic stenosis are (1) optimizing preload with small intravenous fluid boluses prior to induction and using hemodynamically neutral and appropriately dosed induction agents to avoid preload reduction, (2) treating brady- and tachydysrhythmias prior to intubation (including cardioversion when indicated), and (3) having a push-dose vasopressor or vasopressor infusion ready to treat hypotension before and after intubation. Phenylephrine is the vasopressor of choice for patients with aortic stenosis.

Morbid Obesity

Patients with morbid obesity have physiologic and anatomic features that affect oxygen reserve, bag-valve-mask ventilation, and glottic view. Appropriate positioning during preoxygenation and intubation is key for these patients. Some literature suggests that positioning these patients in a ≥25° head-elevated position or a reverse Trendelenburg during preoxygenation can delay desaturation during apnea. The authors recommend using noninvasive positive pressure ventilation for preoxygenation. If bag-valve-mask ventilation is needed between intubation attempts, anticipate difficulty and use a two-person, two-handed technique. Instead of using the standard "sniffing" position for intubation, place blankets or pillows behind the back, neck, and head of patients with morbid obesity to align their external auditory canal with the sternal notch, which may improve glottic views during intubation attempts. A supraglottic airway device should be available for airway rescue breathing because these patients are anticipated to have more rapid desaturation, more difficulty with bag-valve-mask ventilation, and less clear glottic views.

Pregnancy

The authors report higher rates of failed intubation in pregnant patients compared with nonpregnant patients. This finding is explained by the physiologic and anatomic changes during pregnancy that can lead to difficulty with glottic views, difficulty with passing an endotracheal tube, reduced oxygen reserve, and an increased aspiration risk. Their recommendations for these patients include:

- Preparing for a difficult airway with appropriate backup equipment and personnel;
- Anticipating rapid desaturation and providing adequate preoxygenation before induction and apneic oxygenation after induction;
- Positioning patients with the head of the bed elevated between 20° and 30° to minimize accelerated desaturation and the risk of aspiration;
- Considering displacing the uterus to the left side to improve preload; and
- Using a smaller endotracheal tube (7.0 mm) to improve passage.

Diagnosis and Treatment of Sexually Transmitted Infections

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Objective

On completion of this article, you should be able to:

- Discuss the diagnosis and treatment of common STIs encountered in the emergency department.

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KEY POINTS

- Many STIs have overlapping urogenital symptoms, and their extragenital symptoms can mimic other common emergency department presentations.
- Emergency physicians should be familiar with first-line treatments for common STIs; factors that affect treatment dose, duration, and route of administration; and factors that affect the need for alternative therapy.
- Most patients with STIs are asymptomatic; thus, screening interventions and safe sex practices for sexually active patients should be encouraged. Concurrent treatment of these patients' sex partners is also recommended.

Sexually transmitted infections (STIs) are frequently encountered in the emergency department. A cause for public health concern, STIs were estimated to occur in nearly 1 in 5 adults in 2018, with rates of gonorrhea, chlamydia, and syphilis on the rise. Additionally, a pattern of antimicrobial resistance against certain STIs may limit therapeutic options. STIs are also associated with HIV acquisition and transmission. The current diagnosis and treatment of STIs is highly relevant to emergency medicine because many of these patients seek care in the emergency department. This review summarizes current evidence on the epidemiology, diagnosis, treatment, and prevention of commonly encountered STIs. Readers are encouraged to review current guidelines for dosing of the first-line and alternative treatments mentioned. Overall, most patients with STIs are asymptomatic. Screening interventions and safe sex practices for sexually active patients should be encouraged. Concurrent treatment of these patients' sex partners is also recommended.

Gonorrhea

Gonorrhea has increased in all US regions in both men and women. The highest rates are in Black patients and patients aged 20 to 24 years. Gonorrhea can lead to urogenital, extragenital (ie, ocular, oropharyngeal, rectal), and disseminated infections. The majority of patients with genital and extragenital gonococcal infections are asymptomatic. When patients are symptomatic, clinical manifestations depend on the site of involvement. Gonococcal urethritis presents with dysuria and can include urethral discharge in men. Urogenital disease in women may present with vaginal pain, itching, discharge, bleeding, or pelvic inflammatory disease (PID) (which includes symptoms of lower abdominal pain and dyspareunia). Gonococcal conjunctivitis presents with ocular pain, conjunctival injection, and purulent discharge; the condition should be suspected in symptomatic infants born to infected mothers. Gonococcal pharyngitis — with symptoms that include sore throat, pharyngeal exudates, and cervical adenitis — may be indistinguishable from other forms of pharyngitis. Gonococcal proctitis should be considered in patients with anorectal pain, discharge, bleeding, and tenesmus. Disseminated gonococcal infection can cause purulent arthritis and should be highly suspected in select patients with monoarticular joint pain and a hot, swollen joint. Disseminated disease can also manifest as arthritis-dermatitis syndrome, meningitis, and endocarditis.

The diagnostic test of choice for gonococcal infections is the nucleic acid amplification test (NAAT), which has high specificity. Samples for the test should be taken from the sites involved. For diagnosing urogenital infections, clinician-collected or self-collected vaginal swabs are preferred for female patients and first-catch urine samples are preferred for male patients; endocervical or urethral swabs are also acceptable. Cultures should be collected for susceptibility testing when antimicrobial resistance is suspected (ie, for cases that have failed treatment).

Ceftriaxone is the antimicrobial of choice for all gonococcal infections; however, dose and route of administration vary based on the site involved. Alternative therapy may be required in patients with cephalosporin

allergy. Because of antimicrobial resistance patterns, fluoroquinolones and macrolides are no longer recommended. Cephalosporin- and multidrug-resistant gonococci are emerging.

Chlamydia

Among the notifiable STIs, *Chlamydia trachomatis* is the most common in the United States. Rates are increasing in young men and women. The highest rates are in young women aged 20 to 24 years, and prevalence is higher in women and Black patients. Like gonorrhea, chlamydia can lead to urogenital and extragenital (ie, ocular, oropharyngeal, and rectal) infections; most infections at any site are asymptomatic, and clinical manifestations depend on the site involved. Also, symptoms of chlamydia mimic gonorrhea — urogenital symptoms include urethritis, epididymitis, cervicitis, and PID, and extragenital symptoms include conjunctivitis, pharyngitis, and proctitis. Reactive arthritis and perihepatitis (Fitz-Hugh–Curtis syndrome) are unique complications of chlamydial infections. Infants born to infected mothers are at risk of conjunctivitis and pneumonia. *Chlamydia trachomatis* serovars L_1 through L_3 can cause a unique STI, lymphogranuloma venereum (LGV). LGV is marked by an initial painless ulcer that can progress to enlarged, painful inguinal adenopathy; it should be suspected in patients with painless genital ulcers or an inguinal abscess, especially if those patients belong to a group with higher LGV prevalence (ie, men and transgender women who have sex with men).

Because of their similar presentations, testing for chlamydia and gonorrhea should occur concomitantly. The diagnostic test of choice for chlamydial infections is also a NAAT, with samples taken from the sites involved. Doxycycline is the antimicrobial of choice for all chlamydial infections; however, dose duration varies for severe chlamydial proctitis and LGV. Alternative therapy is required for patients allergic to doxycycline and for pregnant patients. Because of results from comparative trials, azithromycin is no longer first line compared to doxycycline.

Syphilis

Syphilis rates are also increasing in the United States, with an alarming rise in congenital syphilis through maternal transmission during pregnancy. Prevalence is higher in Black patients, men and transgender women who have sex with men, and patients with HIV. The spirochete *Treponema pallidum* causes syphilis. Clinical manifestations vary based on the characteristic stages (ie, primary, secondary, tertiary). In its early stage, the infection can go unrecognized, and during its latency period, patients may be asymptomatic. At any stage, however, ocular, otic, and CNS manifestations can occur.

Days to weeks after exposure, primary syphilis manifests as a painless genital ulcer with inguinal adenopathy but can also be associated with multiple shallow and painful ulcers. Left untreated, secondary syphilis can occur 4 to 10 weeks later, with signs and symptoms of a macular rash (with or without associated joint pain), abdominal pain, and hair loss. Importantly, however, the classic maculopapular rash, which involves the palms and soles, does not occur in all patients with secondary syphilis. A period of latency, known as latent syphilis, can occur and is characterized by seroreactivity without clinical manifestations of infection. Some patients maintain lifelong latency after exposure to the spirochete, while others develop symptomatic infections after a latency period. Tertiary syphilis is a late symptomatic disease that can develop years to decades after the initial infection. Tertiary syphilis includes gummatous, cardiac, psychiatric, or neurologic manifestations (eg, meningitis, general paresis, and tabes dorsalis).

Confirmatory testing depends on the stage and site of infection. Primary syphilis can be confirmed with direct detection of *T. pallidum* (ie, via dark-field microscopy or polymerase chain reaction) in lesion exudates, whereas later stage infection requires algorithmic interpretation of the serologic tests, treponemal antibody tests (*T. pallidum* particle agglutination assay) and nontreponemal antiphospholipid antibody tests (rapid plasma reagin or venereal disease research laboratory test). A diagnosis of neurosyphilis requires CSF interpretation and CSF serologic testing. Penicillin is the antimicrobial of choice for all stages of syphilis; however, the dose depends on the stage, and route of administration varies between ocular, otic, and neurosyphilis. In allergic patients, penicillin desensitization may be required.

***Mycoplasma genitalium* Infection**

M. genitalium appears to predominantly affect the urogenital tract in either sex and is linked to cases of acute, persistent, or recurrent urethritis in men and cervicitis, PID, and infertility in women. Asymptomatic carriage may be common. Urogenital infections should be diagnosed using a NAAT with clinician-collected or self-collected vaginal swabs preferred for female patients and first-catch urine samples preferred for male patients; endocervical and urethral swabs are also acceptable. Sequential treatment with doxycycline followed by moxifloxacin is the treatment of choice for *M. genitalium*. A high prevalence of macrolide resistance limits the use of macrolides. Treatment failure to moxifloxacin has been reported.

Genital Herpes

Genital herpes can be caused by herpes simplex virus types 1 (HSV-1) and 2 (HSV-2). Fortunately, infection rates are declining. HSV-1 predominantly causes orolabial disease, but oral-genital and genital transmission of HSV-1 can lead to genital herpes. HSV-2, by contrast, predominantly causes anogenital disease. Most infected patients are asymptomatic. Symptoms can occur as part of a first-onset initial infection or a less severe recurrent infection. Extragenital complications can also occur, including keratitis, meningitis, and encephalitis. Symptomatic patients typically present with multiple painful vesicular genital or anal lesions that may be associated with lymphadenopathy or constitutional symptoms.

Herpes is often diagnosed clinically, but a NAAT for HSV performed on swabs from lesions confirms the diagnosis. For asymptomatic patients, serologic testing is available to detect HSV-1 or HSV-2 antibodies. Although no cure

for herpes is available, antiviral therapy with valacyclovir, acyclovir, or famciclovir may reduce symptoms, symptom duration, and rates of recurrence. Which antiviral is selected depends on dose frequency and costs. Treatment duration differs based on whether the presentation is initial or recurrent.

Trichomoniasis

Trichomonas vaginalis, the causative agent of trichomoniasis, is the most prevalent nonviral STI in the United States. In contrast to most other STIs, trichomoniasis is more prevalent in women older than 40 years and is rare among men and transgender women who have sex with men. Most infected patients are asymptomatic. Trichomoniasis affects the urethra, epididymis, and prostate in men and the cervix, urethra, vagina, and genital glands in women. Symptomatic disease manifests as pain and inflammation of the affected region. The stereotypical "strawberry cervix" in women is suggestive of trichomoniasis. A NAAT for *Trichomonas* is the diagnostic test of choice, with sample collection similar to collections for gonorrhea, chlamydia, and *M. genitalium*. In women, microscopy of vaginal secretions (ie, wet mount) can assist with diagnosis but has low sensitivity. Metronidazole is the treatment of choice, with higher doses and longer duration of therapy recommended if initial treatment fails. Refractory cases may occur, in which case nitroimidazole resistance should be suspected.