The LLSA Literature Review

Synopses of articles from ABEM’s 2019 Lifelong Learning and Self-Assessment Reading List

FROM THE EDITORS

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

From December 2018 through December 2019, each monthly issue of Critical Decisions has provided a summary of one of the articles from ABEM’s 2019 reading list, with bullets highlighting the elements relevant to emergency medicine practice. This online supplemental issue includes a full collection of those summaries, which are intended to highlight the important concepts of each article. We are pleased to offer this benefit FREE to ACEP members, and hope you find it useful. ACEP members also can download full versions of the articles by logging in at acep.org/llsa.

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

Best wishes,

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Critical Decisions in Emergency Medicine is the official CME publication of the American College of Emergency Physicians. Additional volumes are available.

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ISSN2325-0186(Print) ISSN2325-8365(Online)
New-Onset Seizures

By Neil A. Ray, MD; and Michael E. Abboud, MD
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Reviewed by Andrew J. Eyre, MD, MHPEd


Seizures are globally defined as transient episodes of abnormal activity triggered by excessive or synchronous central nervous system activation. These events can range from staring spells to generalized tonic-clonic movements. Epilepsy, subsequently, is defined as two or more unprovoked seizures that occur more than 24 hours apart, or a single unprovoked seizure with a high risk of recurrence within the next 10 years.

The first step in the acute evaluation of a new-onset seizure is to distinguish the diagnosis from “mimics” such as transient ischemic attacks, complex migraines, syncope, and psychogenic nonepileptic seizures. Seizures can often be diagnosed based on the patient’s history, physical examination, and any witness reports. Risk factors for the disorder include excessive sleep deprivation, illicit drug or alcohol use, metabolic derangements, toxin exposures, organ failure, and certain medications.

Any patient with a new-onset seizure should undergo neuroimaging to identify lesions that could have contributed to the event. This is especially important for those who present with new neurological deficits, prolonged altered mental status, recent trauma, or a prolonged headache, or if a structural brain lesion is suspected. While CT scans are frequently employed and may be sufficient, they can miss important lesions. MRI is more sensitive than CT for detecting intracranial abnormalities (30% vs 10%, respectively).

An electroencephalogram (EEG) should be considered for patients who do not return to their neurological baseline within 30 to 60 minutes after the seizure ends, and for anyone with a focal neurological deficit or a level of consciousness that waxes and wanes. For patients who have fully recovered, an EEG can be safely deferred to an outpatient setting. A lumbar puncture should be reserved for cases that are concerning for encephalitis, subarachnoid hemorrhage, or meningitis.

Two-thirds of patients with new-onset seizures do not necessitate treatment. In addition, the delayed initiation of antiepileptic agents has had no effect on long-term prognosis when compared to immediate initiation. Antiepileptic medications can be classified as broad spectrum (effective for most generalized and focal seizures) and narrow spectrum (more effective for focal seizures). The adverse effects associated with these drugs usually occur within the first few days of therapy and include somnolence, dizziness, blurry vision, difficulties with concentration, memory impairment, and skin rashes. Medication decisions should be made with the help of a neurologist and/or hospital guidelines.

Patients with new-onset seizures should be carefully counseled about the various activities that can trigger these events or lower the seizure threshold. In addition, clinicians must emphasize the importance of avoiding potentially lethal activities, including swimming, taking baths, operating heavy machinery, climbing, and using ladders. Each state has specific laws and regulations that may limit driving after a seizure. Patients should be counseled accordingly, and physicians should remain aware of the state-specific laws for mandated reporting.

**KEY POINTS**

- Seizures should be distinguished from mimics such as transient ischemic attacks, complex migraines, syncope, and psychogenic nonepileptic seizures.
- MRI is more sensitive than CT for detecting intracranial abnormalities (30% vs 10%, respectively).
- An EEG should be considered for patients who do not return to their neurological baseline within 30 to 60 minutes, have a focal neurological deficit, or have a waxing/waning level of consciousness.
- Decisions regarding antiepileptic drugs should be made with the help of a neurologist and/or hospital guidelines.
- Patient education and counseling can help minimize the risk of recurrence.
Physician Burnout

By Alexa Gips, MD; and Laura Welsh, MD; Boston University, Boston, Massachusetts
Reviewed by Andrew J. Eyre, MD, MHPEd


Physician burnout, defined by exhaustion, cynicism, and decreased effectiveness, affects at least 50% of US physicians. Strategically savvy health care organizations must tackle this escalating syndrome, which can diminish patient safety and satisfaction.

Preventative approaches must reach beyond individual physician tactics (eg, mindfulness, stress-management workshops) to address institutional solutions. Organizational strategies to reduce common stressors and promote well-being include:

1. Physician burnout and well-being must be publicly recognized by hospital leaders and regularly evaluated. Standardized tools should be used to compare physician satisfaction rates to national benchmark data.

2. While many aspects of burnout are universal, others are unique to certain specialties and environments. A uniform assessment allows organizations to identify and engage units that are at greatest risk.

3. Effective leadership can significantly reduce burnout and improve work satisfaction. Organizations must choose and train competent leaders and continuously measure their performance.

4. Physicians need peer support. These important personal connections can be facilitated through space (eg, inclusive meeting rooms stocked with food and computers) or time (eg, protected hours for small physician groups or shared meals).

5. Evidence points to productivity-based compensation as a risk factor for burnout. Other options worthy of consideration include salaried models and productivity-based approaches that encourage self-care.

6. Similarly, organizations should enact policies that emphasize work-life balance. This includes flexible work schedules, ample vacation time, and time off for important life events.

7. Health care organizations must be mindful to align their actions with their stated mission. The extent to which an institution is living up to its claimed values can be evaluated by asking its staff. These important, collaborative conversations can help identify any potential misalignments.

8. Any strategy that places the burden of combating burnout solely on the physicians themselves is doomed to fail. Organizations can supply their clinicians with tools to objectively assess and foster their own well-being while providing relevant training in areas like resilience, narrative medicine, and mindfulness.

9. Cutting-edge programs should focus on the creation of effective strategies that can be adopted by other organizations.

KEY POINTS

- Physician burnout is a significant, growing issue that negatively affects patient satisfaction and safety.
- Burnout should not be viewed as an individual physician responsibility but rather as a systemwide problem that requires a strategic response.
- Organizations can tackle physician burnout effectively without compromising their other core goals or financial health.
Unexpected deliveries are rare, albeit stressful, emergency department events for which clinicians must always be prepared. Although the vast majority of newborns (90%) require minimal interventions (eg, warming, drying, or stimulation) as they transition to the extrauterine environment, emergency physicians must recognize when and how to resuscitate the remaining 10%.

In November 2015, the American Heart Association (AHA) published new guidelines that highlight important factors that must be addressed in the initial management of newborns, including the infant’s gestational age, tone, and respiratory effort. If these three components are reassuring (ie, the patient is a full-term, crying newborn with good tone), the neonate can likely be placed with the mother and routine care may be continued. However, if these elements are concerning or cannot be adequately addressed, further investigation and intervention are appropriate (Figure 1).

As with any resuscitation, it is paramount to effectively manage the neonate’s airway, breathing, and circulation.

**Airway**

Deep suctioning of the nasopharynx should generally be avoided given the potential risk of bradycardia or a vagal response. In addition, the updated guidelines discourage routine tracheal intubation for depressed newborns with meconium-stained amniotic fluid. The recommendations emphasize the importance of augmenting the patient’s respiratory effort with positive-pressure ventilation (PPV). Intubation can be considered if there is no improvement.

**Breathing**

Clinicians should evaluate the patient’s heart rate and supplement with PPV when indicated. Specifically, PPV with a bag-valve-mask (BVM) should be initiated if the heart rate falls below 100 bpm or the patient’s respiratory effort is inadequate. Preductal oxygen monitoring, which provides up-to-date oxygen saturation levels, can be achieved by placing a pulse oximetry probe on the right upper extremity. If the infant’s heart rate remains low and/or saturations do not improve, it may be appropriate to place an advanced airway using an endotracheal tube (ETT) or laryngeal mask airway.

**Circulation**

Careful circulatory monitoring is a critical component of newborn resuscitation. The patient’s heart rate should be monitored with a three-lead ECG. If the heart rate remains below 60 bpm despite adequate ventilation and the placement of an advanced airway, chest compressions should be initiated (3 compressions to 1 breath). The two-thumb compression technique is preferred. If bradycardia persists,
Although 90% of neonates require relatively minimal interventions, emergency clinicians must be prepared to employ additional resuscitation efforts, if needed. The clinician should evaluate for other reversible causes and prepare for the potential administration of medication.

Emergency clinicians must be prepared to employ additional resuscitation efforts, if needed. Further resuscitation efforts include the administration of epinephrine (IV or ETT), crystalloid solution, and blood (10 mL/kg) if there is concern for hypovolemia.

**Additional Considerations**

Delayed umbilical cord clamping (30-60 seconds after birth) is recommended for uncomplicated deliveries. Although newborn temperatures should typically remain between 36.5°C (97.7°F) and 37.5°C (99.5°F), there may be specific circumstances in which therapeutic hypothermia is indicated (eg, when there is a concern for hypoxic-ischemic encephalopathy).

Glucose monitoring may be especially appropriate for infants with risk factors for glucose dysregulation. Newborns should undergo prenatal laboratory tests and additional standard-of-care treatments, including the administration of vitamin K and erythromycin eye drops; however, these interventions do not need to occur in the emergency department. In addition, Apgar scores should be assessed and documented.

**KEY POINTS**

- Although 90% of neonates require relatively minimal interventions, emergency clinicians must be prepared to employ additional resuscitation efforts, if needed.
- Management of the patient’s airway, breathing, and circulation remain essential components of newborn resuscitation.
- Monitoring should include the use of a three-lead ECG and oxygen saturation probe (placed on the right upper extremity).
- PPV with a BVM is a cornerstone of neonatal management and should be initiated if the patient’s heart rate drops below 100 bpm.
- Cardiopulmonary resuscitation (3 compressions to 1 breath) should be initiated if the neonate’s heart rate drops below 60 bpm.

**AHA Neonatal Resuscitation Algorithm — 2015 Update**

1. **Antenatal counseling**
   - Team briefing and equipment check

2. **Birth**
   - Team gestation?
     - Good tone?
     - Breathing or crying?

3. **Warm infant and maintain normal temperature, position airway, clear secretions if needed, dry, stimulate**

4. **Apnea or gasping? HR below 100 bpm?**
   - NO
   - YES

5. **Consider ECG monitor**

6. **PPV, SpO2 monitor, Consider ECG monitor**

7. **HR below 100 bpm?**
   - NO
   - YES

8. **Check chest movement, Ventilation corrective steps if needed**
   - ETT or laryngeal mask if needed

9. **HR below 60 bpm?**
   - NO
   - YES

10. **Intubate if not already done**
    - Chest compressions
    - Coordinate with PPV
    - 100% O2
    - ECG monitor
    - Consider emergency UVC

11. **HR below 60 bpm?**
    - NO
    - YES

12. **IV epinephrine if HR persistently below 60 bpm**
    - Consider hypovolemia
    - Consider pneumothorax

**Targeted Preductal SpO2 After Birth**

<table>
<thead>
<tr>
<th>Time (min)</th>
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<td>60%-65%</td>
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<tr>
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<td>65%-70%</td>
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<tr>
<td>3</td>
<td>70%-75%</td>
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<tr>
<td>4</td>
<td>75%-80%</td>
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<tr>
<td>5</td>
<td>80%-85%</td>
</tr>
<tr>
<td>10</td>
<td>85%-95%</td>
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</tbody>
</table>
Fire-Related Inhalation Injury

By Daniel Tonellato, MD; and Andrew Eyre, MD, MHPED
Massachusetts General Hospital, Brigham and Women’s Hospital, and Harvard Medical School, Boston, Massachusetts

Insidious and pathophysiologically complex, fire-related inhalation injuries can be particularly challenging to diagnose and manage. An estimated 5% of burn patients who require hospital admission have an associated inhalation injury, which is an independent risk factor for death. In addition to direct thermal injuries, further damage can be inflicted by inhaled gases or toxins.

Any patient who has been exposed to fire within an enclosed space should be evaluated for inhalation injuries, which are generally diagnosed based on a careful history and physical examination. While not entirely diagnostic, physical findings such as burns near the mouth or nose, stridor, voice changes, wheezing, black or carbonaceous sputum, burned nasal hairs, and visible soot in the airways can indicate an inhalation injury. Flexible bronchoscopy can be used to better visualize the airway and oropharyngeal structures. Mucosal burns, sloughing, erythema, edema, or visible debris can also suggest an inhalation injury. While CT scans and plain films may be used to evaluate these cases, initial chest x-rays are generally nondiagnostic.

Inhalation injuries do not always require intubation, particularly if airway patency appears to be unthreatened and skin burns cover less than 20% of the patient’s total body surface area (TBSA). Intubation is recommended for those with facial edema, hoarseness, or stridor, and for patients who require significant volume resuscitation (>20% TBSA burn). In such cases, the endotracheal tube should be secured and monitored closely, as reintubation can be extremely difficult due to progressive inflammation and edema. Inhalation injuries often result in early bronchospasm, which responds well to albuterol. Prophylactic steroids and antibiotics are not recommended.

Air temperatures vary greatly during structural fires, from near normal at floor level to hundreds of degrees Fahrenheit at torso and head levels. Direct thermal burns (ie, direct heat injuries) are typically supraglottic, whereas steam and aerosolized chemicals often cause subglottic injuries. As might be expected, smaller particles are more commonly responsible for distal airway damage. The body’s inflammatory response and cytokine cascade can cause secondary injuries that necessitate increased fluid resuscitation in patients with cutaneous burns. Secondary injuries can also precipitate bronchospasm, vasospasm, bronchial exudates, and ventilation-perfusion mismatch.

Fire can rapidly consume available oxygen, a process that can precipitate anoxia and subsequent brain injuries. Similarly, carbon monoxide (CO) levels greater than 50% are associated with death. Levels between 30% and 50% frequently lead to myocardial ischemia and unconsciousness. Carboxyhemoglobin levels of 20% to 30% can cause muscle weakness and impaired cognition; levels between 10% and 20% commonly cause headache and nausea. Significant CO exposure can be treated with 100% oxygen for 6 hours. Hyperbaric oxygen therapy is controversial but can be considered.

Indoor fires that involve polymer combustion place patients at risk of cyanide poisoning, which can be confirmed by lactic acidosis that persists despite other indicators of a successful resuscitation (resolution of hypotension, urine production, resolution of tachycardia). These patients can be managed with hydroxocobalamin, with sodium nitrite and sodium thiosulfate as potential adjunct therapies.

Thermal airway injuries can be diagnosed by the presence of hoarseness, stridor, significant facial or neck burns, or increased work of breathing, all of which necessitate intubation. Lung-protective ventilation strategies, bronchoscopy, and the aggressive treatment of respiratory infections are beneficial.

KEY POINTS

- Inhalation injuries should be suspected in any patient who presents after a structure fire or with significant cutaneous burns.
- Burn patients with signs of airway compromise (eg, hoarseness, stridor, facial or neck burns, or increased work of breathing) should be intubated early.
- CO poisoning can lead to neurological deficits and death. These patients should be treated with 100% oxygen. Hyperbaric therapy can be considered.
- Cyanide poisoning can be confirmed by persistent lactic acidosis in patients with an otherwise successful resuscitation. Such cases should be treated with hydroxocobalamin.
Nausea and Vomiting in Pregnancy

By Kelly Mayo, MD; and Laura Welsh, MD, Boston University, Boston, Massachusetts
Reviewed by Andrew Eyre, MD, MHPEd


Most pregnant women experience nausea and vomiting at some point during their pregnancy. Symptoms, which generally start between 6 and 8 weeks of gestation and subside by 20 weeks, can affect day-to-day functioning and lead to dehydration, electrolyte imbalances, and even hospitalization.

As many as 3% of pregnant women suffer from hyperemesis gravidarum, a severe form of the disorder that is characterized by intractable vomiting. Treatment should be focused on providing symptomatic relief and preventing complications, such as renal impairment, Wernicke encephalopathy, and extreme weight loss.

First-line treatments, including simple lifestyle changes and over-the-counter remedies, are reserved for mild to moderate cases. Both ginger and pyridoxine can improve mild symptoms (level A, class IIa), and high doses of pyridoxine may be more effective than low doses. Acupressure can also help improve mild nausea and vomiting (level A, class IIa); however, the benefits of nerve stimulation and acupuncture are unclear.

Second-line treatments, including antiemetic medications, intravenous (IV) fluids, and electrolyte repletion can be used to manage moderate to severe cases. Combined treatment with doxylamine and pyridoxine can help alleviate moderate symptoms (level A, class IIa); however, there is limited evidence regarding the effectiveness of antihistamines alone (level B, class IIa). It is also unclear whether combined treatment with psychotherapy and vitamin B₆ is more effective than vitamin B₆ alone (level B, class IIa).

IV dopamine antagonists (eg, metoclopramide, promethazine) are also appropriate second-line treatments (level A, class IIa). In addition, serotonin receptor antagonists can effectively manage symptoms of any severity (level A, class IIa). IV ondansetron appears to be as effective as IV metoclopramide for managing pregnancy-related nausea and vomiting but with fewer adverse effects. There is limited evidence to suggest the superiority of dextrose saline over normal saline (level B, class IIa).

Finally, third-line treatments are reserved for patients with moderate to severe symptoms for whom other therapies have failed. Although the benefits of corticosteroids (eg, hydrocortisone) are unclear, these agents may be considered for patients with severe hyperemesis gravidarum (level A, class IIb). The benefits of other third-line treatments, such as nasogastric feeding, transdermal clonidine, and gabapentin, are unclear.

Editor’s Note: There is ongoing debate regarding the safety of ondansetron during pregnancy. The drug may increase the risk of congenital malformations; however, further research is needed. Prior to prescribing ondansetron, clinicians should counsel pregnant patients regarding its benefits and potential risks. For more information, consult the following references:
An estimated 18% of women and 9% of men in the United States suffer from migraine headaches, a complaint that leads to more than 1.2 million emergency department visits annually. Typical symptoms include unilateral throbbing headache pain with photophobia, phonophobia, nausea, or vomiting. The pain may be preceded by visual or sensory auras or accompanied by neurologic symptoms.

Diagnostic Testing
While new migraines are seldom diagnosed in the emergency department, it is helpful to understand the International Classification of Headache Disorders criteria. Migraine remains a clinical diagnosis; as such, routine laboratory studies and brain imaging are not indicated for patients with typical symptoms.

Treatment
Acute migraine symptoms can be managed with three classes of medications: antidopaminergics, triptans, and nonsteroidal anti-inflammatory drugs (NSAIDs). Monotherapy with antidopaminergic agents, such as metoclopramide, prochlorperazine, droperidol, or haloperidol, is recommended. Metoclopramide should be the first-line treatment for any pregnant patient with a migraine. The most common side effects of treatment are extrapyramidal symptoms (EPS). Dual therapy with diphenhydramine and prochlorperazine is recommended; however, diphenhydramine should not be combined with metoclopramide. Additionally, a slow rate of drug administration can decrease the risk of EPS. Symptoms that develop following treatment can be managed with diphenhydramine or midazolam.

Triptans, which are commonly used for the outpatient treatment of migraines, decrease nociceptive transmission within the trigeminal pathway. Subcutaneous sumatriptan is highly effective for the treatment of acute headaches (therapeutic result in ≈34 minutes), but the drug has numerous adverse effects and a 66% rate of headache recurrence within 24 hours. Overall, intravenous antidopaminergics are a more effective and well-tolerated option. Ketorolac, the most commonly used parenteral NSAID for acute migraine management, can be combined with antidopaminergic or triptan medications.

There is little evidence to support the use of other parenteral agents for the acute treatment of migraine. Although ketamine and propofol can resolve headache pain in the short term, the rate of recurrence is unknown. Nerve blocks are often used to treat migraine pain due to their low-risk profile; however, there is little evidence to support this approach.

Although more than 50% of migraine-related visits include the administration of an opioid, these drugs are less effective than other treatments and should not be used as a primary therapy. Parenteral dexamethasone can reduce headache recurrence within 72 hours of discharge. A specialist referral should be considered for patients with chronic migraines, psychiatric comorbidities, and headaches related to concomitant medication overuse.

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

We are military service members. This work was prepared as part of our official duties. Title 17 U.S.C. 105 provides that “Copyright protection under this title is not available for any work of the US Government.” Title 17 U.S.C. 101 defines a US Government work as a work prepared by a military service member or employee of the US Government as part of that person’s official duties.
The boarding of intubated, ICU-bound patients is an ever-growing challenge for emergency physicians, who are increasingly responsible for the initial management of these cases. This article summarizes two important ventilator strategies that can be readily incorporated into the care of these critically ill patients.

**LUNG PROTECTIVE STRATEGY**

The lung protective strategy focuses on low-tidal volume ventilation for the prevention of ventilator-induced lung injury and subsequent acute respiratory distress syndrome. This can be accomplished by considering that the volume of useful lung parenchyma is diminished in patients with respiratory failure. Although appropriate for any intubated patient, this strategy is specifically indicated for high-risk patients and those with signs of acute lung injury.

- **Mode** — Volume assist-control
- **Tidal volume** — Start with a tidal volume ($V_t$) of 8 mL/kg of predicted body weight (PBW) or below, as tolerated, and reduce by 1 mL/kg at intervals of 2 hours or less until $V_t = 6$ mL/kg PBW. $V_t$ can be adjusted to manage acidosis and CO$_2$ retention but should not be increased by more than 8 mL/kg.
- **Inspiratory flow rate** — An initial setting of 60 L per minute usually leads to adequate flow and can be titrated up, as needed, for comfort. Ignoring this parameter can lead to increased sedation/analgesia requirements.
- **Respiratory rate** — An initial rate of 15 to 16 breaths per minute can be used to achieve normocapnia in most patients. A blood gas measurement should be obtained 20 to 30 minutes later to titrate PaCO$_2$ to the patient’s acid-base status. Rates as high as 30 to 40 breaths per minute may be required. Permissive hypercapnia should be considered if PaCO$_2$ goals cannot be safely achieved with rapid respiratory rates.

- **PEEP** — Initial levels should be set to 5 cm H$_2$O to provide an “air stent.” This setting can prevent the collapse and shearing of small airways while helping the patient overcome the resistance of the ventilator tubing.
- **PEEP and FiO$_2$** — Set the lowest possible FiO$_2$ (usually 30%-40%) to maintain a goal oxygen saturation between 88% and 95% to prevent hyperoxia. The NIH-NHLBI ARDS Network recommends increasing FiO$_2$ and PEEP in tandem to facilitate alveolar recruitment.
- **Plateau pressure** — Assess every 30 to 60 minutes by pressing the inspiratory hold button at the end of a breath. A plateau pressure of 30 cm H$_2$O or more can cause alveolar injury. This risk can be avoided by decreasing $V_t$ by 1 mL/kg (minimum = 4 mL/kg PBW) until a value of less than 30 cm H$_2$O is achieved. Permissive hypercapnia may be required.
### Summary of the Two Ventilator Strategies

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<thead>
<tr>
<th></th>
<th>Lung Protective Strategy</th>
<th>Obstructive Strategy</th>
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</thead>
<tbody>
<tr>
<td><strong>Mode</strong></td>
<td>Volume assist-control</td>
<td>Volume assist-control</td>
</tr>
<tr>
<td><strong>Tidal volume</strong></td>
<td>Start at 8 mL/kg PBW; adjust for plateau pressure goal.</td>
<td>8 mL/kg PBW</td>
</tr>
<tr>
<td><strong>Inspiratory flow rate</strong></td>
<td>Start at 60 L/min; adjust for comfort.</td>
<td>60-80 L/min</td>
</tr>
<tr>
<td><strong>Respiratory rate</strong></td>
<td>Start at 16 breaths/min; adjust for PaCO₂ goal.</td>
<td>Start at 10 breaths/min; adjust to allow full expiration.</td>
</tr>
<tr>
<td><strong>PEEP</strong></td>
<td>Start at 5 cm H₂O; adjust according to the table.</td>
<td>0 cm H₂O (Some may treat the patient with PEEP 5 cm H₂O.)</td>
</tr>
<tr>
<td><strong>FiO₂</strong></td>
<td>Start at 40%; adjust according to the table.</td>
<td>Start at 40%; adjust for SpO₂ 88%.</td>
</tr>
<tr>
<td><strong>Check for safety</strong></td>
<td>Measure plateau pressure. If 30 cm H₂O, decrease the tidal volume by 1 mL/kg.</td>
<td>Measure the plateau pressure or observe the flow/time graph. If the plateau pressure reaches 30 cm H₂O or the flow/time graph shows incomplete expiration, decrease the respiratory rate.</td>
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### FiO₂ and PEEP Scale from ARDSnet ARMA Trial

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### Obstructive Strategy

The obstructive strategy is used for patients with reactive airways, typically from asthma or COPD. Because these patients experience air trapping and barotrauma when exposed to the rapid respiratory rates and lower tidal volumes of the lung protective strategy, a different approach is required. In such cases, the primary goal is to allow the patient time to exhale, primarily by reducing the respiratory rate and allowing for permissive hypercapnia.

**Mode** — Volume assist-control  
**Tidal volume** — Set the initial levels to 8 mL/kg PBW.

### Key Points
- Volume assist-control mode ventilation allows for patient-initiated breaths and greater control of pertinent parameters.
- Ventilator mode, tidal volume, inspiratory flow rate, respiratory rate, PEEP, FiO₂, and plateau pressure are key ventilator parameters for managing mechanically ventilated patients.
- A lung protective strategy of ventilation, which focuses on low-tidal volume ventilation (≤8 mL/kg PBW), should be used when managing any intubated patient.
- An obstructive strategy of ventilation focuses on reducing the respiratory rate to allow patients with obstructive lung diseases time to exhale, thereby reducing the danger of air trapping.
- Plateau pressure is measured by pressing the inspiratory hold button at the end of a breath (inspiratory pause). It should be maintained at levels below 30 cm H₂O to reduce the risk of alveolar injury.

**Inspiratory flow rate** — Set to 60 to 80 L per minute. Shorter inhalation times theoretically can be used to allow for even longer exhalation. However, this approach often results in higher peak pressures with limited added benefit.  
**Respiratory rate** — Set to 8 to 10 breaths per minute to allow time for exhalation. Permissive hypercapnia can help limit the effects of air trapping and barotrauma.  
**PEEP** — Set the PEEP between 0 and 5 cm H₂O.  
**FiO₂** — Set the lowest possible FiO₂ (usually 30%-40%) to maintain a goal oxygen saturation between 88% and 95%.  
**Plateau pressure** — Measure the patient’s plateau pressure or observe the flow/time graph. Decrease the respiratory rate if the plateau pressure is greater than 30 cm H₂O, or the flow/time graph shows incomplete expiration (ie, expiratory flow does not return to baseline before the subsequent inspiration is initiated).
Mesenteric ischemia is a condition that is prototypical of emergency medicine: a rare, acute, life-threatening process that presents as abdominal pain, a common chief complaint. Astute recognition, speedy diagnosis, and time-sensitive interventions play a critical role in patient outcomes.

Mesenteric ischemia is a vascular emergency caused by impaired blood flow to one or more of the primary vessels that comprise the mesenteric circulation: the celiac artery, superior mesenteric artery, and inferior mesenteric artery (or their accompanying veins). In cases of arterial or venous occlusion, blood flow is insufficient to meet the metabolic demands of visceral organs, a process that can lead to visceral injuries and myocardial infarction (MI).

Disease Subtypes
The pathophysiological process, which can be acute or chronic, is divided into three main subtypes: arterial occlusion, venous thrombosis, and nonocclusive (in which vasospasm impairs regional perfusion, leading to ischemia). In contrast to other vascular disorders, mesenteric ischemia is more common in women.

Arterial occlusion is the most common cause of mesenteric ischemia, and any acute case is a vascular catastrophe that constitutes a surgical emergency. Etiologies include embolic occlusion, thrombotic occlusion of a previously stenotic vessel, dissection, and arterial inflammation. Arterial occlusion should be suspected in patients who present with pain that is out of proportion to the examination and in those with acute, severe abdominal pain (with or without tenderness) who have risk factors for atherosclerosis (eg, peripheral vascular disease), arterial embolism (eg, atrial fibrillation, recent MI), arterial dissection, or arterial inflammation (eg, vasculitides).

Venous thrombosis impairs the venous outflow and leads to visceral edema. These patients tend to present with a more subacute onset than those with acute arterial occlusion. Risk factors for venous thrombosis include inherited or acquired thrombophilia, trauma, local inflammation from adjacent surrounding visera (eg, pancreatitis, diverticulitis), and idiopathic thrombosis.

Nonocclusive mesenteric ischemia results from various low-flow/shock states (eg, sepsis, hemorrhage, MI with shock, congestive heart failure, recent cardiac surgery, hemodialysis) with patent mesenteric vessels. In such cases, vasospasm shunts blood to more vital organs, a process that can lead to abdominal visceral malperfusion.

KEY POINTS
- Acute mesenteric ischemia is a surgical emergency in which early recognition, rapid diagnosis, and emergent treatment are paramount to survival.
- Unlike other vascular disorders, mesenteric ischemia is more common in women.
- A history of atherosclerotic and vascular diseases (eg, coronary artery disease, peripheral vascular disease, cerebrovascular disease) and/or atrial fibrillation should heighten suspicion for mesenteric ischemia in patients with severe abdominal pain.
- Laboratory tests are nonspecific for mesenteric ischemia. Abdominal CTA is the diagnostic test of choice in the emergent setting.
- Initial management includes hemodynamic monitoring and support, fluid resuscitation, correction of electrolyte abnormalities, pain control, anticoagulation, and the initiation of broad-spectrum antibiotics.
- Surgical repair (endovascular or open) is the only definitive therapy.
Chronic mesenteric ischemia follows a more indolent course, owing to support from collateral vascular networks that maintain visceral perfusion. This pathology should be suspected in patients with chronic abdominal pain that is characteristically postprandial and associated with early satiety, weight loss, and altered bowel patterns, particularly when an extensive gastroenterological workup is negative for an alternative etiology.

**Testing and Treatment**

No laboratory markers are sensitive or specific enough to diagnose or rule out mesenteric ischemia. The most useful studies are those that evaluate for alternative diagnoses, measure serum electrolyte levels, determine acid-base status, and assess for evidence of infection. A normal serum lactate level cannot rule out mesenteric ischemia, as elevated lactate is a late finding that often indicates segmental, severe ischemia and/or an irreversible bowel injury. In the emergent setting, computed tomography angiography (CTA) of the abdomen is the imaging modality of choice, given its speed and accuracy (95%-100%). When CTA is contraindicated, magnetic resonance angiography can be considered. While sensitive and specific, the accuracy of ultrasound is operator dependent. In addition, the test takes longer to complete, can be difficult to perform in obese patients with large amounts of bowel gas and/or calcified vessels, and may be painful.

The emergent treatment of acute arterial mesenteric ischemia starts with immediate intravenous fluid resuscitation and the correction of electrolyte and acid-base abnormalities. Hemodynamic monitoring should drive the fluid requirements, which can be as high as 10 to 20 L of crystalloid on the first day. Vasopressors should be avoided if at all possible. It is critical to initiate early anticoagulation with heparin and administer broad-spectrum antibiotics that cover bowel flora.

An emergent consultation with a vascular or acute care surgeon is necessary, as endovascular or open repair is the definitive management strategy for revascularization. Venous mesenteric ischemia can often be treated with anticoagulation alone. However, an early surgical consultation is warranted in such cases, as transhepatic and percutaneous mechanical thrombectomy, thrombolysis, and open intra-arterial thrombolysis may be required if the patient is refractory to treatment.

When managing nonocclusive mesenteric ischemia, it is imperative to address the underlying cause, provide hemodynamic support, and limit the use of systemic vasoconstrictors. Emergent open surgical exploration is indicated for all forms of the disease in patients with signs of overt peritonitis.
This summary outlines a three-step process for the evaluation of a possible BRUE.

1. Is this a BRUE?
   - Is the patient younger than 1 year?
   - Is the patient well appearing?
   - Did the child present following a sudden, brief, resolved event that included one or more of the following: cyanosis or pallor; absent, decreased, or irregular breathing; a marked change in tone (hyper- or hypotonia); or altered responsiveness?
   - Is the event unexplained (eg, no history or symptoms of gastro-esophageal reflux disease [GERD], feeding difficulties, or airway abnormalities)?

If the answer to any question is NO, BRUE should be ruled out, and the patient should undergo further evaluation. If the answer to all four questions is YES, BRUE should be diagnosed, and risk stratification should proceed.

2. Is the patient low risk?
   - Is the child older than 60 days?
   - Was the child born at or after 32 weeks of gestation, and is the corrected gestational age 45 weeks or more?
   - Did the event last less than 1 minute?
   - Is this the first such event?
   - Are the patient’s history and examination normal (eg, no family history of sudden cardiac death; no nondiagnostic social, feeding, or respiratory problems)?

If the answer to any question is NO and the child required CPR by a trained medical provider, the case should be considered HIGH RISK. Although these patients require further evaluation, there is no evidence to guide their management. If the answer to the above questions is YES and no CPR was required, the patient should be considered LOW RISK; treatment should proceed according to the following guidelines.

3. When managing low-risk BRUE, clinicians:
   - SHOULD educate caregivers, provide resources for CPR training, and use a shared decision-making approach regarding the child’s evaluation, disposition, and follow-up care.
   - MAY offer or consider pertussis testing, obtain an ECG, and provide brief monitoring (eg, pulse oximetry, serial examinations).
   - SHOULD NOT reflexively initiate laboratory testing, diagnostic imaging, GERD studies, electroencephalography, or home cardiac or respiratory monitoring.

In addition, there is no need to prescribe GERD or seizure medications or admit a low-risk patient solely for cardiovascular or respiratory monitoring.

KEY POINTS
- Children who meet the criteria for BRUE without high-risk features can be safely discharged without undergoing invasive testing, diagnostic imaging, or inpatient admission.
- There are no formal guidelines regarding the evaluation or disposition of high-risk BRUE cases.
- The patient’s parents should be involved in clinical decision making.
Children with nontraumatic hip pathologies frequently present with pain in the hip, thigh, or knee; an altered gait; or refusal to bear weight. In patients with a hip-joint pathology (eg, effusion, hemarthrosis, or fracture), the hip rests in flexion, abduction, and external rotation. An ultrasound of the hip can identify an effusion but cannot distinguish between sterile and septic joint effusions. When ordering hip radiographs, it is important to obtain both comparison and pelvic views (specifically, anteroposterior [AP] and frog-leg views). MRI is sensitive and specific; however, timing, costs, sedation considerations, and availability can limit the modality’s applicability.

Pediatric nontraumatic hip complaints can be caused by a spectrum of disorders that range from benign and self-resolving to life-threatening. Any child who presents with hip or knee pain accompanied by an altered gait or refusal to bear weight should be evaluated for these diagnoses (see table next page). Proper diagnostic testing and disposition decisions can be guided by a careful physical examination, a complete medical history, and focused imaging and laboratory evaluations.
| Disorder | Description | Epidemiology | History/Examination | Diagnosis | Clinical Course | Management |
|----------|-------------|--------------|---------------------|-----------|----------------|
| TRANSIENT SYNOVITIS | Self-limited inflammation and effusion of hip joint of unknown etiology | Most common cause of nontraumatic hip pain; Mean age 4.7 yrs, with range of 3–8 yrs; Male predominance; Usually unilateral; History of preceding illness | Acute-onset pain with limp or unwillingness to bear weight; Usually well appearing | Clinical diagnosis is based on history and examination; Improvement with NSAIDs is reassuring. | Disease is self-limited and typically resolves in 3–10 days. | Arrange follow-up with a primary care physician. |
|         |             |              |                     |           |                | Prescribe scheduled NSAIDs. |
|         |             |              |                     |           |                | Counsel parents on anticipatory guidance and reasons to return. |
| LEGG-CALFÉ-PERTHES DISEASE | Idiopathic avascular necrosis of the capital femoral epiphysis | Children aged 2–12 yrs; 4 times more common in boys; Bilateral in 10%–15% of cases; Incidence: 0.2–19.2 per 100,000; Obesity and hypercoagulability (common predisposing factors) | Subacute symptoms with pain or painful limp; Limp often noticed incidentally to minor trauma; Limited hip abduction and internal rotation | Radiographs can confirm the diagnosis; however, x-rays may be normal early in the disease course. | Disease is self-limited to 1–2 yrs. | An orthopedic referral for ongoing outpatient management is required. |
| SLIPPED CAPITAL FEMORAL EPIPHYSIS | Displacement of the femoral head from the femoral neck through the epiphyseal plate | Estimated incidence: 10 per 100,000 children; More common in boys; Mean age 12 yrs old; <24% with bilateral disease | Subacute presentation; History of recent, minor trauma that does not explain symptoms; Knee pain (15%–50%); Altered gait (common) | On AP films, the Klein line — from the superior aspect of the femoral neck — should intersect the epiphysis of the femoral head (low sensitivity). | Prognosis is excellent if the intervention is made before severe displacement or osteonecrosis occurs. | Request an immediate orthopedic consultation. |
|         |             |              |                     |           |                | Treatment involves stopping the epiphyseal cartilage and preventing progression. |
|         |             |              |                     |           |                | Maintain non-weight-bearing status. |
| OSTEITARICULAR INFECTIONS SEPTIC ARTHRITIS (AND OSTEOMYELITIS) | Inflammation of joint space from infection, osteomyelitis may be an isolated problem or coexist with septic arthritis | Most commonly spread hematogenously from distant source; In children <2 yrs, close proximity of blood vessels to the proximal femoral physis allows infection to spread into the epiphyseal and contiguous hip joint; cited as a reason septic arthritis is found most commonly in youngest populations | Classically high fever, toxic appearance; Hot, painful, swollen joint of short duration (<1 wk); Severe or complete resistance to passive range of motion; Inability to bear weight or use the affected joint (common) | Gold-standard treatment requires isolating the pathogen from the site of infection or isolating the pathogen from blood; Obtain blood and joint cultures, CBC, and inflammatory markers. | Obtain cultures prior to giving antibiotics. Consult with orthopedics if perioperative antibiotics are considered. | An emergent orthopedic consultation and surgical intervention are necessary. |
|         | Monoarticular hip arthropathy | Rare cause of acute, nontraumatic hip pain, however, testing may be warranted if in an endemic region and a septic hip is suspected; Most common in children aged 5–15 yrs | Mean incubation is 3.4 mom (2 wks–2 yrs). | Synovial studies cannot differentiate Lyme from septic arthritis. | Varies by stage of Lyme disease | Provide empiric broad-spectrum antibiotics to cover staphylococci and patient-specific risks. |
| LYME ARTHRITIS | Osteosarcoma and Ewing sarcoma: most common primary bone cancers | Peak incidence in adolescence | Tumor-related pain, with or without a mass, painless mass, or pathologic fracture; Most common symptoms: pain (with or without activity), palpable mass; Constitutional symptoms uncommon | Laboratory studies are often normal. | Varies by severity | Provide an immediate oncology referral. |
|         | Leukemia: can cause leg pain and a limp due to bone-marrow expansion | | | Plain radiography (the first-line diagnostic test) will reveal both malignant and benign patterns. | | |
Thrombolytics for Acute Pulmonary Embolism

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Up to 25% of acute pulmonary embolisms (PEs) result in sudden death, and between 17% and 50% of these patients die within 3 months of the initial event. However, overall mortality is dependent on a variety of factors, including the patient’s age, stability, and initial symptoms as well as the presence of comorbidities.

Massive PEs often present as pulselessness, persistent bradycardia, signs of shock, or sustained hypotension without a secondary cause. Submassive PEs are defined by a near-normal blood pressure with evidence of cardiopulmonary stress, which may include right ventricular (RV) dysfunction, a new elevation in brain natriuretic peptide (BNP) or pro-BNP, a newly elevated troponin level, a right bundle branch block, and signs of right heart strain. Submassive events account for approximately 20% of all PEs, with an in-hospital mortality rate as high as 5%. Morbidity can be severe, with an increased risk of pulmonary hypertension, impaired quality of life, persistent RV dysfunction, and recurrent thrombus formation.

Although treatment is required to reduce the risk of potentially fatal complications, the benefits of thrombolytic therapy must be weighed against its risks. Thrombolysis is recommended for the management of massive PEs, and there is widespread support for the delivery of systemic thrombolytics to patients undergoing CPR with echocardiographic evidence of a massive PE with no other identified cause.

Research suggests that thrombolytic therapy may reduce long-term pulmonary hypertension in patients with submassive PEs; however, the treatment is only endorsed for patients at low risk of bleeding. Thrombolytics may provide little benefit to those with a pulmonary reserve and no prior lung disease; however, the treatment may be more effective for those with coexisting conditions (eg, heart failure or obstructive lung disease). Signs of clinical decompensation, including hypoxia, worsening tachypnea or tachycardia, and even brief episodes of hypotension, should prompt consideration for thrombolysis.

Overall, research findings have been heterogeneous; some large studies have demonstrated the benefits of thrombolytics, while others have not. As such, a definitive recommendation for the treatment of submassive PEs has remained elusive. The use of catheter-directed or half-dose systemic thrombolytics or a surgical embolectomy may improve long-term outcomes while mitigating the risk of bleeding. Catheter-directed therapy and a surgical embolectomy can reduce RV dilatation and pulmonary hypertension, decrease the clot burden, and minimize the risk of intracranial hemorrhage. These alternative treatments can be used for patients with an increased risk of bleeding (ie, patients >65 years) and those who fail to improve with initial thrombolytic dosing.

Ultimately, treating physicians must use their own judgment regarding the relative merits of each therapy with input from specialists and the patient.

KEY POINTS
- Thrombolytic therapy is warranted for massive PEs accompanied by hemodynamic collapse or cardiac arrest.
- Submassive PEs present a clinical quandary due to inconsistent research findings, various outcome measures, and different treatment protocols.
- Thrombolytic therapy appears to improve long-term functional outcomes in patients with submassive PEs, but the risk of bleeding significantly increases in those older than 65 years.
- Half-dose thrombolytic therapy, a surgical embolectomy, and catheter-directed treatments may provide significant benefits and reduce the risk of bleeding.
- Further studies are needed to assess risk stratification, functional outcomes, and treatment protocols with thrombolytic dosing.
MRI Safety in Patients With Cardiac Devices

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Many patients who undergo placement of a permanent pacemaker (PPM) or implantable cardioverter-defibrillator (ICD) subsequently develop an indication for MRI. Unfortunately, some are denied the opportunity to undergo the test due to safety concerns. Despite this common trepidation, MRI poses no additional risks to patients with contemporary PPM and ICD systems, most of which are labeled by the FDA as “MRI conditional.”

Furthermore, the Centers for Medicare & Medicaid Services has determined that access to MRI improves outcomes in patients with modern PPMs and ICDs. Devices that do not carry the MRI-conditional designation are termed “legacy” systems.

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

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

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

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

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

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


KEY POINTS

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

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

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

The ABCD² score is the most researched and widely used risk-stratification tool for patients with TIAs; however, this approach may not be generalizable to all institutions. Unfortunately, existing clinical decision guidelines, including the ABCD² score, do not appear to reliably predict a patient’s risk of an early, recurrent stroke.

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

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

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

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

Of note, observation unit protocols yield shorter lengths of stay and lower costs. More importantly, the rate of stroke in patients who receive observation care is similar in those who are risk stratified using the ABCD² score.

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