Seizures and epilepsy are common, serious neurologic diseases in children and adolescents. Convulsions can be a manifestation of epilepsy or occur secondary to a complication of a systemic or central nervous system disorder. The emergency physician is usually the first provider to evaluate and stabilize children with suspected seizures. Here, we will review recent literature and share our personal experience in the approach to a seizing child in the emergency department.

Epilepsy is defined as the predisposition to generate seizures. While generalized or focal shaking in a child readily raises a concern for seizure in caregivers and doctors alike, subtle manifestations, such as brief episodes of lip smacking in temporal lobe epilepsy or head bobbing in infantile spasms, can be challenging to correctly detect as signs of a serious neurologic disorder.

As with any other patient presenting to the emergency department, assessment of seizing children starts with determining stability and urgently addressing the ABCs.

Defining sepsis has been an ongoing challenge for the physicians who treat this critical condition.

Before 2000, solid epidemiology data on sepsis were lacking. No formalized, generally accepted definition existed, even for septicemia, which was more commonly used. Additionally, prior to 2002, diagnostic codes for sepsis, severe sepsis, and septic shock were nonexistent. Martin et al estimated that there were more than 10 million septic patients during a 22-year span, increasing 9 percent annually from 164,000 cases to 660,000. Angus et al estimated 750,000 cases, representing more than suspected seizures. Here, we will review recent literature and share our personal experience in the approach to a seizing child in the emergency department.

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Think ELIQUIS—For your appropriate patients with NVAF or DVT/PE

**IMPORTANT SAFETY INFORMATION**

**WARNING: (A) PREMATURE DISCONTINUATION OF ELIQUIS INCREASES THE RISK OF THROMBOTIC EVENTS, (B) SPINAL/EPIDURAL HEMATOMA**

(A) Premature discontinuation of any oral anticoagulant, including ELIQUIS, increases the risk of thrombotic events. If anticoagulation with ELIQUIS is discontinued for a reason other than pathological bleeding or completion of a course of therapy, consider coverage with another anticoagulant.

(B) Epidural or spinal hematomas may occur in patients treated with ELIQUIS who are receiving neuraxial anesthesia or undergoing spinal puncture. These hematomas may result in long-term or permanent paralysis. Consider these risks when scheduling patients for spinal procedures. Factors that can increase the risk of developing epidural or spinal hematomas in these patients include:

- use of indwelling epidural catheters
- concomitant use of other drugs that affect hemostasis, such as nonsteroidal anti-inflammatory drugs (NSAIDs), platelet inhibitors, other anticoagulants
- a history of traumatic or repeated epidural or spinal punctures
- a history of spinal deformity or spinal surgery
- optimal timing between the administration of ELIQUIS and neuraxial procedures is not known

Monitor patients frequently for signs and symptoms of neurological impairment. If neurological compromise is noted, urgent treatment is necessary.

Consider the benefits and risks before neuraxial intervention in patients anticoagulated or to be anticoagulated.

**CONTRAINDICATIONS**

- Active pathological bleeding
- Severe hypersensitivity reaction to ELIQUIS (e.g., anaphylactic reactions)

**IMPORTANT SAFETY INFORMATION**

**WARNINGS AND PRECAUTIONS**

- Increased Risk of Thrombotic Events after Premature Discontinuation: Premature discontinuation of any oral anticoagulant, including ELIQUIS, in the absence of adequate alternative anticoagulation increases the risk of thrombotic events. An increased rate of stroke was observed during the transition from ELIQUIS to warfarin in clinical trials in atrial fibrillation patients. If ELIQUIS is discontinued for a reason other than pathological bleeding or completion of a course of therapy, consider coverage with another anticoagulant.

- **Bleeding Risk:** ELIQUIS increases the risk of bleeding and can cause serious, potentially fatal, bleeding.
  - Concomitant use of drugs affecting hemostasis increases the risk of bleeding, including aspirin and other antiplatelet agents, other anticoagulants, heparin, thrombolytic agents, SSRIs, SNRIs, and NSAIDs.
  - Advise patients of signs and symptoms of blood loss and to report them immediately or go to an emergency room. Discontinue ELIQUIS in patients with active pathological hemorrhage.
  - The anticoagulant effect of apixaban can be expected to persist for at least 24 hours after the last dose (i.e., about two half-lives). An agent to reverse the anti-factor Xa activity of apixaban is available. Please visit www.andexxa.com for more information on availability of a reversal agent.

- **Spinal/Epidural Anesthesia or Puncture:** Patients treated with ELIQUIS undergoing spinal epidural anesthesia or puncture may develop an epidural or spinal hematoma which can result in long-term or permanent paralysis.

The risk of these events may be increased by the postoperative use of indwelling epidural catheters or the concomitant use of medicinal products affecting hemostasis. Indwelling epidural or intrathecal catheters should not be removed earlier than 24 hours after the last administration of ELIQUIS. The next dose of ELIQUIS should not be administered earlier than 5 hours after the removal of the catheter. The risk may also be increased by traumatic or repeated epidural or spinal puncture. If traumatic
**WARNING AND PRECAUTIONS (cont’d)**

- **Profound Hypotension:** Hypotension occurs frequently and may require treatment. Monitor patients frequently and be prepared to treat.

- **Protracted Hemorrhage:** ELIQUIS may cause prolonged bleeding. Monitor patients for signs of bleeding and be prepared to treat.

- **Hypersensitivity Reactions:** Severe hypersensitivity reactions to ELIQUIS have occurred. Stop ELIQUIS in patients with these reactions.

- **Other Anticoagulants:** ELIQUIS has no clinically significant effect on the anticoagulant effect of other anticoagulants, including vitamin K antagonists.

- **Combined P-gp and Strong CYP3A4 Inhibitors:** Use caution when coadministered with these inhibitors.

**ADVERSE REACTIONS**

- The most common adverse reactions reported with ELIQUIS are headache, nausea, and bleeding.

**TEMPORARY INTERRUPTION FOR SURGERY AND OTHER INTERVENTIONS**

- ELIQUIS should be discontinued at least 48 hours prior to elective surgery or invasive procedures to reduce the risk of bleeding.

**DRUG INTERACTIONS (cont’d)**

- **Combined P-gp and Strong CYP3A4 Inhibitors:** These inhibitors may increase exposure to apixaban and increase the risk of bleeding.

**PREGNANCY CATEGORY B**

- There are no adequate and well-controlled studies of ELIQUIS in pregnant women. Use ELIQUIS during pregnancy only if the potential benefit outweighs any potential risk to the mother and fetus.

**REFERENCES**


Please see Brief Summary of Full Prescribing Information, including **Boxed WARNINGS**, on adjacent pages.

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ELIQUIS® (apixaban) tablets, for oral use

Brief Summary of Prescribing Information. For complete prescribing information consult ELIQUIS® (apixaban) is indicated to reduce the risk of stroke and systemic embolism in patients with nonvalvular atrial fibrillation. For the treatment of DVT. For the prevention of stroke in patients with atrial fibrillation. For the treatment of acute PE in patients with hemodynamic instability or who require thrombolysis or mechanical thrombectomy. For the prevention of thromboembolism in patients undergoing elective hip or knee replacement surgery. For the treatment of an acute coronary syndrome in patients with NSTE-ACS. For patients with PFO and a contraindication to warfarin. For the prevention of stroke in patients with TM. For patients with PE who present with hemodynamic instability or who may receive thrombolysis or pulmonary embolectomy. For the prevention of thromboembolism in patients undergoing hip or knee replacement surgery. For the prevention of thromboembolism in patients undergoing PCI. For the prevention of thromboembolism in patients with a history of stroke or TIA and who are scheduled to undergo surgery or who have a contraindication to warfarin or other anticoagulant therapy. For the prevention of stroke in patients with AF and prior stroke or TIA. For the prevention of stroke in patients with AF and an increased CHADS2 score. For the prevention of stroke in patients with AF and a CHA2DS2-VASc score of 2 or more. For the prevention of stroke in patients with AF and at least one additional risk factor. For the prevention of stroke in patients with AF and at least one additional risk factor. For the prevention of stroke in patients with AF and at least one additional risk factor. For the prevention of stroke in patients with AF and at least one additional risk factor. For the prevention of stroke in patients with AF and at least one additional risk factor. For the prevention of stroke in patients with AF and at least one additional risk factor.
### Table 4: Adverse Reactions Occurring in ≥1% of Patients in Either Group Undergoing Hip or Knee Replacement Surgery

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Major or CRNM (%)</th>
<th>CRNM (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ELOQUIS (apixaban)</td>
<td>115 (4.3)</td>
<td>261 (9.7)</td>
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<tr>
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<td>187 (6.4)</td>
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### Table 5: Blinding Results from the AMPLIFY-EXT Study

| Treatment           | N=826 | n (%)
<table>
<thead>
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<tbody>
<tr>
<td>ELOQUIS (apixaban)</td>
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### Table 7: Bleeding Events in the AMPLIFY-EXT Study

#### AMPLIFY Study

| Treatment           | N=2676 | n (%)
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#### Treatment of DVT and PE in the AMPLIFY-EXT Study

| Treatment           | N=840  | n (%)
<table>
<thead>
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### Table 8: Adverse Reactions Occurring in ≥1% of Patients Undergoing Extended Treatment for DVT and PE in the AMPLIFY Study

| Treatment           | N=811  | n (%)
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### Table 9: Other Adverse Reactions

| Treatment           | N=840  | n (%)
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### Table 10: Drug Interactions

| Drug Interaction | n (%)
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### Table 11: Safety and Efficacy in Pediatric Patients

| Treatment           | N=840  | n (%)
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### Table 12: Other Adverse Reactions

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### Table 13: Drug Interactions

| Drug Interaction | n (%)
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### Table 14: Safety and Efficacy in Pediatric Patients

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### Table 15: Other Adverse Reactions

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### Table 16: Drug Interactions

| Drug Interaction | n (%)
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### Table 17: Safety and Efficacy in Pediatric Patients

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Congress Passes Comprehensive Opioid Package

On Oct. 24, President Donald Trump signed a sweeping legislative package of bills to address the nation’s growing opioid epidemic. Two ED-specific provisions developed by ACEP are included that would authorize grants to expand the alternatives to opioids (ALTO) program and the ED-initiated medication-assisted treatment (MAT) program that develops best practices for providing a “warm handoff” of patients with opioid use disorder to appropriate community resources and providers to keep them engaged in addiction treatment. Throughout the many months of congressional activity on H.R. 6, ACEP worked closely with congressional leadership, House-Senate conference, and the bill sponsors to ensure these provisions were included in the final package and used the ACEP-preferred language. During the ACEP Leadership & Advocacy Conference last May, hundreds of ACEP members advocated for these provisions with legislators and staff during their Capitol Hill visits.

More ACEP members in the 911 Network responding to action alerts over the past months responded to action alerts over the past months and contacted their legislators about these bills.

In other opioid-related news, ACEP participated in a meeting with White House officials, federal agencies, and other stakeholders to discuss best practices in combating the opioid epidemic. The meeting focused on actions the Trump administration has taken to date, a review of future projects, and policies established at state and local levels that have benefited patients and reduced misuse of opioids. A particular focus was placed on National Take Back Day on Oct. 27 and how meeting participants can promote these drug collection sites.

ACEP Releases New Information Papers

The following information papers were recently approved by the Board of Directors and published on the ACEP website:

- Advocating for a Minimum Benefit Standard Linked to the 80th Percentile of a FAIR Health-Type Usual & Customary Charge Database
- Emergency Ultrasound Standard Reporting Guidelines
- Medicaid ED Copayments: Effects on Access to Emergency Care and the Practice of Medicine
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- Emergency Physician Appointment to PTAC

ACEP submitted a robust set of comments responding to the calendar year 2019 Medicare Part B physician fee schedule (PFS) and Quality Payment Program (QPP) proposed rule. This rule included numerous policies that impact physician payments under Medicare, most notably a proposal that would streamline documentation requirements and create a blended payment rate for office/outpatient evaluation and management (E/M) level 2 through 5 codes. However, the proposal does not initially impact the emergency medicine/E/M code set. The rule also proposes a set of policies related to the third year of the QPP, the performance program established by the Medicare Access and CHIP Reauthorization Act.

news from the college

Updates and Alerts from ACEP
2019 Course Topics
- The ED’s Role in Treating Opioid Addiction
- Coronary Risk Scores – How Good Are They?
- Sepsis – What’s All the Confusion About? - Part 1
- Sepsis – What’s All the Confusion About? - Part 2
- What Radiologists Say We Should/Shouldn’t Order
- 2018 Stroke Guideline Controversies
- Pearls from Risk Management Monthly
- Pearls from ED Leadership Monthly
- Concussion: ED Essentials
- Serious Diseases, Outpatient Treatment - Part 1
- Serious Diseases, Outpatient Treatment - Part 2
- Pediatric Torso Trauma Assessment
- Pregnancy-Related ED Pearls
- The 2018 ATLS Guidelines – What’s New?
- Pediatric Emergency Medicine Pearls - Part 1
- Pediatric Emergency Medicine Pearls - Part 2
- Clinical Consequences of ED Crowding
- Telemedicine in Emergency Care
- Latest Evidence Concerning Resuscitation Fluids
- Learning to Love High Sensitivity Troponins
- Infant Fever: Entering the Era of the Algorithm
- Toddler Fever: The Nucleotides Take Over
- Cannabinoid-Related Emergencies
- Geriatric ED Accreditation – What’s it Mean?
- What’s New in ED Respiratory Support?
- Pediatric Blunt Head Injury Assessment
- Visual Diagnosis Challenges - Part 1
- Visual Diagnosis Challenges - Part 2
- Important Recent EM Literature - Part 1*
- Important Recent EM Literature - Part 2*
- Optimizing ED Operations*
- Diagnostic and Therapeutic Controversies*

*Topics listed with an asterisk (*) are 90-minute faculty panel discussions; all other topics are 30 minutes.

Experience the Course Enjoyed by Over 48,000 of Your Colleagues!

Learn More Online, Visit www.EMACourse.com or Call 1-800-458-4779 (9:00am-4:30pm ET, M-F)
Reuben Strayer, posing with Surgeon General Dr. Jerome Adams, was ACEP’s representative during a meeting about providing buprenophine as MAT.

More Considerations When Using Buprenorphine


As an emergency physician with a special interest in the identification and treatment of opioid use disorder among our patients, I was overjoyed to learn that ACEP Now was publishing a series of articles centered around buprenorphine and its potential as a treatment for emergency patients.

I found that this article in particular was concise yet accurate in its description of buprenorphine’s pharmacology and avoided many of the common misunderstandings surrounding the drug (especially the idea that the naloxone included in Suboxone provides the opioid antagonist property).

However, I believe that it’s critical to correct two misunderstandings that could result in either serious legal trouble or patient harm.

First is the implication of the statement that “...we have a medication we can give 8 mg of sublingually or 0.3 mg of subcutaneously or via IV...” which suggests that the formulation of buprenorphine administered is inconsequential. In the context of treating opioid use disorder and acute opioid withdrawal, this is incorrect, and it is illegal for a physician to prescribe or administer an opioid drug for these purposes unless that drug carries an FDA approval for the treatment of opioid use disorder.

Although there exists an increasing number of buprenorphine formulations on the market, we are limited to those that are FDA approved specifically for the treatment of addiction, such as Suboxone, Subutex, Zubsolv, Sublocade (implant), or the generics thereof when treating opioid withdrawal and depend¬ency. I would instead federal law to admini¬ster a buprenorphine formulation that does not carry this FDA approval such as Buprenex (IM/IV) or Butrans (transdermal); even Belbu¬ca, which is a sublingual tablet nearly identi¬cal to Subutex, is disallowed for use.

The difference between these two groups is that the latter medications are only approved for the treatment of pain, while the former car¬ry an indication for the treatment of opioid use disorder. Unfortunately, this seeming trivial¬ity could mean risking a felony conviction for violating the Controlled Substances Act. Insti¬tutions and providers have been investigated and fined in the past for their use of injected buprenorphine to treat their patients in opioid withdrawal.

The second troublesome statement is the suggestion that, “I could give them buprenor¬phine...and instead of causing precipitat¬ed withdrawal, it would cause precipitated breathing.” Although the author is trying to illustrate pharmacology rather than give us clinical advice, this implied clinical scenario is implausible. As described previously in the same article, buprenorphine has an extreme¬ly high affinity for the mu-receptor, higher than heroin and even higher than naloxone. You can titrate the dosage of naloxone so that it binds just enough receptors to reverse the overdose but still leaves some receptors avail¬able for agonists; this way, you create a happy median between apnea and agony.

In contrast, buprenorphine binds mu-receptors so avidly that it will come to occupy nearly all in the brain, leaving no open recep¬tors for an agonist to bind. This creates leg¬endarily severe precipitated withdrawal. There is no equilibrium between agonism and an¬tagonism. As an analogy, it is the difference between walking down a staircase and jump¬ing out the window; both reduce your alti¬tude. However, with the first you can gradually move up or down, but once you de¬fendratate yourself, there’s no way back up and only one destination, ground level. Naloxone also has a much shorter half-life, so the agony of its precipitated withdrawal will last for about a half hour or less. The half-life of buprenorphine is anywhere from 24 to 48 hours, which ensures ongoing misery for the patient, and with its av¬oid binding, there is no way to titrate with another agonist.

I should also mention that although bu¬prenorphine itself is unlikely to precipitate apnea (due to the “respiratory ceiling” from its partial mu-agonism), it can cause fatal overdoes in combination with other respira¬tory depressants. Since we often administer naloxone to unconscious patients with no knowledge of their recent drug consumption, it would be dangerous to give buprenorphine to such patients. The patient we assume over¬dosed on heroin might have actually taken a few Xanax, and the buprenorphine we ad¬minister might very well push them from hy¬poventilation to full respiratory arrest. Not to mention that the long half-life of buprenor¬phine means that they will be on the ventilator while the drug is metabolized over days.

I felt it was important to clear up these implied misunderstandings since most emergency physicians are unfamiliar with bu¬prenorphine and its applications. I hope that this serves to educate rather than frighten phy¬sicians from adopting buprenorphine in their practice, and I look forward to more coverage of emergency addiction management.

Christopher S. Kang, MD, FACEP, FAEM
Tampa, Florida

ACEP Participates in Buprenorphine Discussion

On Oct. 4, 2018, ACEP participated in a meeting along with other provider groups to discuss with top officials of the Trump administration challenges they have had with providing bu¬prenorphine as MAT. In attendance were the Surgeon General, Vice Adm. Jerome Adams, MD, MPH; Drug Enforcement Administration (DEA) Assistant Administrator John J. Martin; White House Office of National Drug Control Policy Acting Director James Carroll; HHS As¬sistant Secretary for Health Adm. Brett Giroir, MD; Assistant Secretary for Mental Health and Substance Abuse Eleanor McCance-Katz, MD, PhD; and Chief Medical Officer for the Office of the Assistant Secretary for Health, Vanilia Sin¬gh, MD, MACM. ACEP member Reuben Strayer, MD, and ACEP/Washington, D.C., staff partici¬pated on behalf of ACEP.

The provider groups shared the barriers they face in prescribing buprenorphine, in¬cluding those brought by the X-waiver and training process; they offered suggestions for regulatory solutions. The senior admin¬istration officials were particularly inter¬ested in discussing the 72-hour rule, which allows emergency physicians to administer buprenorphine without being waivered, but requires patients to return to the emergency department for each 24-hour dosage, up to a maximum of three days. The officials conclud¬ed the meeting with commitments to examine potential regulatory approaches to add flex¬ibility to the 72-hour rule as well as to provide DEA agents with additional training to ensure their enforcement efforts are appropriate and sensitive to patient needs.

ACEP Weighs in on Draft Legislation Regarding Out-of-Network Billing Issues

ACEP President Vidor Friedman, MD, FACEP, recently met with Sen. Bill Cassidy, MD (R-LA) and Sen. Todd Young (R-IN), two members of the Senate Bipartisan Price Transparency Workgroup, regarding their draft legislation on how to address out-of-network billing is¬sues. The workgroup recently released a dis¬cussion draft of its legislation and requested

feedback and input on how to best address this issue. Dr. Friedman and ACEP staff also met separately with the health staff of the senators in the workgroup for a more detailed discussion of ACEP’s positions on the draft legislation. On Oct. 10, ACEP submitted a let¬ter providing feedback on the draft bill. Read it at https://acep.org/globalassets/new-pdfs/advocacy/acep-response-on-senate-price-transparency-wg-discussion-draft-10.10.2018.pdf. ACEP’s letter is part of an ongoing con¬versation with this bipartisan workgroup that began in March 2018.

Board Member Recognized as Health Care Influencer

Mark S. Rosenberg, DO, MBA, FACEP, has been recognized as a New Jersey health care influ¬encer, primarily for his work in battling the opioid crisis in emergency departments. Dr. Rosenberg, along with Alexis LaPietra, DO, created the ALTO program, which helped spur an 8% percent reduction in opioid prescribing over two years.
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How does gender bias affect the ED, and how do we address it?

by MARIE DELUCA, MD; CLAIRE MIN-VENDITTI, MD; MONICA SAXENA, MD, JD

15 Monday morning. You walk into a well-lit room, taking in the familiar surroundings of several colleagues in easy conversation, laughing and joking. You can’t help but feel a change in the atmosphere when you are noticed. The conversation comes to a halt, and you hear someone mutter, “Now we’re in mixed company.”

This scene may bring to mind a woman accidentally walking into a man’s locker room. In reality, this is not uncommon for women physicians in emergency medicine.

As our news feeds fill with stories of women stepping forward to call out sexual harassment and discrimination, we watch with grim recognition. Gender bias in the workplace often occurs through exploitation of gendered power dynamics, causing women to be treated differently and subsequently fall behind in systems historically built around men. At this critical juncture, we have an obligation to look within our medical community to identify and remedy sexism, from frank harassment to subtle but equally damaging unconscious bias. Marginalization and discrimination do not need to be intentional to cause harm, and in many cases, men are not even aware of the harm they cause.

Emergency medicine is a male-dominated field. Although the medical field has shifted overall to near gender parity for enrolled medical students, only 27 percent of emergency physicians are women.1 Evidence suggests women are also at a statistical disadvantage in emergency medicine training. A recent article found that women training in emergency medicine received inconsistent feedback on their work when compared to men. While men were consistently told how to improve clinical skills, women received conflicting advice, often centering on assertiveness and confidence.2

Another study found that while women and men entered emergency medicine training with similar skill sets, by the end of residency, men achieved training milestones at a higher rate than women. The achievement gap was measured as equivalent to a full three to four months of training.3

We must ask ourselves what sort of culture exists in our field that causes women to fall behind.

In discussions with female colleagues across several institutions, we discovered common themes. Many male doctors seem unconsciously uncomfortable working with female doctors, and the social environment of the emergency department often centers on gender norms. We hear remarks disparaging female physicians who make more money than their significant others, jokes about women’s clinical judgment being clouded by feelings, and questions about whether women may be inherently less suited for the job.

Emergency medicine is not alone; gender-based exclusion has been described in other medical subspecialties and at multiple training levels.4 Female medical students reported a broad range of discriminatory behaviors from male attendings, such as failing to make eye contact, employing only male pronouns when referring to physicians, and disproportionately encouraging male students compared to their female counterparts. Medical students even reported that these smaller actions disrupted learning more than blatant harassment and discrimination.

Studies show gendered stereotypes adversely affect women’s training and advancement, leading to underperformance and eventually causing women to be treated differently and subsequently fall behind.5

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en’s work performance. Repeated comments that reinforce gender stereotypes, either blatant or subtle, exert a substantial force that influences not only women’s training but also their identity as doctors. One study found that female medical trainees did not feel equipped to navigate unprofessional behavior from male superiors. As a result, the women experienced guilt and resignation that this would become a part of their professional identity.

Personal relationships built early in our medical careers lay the foundation for our professional trajectory. In studies of gender gaps in academic medicine, bias in the workplace is often cited as a limiting factor for career advancement. If fecundity and a sense of camaraderie are restricted to our male colleagues from the start of training, this could have downstream career effects.

Even though seemingly subtle, repeated comments and acts of gender discrimination add up to a substantial force that causes women to question their legitimacy and place in medicine, affecting both their careers and their identities as women and physicians.

Let’s Take Action

How do we fix this? We propose three immediate actions for leadership in our field:

1. Implement bias intervention training and discussions. Bias interventions can occur on both an individual and institution-wide scale. Attendings can incorporate their own experiences into their lectures and grand rounds. A randomized controlled trial of bias interventions found that faculty members trained to acknowledge and actively disrupt their own bias habits were better able to promote gender equity.

2. Work to increase the ratio of women faculty members and create mentorship programs for female residents. A narrative review of studies on women in academic medicine found that a lack of mentorship constitutes a significant barrier for many women who wish to pursue an academic career. Seeing women in positions of leadership inspires other women and helps create an environment supportive of female physicians and trainees. Inviting female lecturers and speakers and creating mentorship and networking events for women are also structured ways for programs to support their female residents.

3. Promote and support research on gender in medicine. Research on the manifestations and impacts of gender bias in our field must be encouraged. This includes increasing support from academic journals for publication of material related to gender bias and creating platforms for sharing ideas, such as the FemnEM conference. This scholarly work ultimately benefits all of us, and academic departments should provide financial support and academic recognition equal to similar departmental administrative and academic endeavors.

All emergency physicians, both men and women, must recognize the presence of sexism within our field and take action to reduce its effects. Let’s acknowledge this issue, open up dialogue, and together build a stronger, more inclusive space for us all.

References

Careful attention should be given to the possibility of continuous seizure activity even if no apparent convulsions are seen. It’s helpful to consider the possibility that seizures were provoked so that their causes can be diagnosed and addressed. Table 1 enumerates causes of provoked seizures in patients without epilepsy.

Published guidelines on imaging in children with new-onset seizures note that in only approximately 2 to 5 percent of cases, the results altered intermediate medical management. While MRI is the most accurate diagnostic modality in pediatric patients, for unstable patients in whom space-occupying lesions need to be excluded, a noncontrast CT scan of brain is the modality of choice as it is rapid, is readily available, and generally does not require sedation. It is unusual to find an acute abnormality on imaging of a normally developed child (not an infant) with a completely normal neurological examination after a brief, nonlocalizing seizure. In these patients, imaging can be done on an outpatient basis as necessary. The same can be said for laboratory testing.

It is important to note that there are few studies prospectively evaluating timing of imaging in children with new-onset epilepsy. Some high- and low-risk characteristics are based on electroencephalogram (EEG) results. Most guidelines also exclude neonatal seizures. A seizure in a neonate almost always requires expedited imaging and an EEG. An otherwise healthy and developmentally normal child older than 24 months after a brief first-time generalized seizure who quickly returns to normal can be discharged without medications with follow-up by pediatric neurology. All of these patients will ultimately need an EEG, and most will require an MRI of the head. As in adults, most children suffering a first unprovoked seizure do not have another one, especially if their EEG is normal. The risk is higher in children with autistic spectrum disorders and if the seizure happens during sleep. If it occurs, recurrent seizure is most common in the first six months after the first one. While sudden unexpected death in epilepsy (SUDEP) is a known and feared entity that is currently impossible to predict in any given patient, most excess mortality in children with epilepsy is not caused by a seizure itself. Great care should be taken when evaluating infants. Sepsis, meningitis, or disseminated herpes infection can manifest initially with a seizure with or without an abnormal temperature. Approach discharge instructions related to trauma and burn prevention, avoiding driving and taking care of babies, and not swimming or lying in a bath without close supervision are of paramount importance.

Febrile Seizures

Febrile seizures are most commonly defined as those occurring in children of defined age (6 to 60 months, per the American Academy of Pediatrics 2008 guideline) without prior afebrile seizures or neurological abnormalities and occurring in association with acute febrile illness when no other precipitating condition is identified. Complex febrile seizures have focal onset, have lateralizing signs, last longer than 15 minutes, are associated with prolonged postictal deficits, or have focal onset, have lateralizing signs, last longer than 15 minutes, are associated with prolonged postictal deficits, or occur more than once in a given acute febrile illness. These represent approximately 40 percent of all febrile seizure presentations. Febrile seizures that are not complex are defined as simple. Children typically are highly febrile if presenting soon after the seizure; low-grade fever is unusual with febrile seizures unless antipyretics were already given. Seizures lasting more than five minutes currently meet criteria for status epilepticus (SE). Management of patients with simple febrile seizures in the emergency department is similar to the management of children presenting with fever without a seizure and focuses on exclusion of serious illness such as meningitis or sepsis. The 2011 guidelines from the American Academy of Pediatrics do not recommend routine imaging, EEG, lumbar puncture, blood work, or urinalysis solely because of a simple or complex febrile seizure. Unimmunized young children (less than one year old), those on antibiotics, or those with sick appearance warrant a more extensive workup. Children seizing in the emergency department can be given benzodiazepines with intramuscular (IM), IV, intranasal, and rectal routes described. The decision to prescribe rectal diazepam for use

### Table 1: Select Causes of Provoked Seizures

<table>
<thead>
<tr>
<th>EXAMPLES</th>
<th>CONDITIONS</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased intracranial pressure</td>
<td>Meningitis, encephalitis, brain parasites and neoplasms, intracranial hematoma, idiopathic cerebral edema</td>
<td>National origin and travel history can point to a possibility of neurocysticercosis or malaria. Cerebral edema can occur as a complication of diabetic ketoacidosis.</td>
</tr>
<tr>
<td>Ischemic insult to brain</td>
<td>Ischemic stroke, anoxic brain injury</td>
<td>Stroke is more common in children with sickle cell disease, congenital heart disease, and acute leukemia.</td>
</tr>
<tr>
<td>Poisons</td>
<td>Wide range of street drugs, various household chemicals, over-the-counter and prescription medications, and folk remedies can cause seizures in overdose or lower seizure threshold and cause susceptible patients to have a seizure</td>
<td>Bupropion is a commonly prescribed antidepressant that causes seizures in overdose. Organophosphate insecticides and chemical warfare agents can cause seizures in exposed individuals. Camphor-containing folk remedies can cause severe seizures. Isoniazid overdose causes seizures that respond to pyridoxine.</td>
</tr>
<tr>
<td>Metabolic derangements</td>
<td>Severe hyponatremia, hypoglycemia, hyp- or hypercalcemia</td>
<td>Infants, especially younger than 6 months, will drop their plasma sodium levels if given too much free water in their formula. Adolescents may be prone to “voluntary” water poisonings as a result of peer pressure.</td>
</tr>
<tr>
<td>Intracranial hemorrhage</td>
<td>Traumatic; from minimal trauma or spontaneous in coagulopathic patients; arteriovenous malformation, berry aneurysm</td>
<td>Possibility of nonaccidental trauma should be carefully considered. Seizure prophylaxis after traumatic brain injury is highly controversial.</td>
</tr>
<tr>
<td>Withdrawal</td>
<td>Neonates exposed in utero to sustained levels of opioids, sedative/hypnotics, and/or alcohol</td>
<td></td>
</tr>
<tr>
<td>Febrile seizures</td>
<td>Typical or atypical</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2: Important Diagnostic Tests for Patients in Status Epilepticus

<table>
<thead>
<tr>
<th>EXAMPLES</th>
<th>CONDITIONS</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consider metabolic causes; obtain bedside glucose and blood electrolytes levels (sodium, potassium, calcium, and magnesium).</td>
<td>Obtain cardiac rhythm strip and ECG looking for signs of toxicological emergencies. In patients with epilepsy, ask caregivers about compliance with medications; send AED levels as appropriate. Consider head CT, lumbar puncture, and brain MRI. Consider IV pyridoxine if any concern for isoniazid toxicity dose, 70 mg/kg to 5,000 mg max or equal to amount of isoniazid ingested. Consider possibility of PNES.</td>
<td></td>
</tr>
</tbody>
</table>

### Table 3: Suggested Intervention Sequence in Treatment of Pediatric Status Epilepticus

1. Rapidly address airway or breathing failure.
2. Administer fluid bolus for hypotension.
3. Correct any known electrolyte or glucose derangements rapidly.
4. Administer bicarbonate IV bolus for known or suspected tricyclic drug overdose.
5. Administer benzodiazepines; midazolam IM 0.1–0.2 mg/kg; lorazepam 0.1 mg/kg or diazepam 0.1–0.3 mg/kg IV push may be repeated once. Buccal and nasal routes are described for midazolam and rectal route for diazepam, but absorption may be unreliable. 6. Second-line agents are valproic acid, phenytoin or fosphenytoin, or levetiracetam. Phenobarbital can be used but would frequently require ventilatory support if given. There are case reports of lacosamide and topiramate used successfully. 7. Refractory seizure activity should prompt general anesthesia with either propofol (caution advised, especially in infants), midazolam, or barbiturates. Ketamine and intravenous magnesium have been used successfully for intractable SE. Other therapies, including ketogenic diet, hypothermia, and neurosurgery for super-refractory SE, are mostly in the pediatric ICU physician’s domain.
in subsequent febrile seizure episodes is controversial and must be balanced with potential of apnea after its administration. As the seizure typically occurs when fever spikes, antipyretics do not help to prevent first or subsequent febrile seizures.

SE is defined as seizure activity lasting more than five minutes or recurrent seizures without recovery in between. It is the most common neurological emergency in childhood. Prolonged seizure activity can permanently damage neurons; the longer a seizure lasts, the less likely it is to stop spontaneously and the less likely it is to respond to standard antiepileptic drugs (AEDs). SE appears to increase expression of drug efflux proteins in the brain, thus decreasing AED levels there. As it is impossible to predict how long a given seizure will last, it’s best to administer appropriate medications without delay by the fastest reliable route available and escalate therapy as necessary. Intramuscular midazolam administration has been shown to be safe and effective for prehospital SE. IV access can be challenging in seizing children, especially if the veins were extensively used in the past. An intraosseous line can be lifesaving in this situation. Early cardiopulmonary monitoring and supplemental oxygen are recommended for all patients.

NOTE: AEDs with US Food and Drug Administration boxed warnings are carbamazepine, felbamate, lamotrigine, perampanel, and valproic acid.

### Table 4: Select Antiepileptic Drugs (AEDs)

While numerous new AEDs are now available, it’s unknown if they are any more effective and safer than the old ones. While AEDs can lower the chance of a seizure occurrence, they do not treat epilepsy or prevent the development of it.

<table>
<thead>
<tr>
<th>DRUG</th>
<th>INDICATIONS</th>
<th>CONTRAINDICATIONS</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenytoin</td>
<td>Focal seizures, generalized tonic-clonic seizures</td>
<td>Not for absence or myoclonic seizures; causes blood sugar to rise</td>
<td>Frequent side effects</td>
</tr>
<tr>
<td>Fosphenytoin</td>
<td>Focal seizures, generalized tonic-clonic seizures</td>
<td>Same as phenytoin</td>
<td>Can be given IM, unlike phenytoin</td>
</tr>
<tr>
<td>Valproic acid</td>
<td>Broad spectrum</td>
<td>Severe liver disorders</td>
<td></td>
</tr>
<tr>
<td>Carbamazepine</td>
<td>Focal seizures, generalized tonic-clonic seizures</td>
<td>Can exacerbate absence and myoclonic seizures</td>
<td>Severe and fatal dermatological reactions, especially in patients of Asian ancestry, where screening for HLA-B*1502 allele is recommended prior to treatment initiation</td>
</tr>
<tr>
<td>Ethosuximide</td>
<td>Absence seizures only</td>
<td>Any other form of epilepsy</td>
<td>Considered first choice for absence seizures, but in our experience is rarely prescribed by neurologists</td>
</tr>
<tr>
<td>Levetiracetam</td>
<td>Myoclonic, focal seizures, generalized tonic-clonic seizures</td>
<td>Caution in patients with suicidal behavior or ideation</td>
<td>Widely used due to low incidence of side effects; unclear how effective it is</td>
</tr>
<tr>
<td>Lacosamide</td>
<td>Focal seizures</td>
<td></td>
<td>Was not studied in children younger than 16 years</td>
</tr>
<tr>
<td>Zonisamide</td>
<td>Focal seizures</td>
<td>Cross-allergic reaction to sulphonamides</td>
<td>Unclear how effective it is</td>
</tr>
<tr>
<td>Perampanel</td>
<td>Focal seizures</td>
<td>Severe renal or hepatic disease</td>
<td>Avoid abrupt withdrawal</td>
</tr>
<tr>
<td>Oxcarbazepine</td>
<td>Focal seizures, generalized tonic-clonic seizures</td>
<td>Not for absence or myoclonic seizures</td>
<td>May be safer with fewer side effects than carbamazepine</td>
</tr>
<tr>
<td>Lamotrigine</td>
<td>Absence, focal seizures, infantile spasms, generalized tonic-clonic seizures</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vigabatrin</td>
<td>Mostly for infantile spasms</td>
<td>Visual field defects</td>
<td></td>
</tr>
<tr>
<td>Phenobarbital</td>
<td>Myoclonic, focal seizures, generalized tonic-clonic seizures</td>
<td>Not for absence or myoclonic seizures</td>
<td>Frequent severe side effects</td>
</tr>
<tr>
<td>Clobazam</td>
<td>Broad spectrum</td>
<td>Not recommended for first-line treatment</td>
<td></td>
</tr>
<tr>
<td>Clonazepam</td>
<td>Broad spectrum</td>
<td>Difficult to use for long-term epilepsy management</td>
<td></td>
</tr>
<tr>
<td>Felbamate</td>
<td>Atonic, tonic seizures in Lennox-Gastaut syndrome</td>
<td>Hepatic disease</td>
<td>For use when other agents are ineffective</td>
</tr>
<tr>
<td>Gabapentin</td>
<td>Focal seizures</td>
<td>Not for absence or myoclonic seizures</td>
<td>Unclear how effective it is</td>
</tr>
<tr>
<td>Pregabalin</td>
<td>Focal seizures</td>
<td>Not for absence or myoclonic seizures</td>
<td>For seizures refractory to other agents only</td>
</tr>
<tr>
<td>Tiagabine</td>
<td>Focal seizures</td>
<td>Not for patients with mood disorders, suicidal ideation</td>
<td>Can cause seizures in patients without a seizure disorder</td>
</tr>
<tr>
<td>Rufinamide</td>
<td>Focal, atonic seizures in Lennox-Gastaut syndrome</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adrenocorticotropic hormone, corticosteroids</td>
<td>Infantile spasms, seizures in Lennox-Gastaut syndrome</td>
<td></td>
<td>Important implications for emergency physicians in infection and shock management</td>
</tr>
<tr>
<td>Cannabinoids</td>
<td>Case reports for treatment of severe drug-resistant epilepsy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ketamine</td>
<td>Case reports for treatment of resistant status epilepticus</td>
<td></td>
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</tbody>
</table>

NOTE: AEDs with US Food and Drug Administration boxed warnings are carbamazepine, felbamate, lamotrigine, perampanel, and valproic acid.
Nonconvulsive SE (NCSE)

Prolonged brain seizure activity on EEG in a patient with altered mental status but without convulsions defines NCSE. It can present separately as an acute confusional state or develop following an observed seizure. A wide variety of symptoms have been described, ranging from aphasia to severe agitation or coma. It appears to have a similar incidence in children and adults and is not rare. In settings where immediate EEG is not available, its recognition can be quite challenging. In our experience, in unclear cases, cautious administration of a weight-appropriate benzodiazepine dose can, at times, result in dramatic improvement in mental status aiding in diagnosis. Similarly to patients with convulsive SE, these patients need emergent workup focusing on diagnosing life-threatening etiologies, continuous EEG monitoring, and expedient AED administration. As is the case with convulsive SE, the prognosis mostly depends on etiology and the degree of neurological impairment.

Psychogenic Nonepileptic Seizure (PNES) and Other Seizure Mimics

PNES is defined as repeated and frequently intractable seizure activity in the absence of epileptogenic changes on concurrently recorded EEG. Video EEG is necessary to firmly establish the diagnosis. Similarly to patients with convulsive SE, patients with PNES need emergent workup focusing on diagnosing life-threatening etiologies, continuous EEG monitoring, and expedient AED administration. As is the case with convulsive SE, the prognosis mostly depends on etiology and the degree of neurological impairment.

Special Populations

Seizures are a common complaint in children presenting to the emergency department. The initial focus should be on stabilization of vital functions as well as rapid diagnostic workup to exclude treatable secondary causes. Febrile seizures are a unique pediatric pathology where management now mostly focuses on the concurrent, acute febrile illness. Convulsive and nonconvulsive SE are true neurological emergencies and should be stabilized as soon as possible. PNES is common, and video EEG monitoring is required to firmly establish the diagnosis.

Conclusion

Seizures are a common complaint in children presenting to the emergency department. The initial focus should be on stabilization of vital functions as well as rapid diagnostic workup to exclude treatable secondary causes. Febrile seizures are a unique pediatric pathology where management now mostly focuses on the concurrent, acute febrile illness. Convulsive and nonconvulsive SE are true neurological emergencies and should be stabilized as soon as possible. PNES is common, and video EEG monitoring is required to firmly establish the diagnosis.

References

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- Lumbar puncture
- Arthrocentesis
- Intra-osseous cannulation
- Wound care: I & D abscesses, suture repair
- Peripheral nerve blocks (facial, rib, wrist, ankle, femoral)

**Non-Cadaver Lab - 7.0 hours of CME**
- Ultrasound Techniques, including vascular access, peripheral nerve blocks, and FAST exams for physicians – taught by EMsono
- EZ-Io demonstration station

**Airway Lab – Optional – 4.0 hours of CME**
- Airway station with manikin intubation using adult and pediatric models (including King Vision, LMA, Combitube, SALT and others as available)

**Prerequisite – 8.5 hours of CME**
Participants must successfully complete 10 on-line EM Skills and Procedures modules as prerequisites for attending the lab. You will be awarded a total of 8.5 CME credits upon completion.

CME: The Center for Emergency Medical Education (CEME) is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

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The Center for Emergency Medical Education (CEME) designates this live activity for a maximum of 26.5 AMA PRA Category 1 Credits™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.
In June 2018, the ACEP Board of Directors approved a clinical policy on the evaluation and management of adult patients presenting with suspected non–ST-elevation acute coronary syndrome (NSTE ACS). In its complete form, this policy can be found on the ACEP website at www.acep.org/patient-care/clinical-policies/non-elevation-acute-coronary-syndromes.

Emergency physicians routinely rule out ACS in patients presenting with chest pain and have become very good at targeting timely interventions in the obvious cases of ST-elevation myocardial infarction but still miss up to 2 percent of acute myocardial infarctions, particularly those with non–ST-elevation myocardial infarction (NSTEMI). The purpose of this policy was to focus on the initial diagnosis and treatment of patients who present with potential NSTE ACS.

In developing the policy, the ultimate outcome measure was the 30-day incidence of major adverse cardiovascular event (MACE). This includes cardiovascular death and myocardial infarction, as well as what some argue is more subjective in terms of actual need, coronary revascularization. Most emergency physicians strive to attain a miss rate of less than 1 percent. However, it is questionable if the benefits of further testing outweigh the risks of harm of untreated disease once that threshold reaches 2 percent, which the committee felt was a more realistic expectation. With shared decision making, patients may be willing to accept rates higher than those to which physicians hold themselves accountable.

Emergency departments are so often congested with patients awaiting serial testing (laboratory and noninvasive) to rule out potential ACS that entire units have been dedicated to observing these patients, yet there is questionable benefit. Researchers have been looking for diagnostic strategies, single or serial troponins, and ECGs to try to identify at-risk patients sooner and expedite their transition of care. One strategy adopted internationally and slowly taking hold in the United States is the advent of high-sensitivity troponins. Although these have great promise for detecting potential disease sooner, without proper protocols, they can lead to excessive false positives. There is great promise in expediting the care of patients suspected of NSTE ACS. Ultimately, the purpose of this policy was to help ED clinicians expedite the care of patients presenting with chest pain who are at risk for NSTE ACS. The first three questions focus on initial identification of patients at low risk for MACE, using history and limited testing. Are there patients with suspected ACS who are safe to discharge based on initial risk stratification? Do serial troponins really help, and how long do we have to wait to do that second troponin? Does getting early noninvasive diagnostic testing for ACS prior to discharge from the emergency department really help decrease MACE rates? The goal was to see if there were strategies to expedite the initial evaluation and discharge of these patients without resorting to prolonged ED stays (four to six hours or longer) while still limiting the number of 30-day MACE.

The fourth and last question asks at the role of early antithrombotic therapy in patients with acute NSTEMI and focuses on timing. Because of early literature and general consensus on the accepted use of heparin and enoxaparin, the literature search and recommendations targeted newer oral antithrombotic agents. The goal was to ensure emergency physicians were not held accountable for timely administration of such agents if such delays were not associated with worse outcomes.

For each critical question, a structured literature review was performed, evidence was systematically graded (see Table 1), and evidence-based recommendations were presented.

### CRITICAL QUESTIONS

1. **In adult patients without evidence of ST-elevation ACS, can initial risk stratification be used to predict a low rate of 30-day MACE?**

   **Patient Management Recommendations**
   - **Level A recommendations.** None specified.
   - **Level B recommendations.** In adult patients without evidence of ST-elevation ACS, the history, ECG, age, risk factors, and troponin (HEART) score can be used as a clinical prediction instrument for risk stratification. A low score ($\leq 3$) predicts 30-day MACE risk rate within a range of 0 to 2 percent.
   - **Level C recommendations.** In adult patients without evidence of ST-elevation ACS, other risk stratification tools, such as thrombolysis in myocardial infarction (TIMI), can be used to predict the rate of 30-day MACE.

2. **In adult patients with suspected acute NSTE ACS, can troponin testing within three hours of ED presentation be used to predict a low rate of 30-day MACE?**

   **Patient Management Recommendations**
   - **Level A recommendations.** None specified.
   - **Level B recommendations.** None specified.
   - **Level C recommendations.** 1. In adult patients with suspected acute NSTE ACS, conventional troponin testing at 0 and 3 hours among low-risk ACS patients (defined by HEART score $0$ to $3$) can predict an acceptable low rate of 30-day MACE.
     - A single high-sensitivity troponin result below the level of detection on arrival to the emergency department or negative serial high-sensitivity troponin results at 0 and 2 hours are predictive of a low rate of 30-day MACE.
     - In adult patients with suspected acute NSTE ACS who are determined to be low risk based on validated accelerated diagnostic pathways that include a nonischemic ECG result and negative serial high-sensitivity troponin testing results both at presentation and at 2 hours can predict a low rate of 30-day MACE allowing for an accelerated discharge pathway from the emergency department.
     - In adult patients with suspected NSTE ACS in whom acute myocardial infarction has been excluded, does further diagnostic testing (eg, provocative, stress test, computed tomography [CT] angiography) for ACS prior to discharge reduce 30-day MACE?

### Patient Management Recommendations

1. **Level A recommendations.** None specified.
2. **Level B recommendations.** Do not routinely use further diagnostic testing (CT coronary angiography, stress testing, myocardial perfusion imaging) prior to discharge in low-risk patients in whom acute myocardial infarction has been ruled out to reduce 30-day MACE.
3. **Level C recommendations.** Arrange follow-up in one to two weeks for low-risk patients in whom myocardial infarction has been ruled out. If no follow-up is available, consider further testing or observation prior to discharge (consensus).

4. **Should adult patients with acute NSTEMI receive immediate antithrombolytherapy in addition to aspirin to reduce 30-day MACE?**

   **Patient Management Recommendations**
   - **Level A recommendations.** None specified.
   - **Level B recommendations.** None specified.
   - **Level C recommendations.** P2Y12 inhibitors and glycoprotein IIb/IIIa inhibitors may be given in the emergency department or delayed until cardiac catheterization.

In conclusion, patients who present with chest pain with low risk for ACS (eg, HEART score $\leq 3$) and a normal troponin at 0 and 3 hours post-presentation may be discharged safely, with less than a 2 percent risk of subsequent 30-day MACE. The advent of high-sensitivity troponins will help accelerate this rule-out protocol. In such low-risk cases, we could find no data to support subsequent noninvasive testing. Our ultimate goal should be to prevent harm from missing MACE, but also from overtesting patients. Finally, our last question confirms that it is acceptable to delay further antithrombolytherapy, beyond heparin, especially if there are concerns over potential adverse bleeding or competing priorities.

**Reference**


DR. TOMASZEWSKI is professor of clinical emergency medicine at the University of California San Diego Health and chief medical officer of El Centro Regional Medical Center.
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es of breast cancer, colon cancer, and AIDS combined. With a reported 500 deaths per day, mortality for sepsis paralleled that of out-of-hospital myocardial infarction, costing $16.7 billion nationally. Mortality estimates for the period ranged from 18 to 75 percent, depending on illness severity and population. A meta-analysis reported mortality as 47 percent from 1991 to 1995 and 29 percent from 2006 to 2009, representing a 3 percent annual reduction across studies.

Most sepsis occurred external to the ICU. Approximately 60 percent of the Angus et al cases were identified outside of the ICU. In a global study, 88 percent of septic patients were identified outside of the ICU (66 percent in the emergency department, 32 percent on the ward, and 12 percent in the ICU). W 2001–2004 Wang et al estimated that there were 571,000 suspected community-acquired severe sepsis cases presenting annually to emergency departments. Mean ED length of stay was almost five hours, with more than 20 percent of the visits lasting in excess of six hours.

2009–2011 Revised estimates total 850,000 ED sepsis visits per year. This constitutes one out of every 120 ED patients. More than 70 percent of septic ED patients were admitted, with 34 percent requiring ICU admission. However, almost half had an ED length of stay of more than four hours, and more than 10 percent stayed more than eight hours.

Gaieski et al demonstrated how sepsis prevalence and mortality rates change depending on the definition applied. However, what is clear is that the majority of sepsis cases are identified and initial resuscitation for them is completed outside of the ICU.

“And yet, at the present time, the subject is by no means fully elucidated, and it is not even possible to give a general definition of the term septicemia, which could currently represent all the different conceptions of its nature ... at the present time.” —W.W. Van Arsdale

SEPSIS DEFINITIONS

1990s Sepsis definitions have evolved over time (see Table 1). The 1991 American College of Chest...
Inflammatory criteria, >1 organ dysfunction (AMS, dysfunction of number of failing organs). How- ever, the model was complex and difficult to standardize and quantify, and it was never universally adopted. Both of the early defini- tions were based primarily on consensus of expert opinion.

2016 The Sepsis-3 definition focused primarily on infection and end-organ dysfunction. Sepsis became infection and worsening or new end-organ dysfunction based upon the Sequen- tial Organ Failure Assessment (SOFA) scale or quick SOFA. Sepsis includes vasopressor- dependent hypotension and excludes lactate. Septic shock was defined as vasopressor-de- pendent hypotension with lactate elevation. The Sepsis-3 process made several important advancements. It would be the first definition conference to: 1) employ data analyses in conjunc- tion with clinical experience to define a cause, solely relying on expert opinion, 2) attempt a standardized process, and 3) acknowledge continued refinement as science and our un- derstanding evolves. However, the Sepsis-3 definitions have not been universally adopted by emergency medicine, critical care, and low- and middle-income countries. The Surviving Sepsis Campaign (SSC) ultimately decided upon an intermediate definition, where sepsis was defined by the previous severe sepsis defi- nition (not including persistent hypotension) and septic shock remained any vasopressor- dependent hypotension. The Surviving Sepsis Campaign (SSC) ultimately decided upon an intermediate definition, where sepsis was defined by the previous severe sepsis defi- nition (not including persistent hypotension) and septic shock remained any vasopressor- dependent hypotension. Henning et al did an excellent job illustrating implications of the different definitions. Unfortunately, sepsis continues to be an elusive condition, recognized by most and unus- takably defined by none.

DEFINITION DEVELOPMENT

Definition usefulness is based upon the pur- pose for which it is derived, combined with the ability to effectively operationalize it within a target environment. Purpose is based upon the context of value and prioritization. Effective operationalization involves pragmatic imple- mentation within the environment to which it is applied. “Emergency medicine is in the business of sensitivity.” —Donald Yealy

IMPORTANT OF PERSPECTIVE

When working in the ICU, the intensivist is concerned about specificity. There are lim- ited resources appropriately reserved for the most acutely ill. If intensivist assessment of a patient denied ICU admission is incorrect, the patient is invariably still in a hospital-based, monitored setting. With unexpected deterio- ration, accommodations can be made and resources leveraged. In the emergency depart- ment, the physician is concerned about sen- sitivity, or who could be missed. An incorrect assessment of a patient who decompensates after being discharged home is not afforded the same accommodations and may have sig- nificant consequences. For example, when the SSC data from the septic shock manuscript were divided into hypotension and normal lac- tate or normotension and lactate ≥4 mmol/L, the mortality was essentially the same at 30 percent. Hypotension was included in the defi- nition of sepsis, and lactate was not. Although the authors never dissuaded using lactate be- cause, from their perspective, lactate added no additional predictive validity for mortality or ICU resources, it was only retained during hypotension. From an emergency medicine perspective, a quick point-of-care test that identifies a patient at risk is of great value. If mortality and a prolonged ICU stay are averted, it could signal a job well-done through early identification and treatment rather than unnecessary information. If definition usefulness is based on priori- ties, it seems important to know what is valued the most by the caregivers who are implement- ing the definition.

“Labeling already differentiated ICU patients is very different than sorting, or triaging, undifferentiated patients in the ED or on hospital wards.” —David Gateski and Munish Goyal

SURVIVING SEPSIS CAMPAIGN

AECF’s involvement with the SSC began in 2003, when an emergency physician who had just completed a critical care fellowship was in Amsterdam taking the European critical care board exams. As the US boards were not an option. While there, the American Thoracic Society paid him a lecture. The physician went to the SSC presentation in an auditorium filled with hundreds of intensivists and listened to the Phase II and III plans for this international critical care collaborative. During the question period, the emergency physician asked a simple inquiry: “If 50 per- cent of sepsis in the United States presents through the emergency department, would it be of benefit to have emergency medicine involved?” A proposal was drafted, and conversa- tions with the AECF Board of Directors began through Arthur Kellermann, MD, MPH, FACEP. A lecture with panel discussion was arranged for the AECF Scientific Assembly. A room accommodating 200 people, approximately 25 people attended that inaugural lecture. There were no other sepsis talks at AECF that year. Sepsis didactics became a standing compo- nent of Scientific Assembly. An article documenting AECF’s alliance with the SSC and a letter from the AECF president regard- ing AECF’s involvement were sent to 35,000 members. During this time, sepsis mortal- ity was high, with an estimated 500 deaths per day in the United States alone. As part of Phase I, Graham Ramsay, MD, conducted a survey of more than 1,000 physicians from six countries and found fewer than 17 percent of physicians agreed on a common definition or a standard treatment. In 2003, AECF par- ticipated with representatives from 11 interna- tional medical professional organizations in the creation of the first set of sepsis man- agement guidelines advising a standardized management approach to sepsis. Guided by Phillip Dellinger, MD, there was meaningful collaboration. During periods of intense aca- demic discussion, where few agreed on how specific data were interpreted, he would care- fully consider the different perspectives and then skillfully find common ground. AECF contributed meaningfully with 100 percent of the major content revisions and 75 per-
The editor was written in response to the Hour-1 Bundle, it was not published due to a Society of Critical Care Medicine (SCCM) policy of having concerns directed to the guideline committee. This is a challenging time. ACEP is working diligently with SCCM and the SSC. Although individual service lines can incrementally impact mortality, no service line can truly optimize sepsis survival alone. From 2003 to 2015, patient survival has improved due in part to this global cooperative work. We hope we can get back to the real mission, caring for the patients we are called upon to serve. Our patients need us all to be more than individual service lines; they need us to provide a service.  

Part 2 of this history will appear in the December issue.

References
26. Wang HE, Shapiro NI, Angus DC, et al. National estimates of severe sepsis and septic shock: impact mortality, no service line can truly optimize sepsis survival alone. From 2003 to 2015, patient survival has improved due in part to this global cooperative work. We hope we can get back to the real mission, caring for the patients we are called upon to serve. Our patients need us all to be more than individual service lines; they need us to provide a service.
Derm Intervention

by GINA VENTO, MD; MATTHEW ROEHRS, DO; AND DAVID EFFRON, MD, FACEP

The Case

A 30-year-old man presented to the emergency department with a left leg wound that had been present for five years and “worsening over the past four months.” The patient presented because he was on the verge of losing his job because of the odor from the wound. The patient described the lesion as painful, making it difficult to ambulate. He denied recent trauma to the area. However, he had a remote history of a car accident years ago but didn’t remember any injury to the leg. He reported subjective fevers, nausea, and vomiting over the past few weeks. His past medical history was significant for smoking and nephrotic syndrome. No family history of autoimmune disorders was reported, and he denied any prior dermatological conditions.

Upon entering the patient’s room, an intense, putrid odor was noted. The patient appeared in no acute distress, and his vital signs were normal. The patient had large ulcers with undermined borders and erythematous edges on the entire aspect of his left lateral and medial leg (see Figures 1 and 2). The leg was diffusely tender to palpation, with distal strength and sensation intact. No edema was noted. Dorsalis pedis and posterior tibial pulses were intact.

Workup for the patient included basic labs and a wound culture. Radiographs of the tibia showed a large soft tissue defect overlying the medial aspect of the proximal and mid tibia with no periosteal reaction to suggest osteomyelitis. The patient’s labs were significant for a leukocytosis of 13.7, hemoglobin of 9.7, normal lactate, and renal dysfunction with a creatinine of 2.67. The patient was started on vancomycin and Zosyn (piperacillin and tazobactam) and admitted to the medicine service. The patient did not show up for his follow-up visits and was lost to follow-up.

Background

Skin complaints are common reasons for visits to the emergency department, with skin infections, such as cellulitis, being one of the most common. Differentiating toxic skin conditions from benign skin conditions is essential. This patient’s condition could be concerning for a necrotizing infection, but that possibility is less likely given the time frame. Initial concern was for a chronic infection. The patient’s wound cultures grew *Staph*, which could have been a contaminant versus infection. Punch biopsy revealed acute and chronic inflammation. Dermatology concluded that this presentation was consistent with pyoderma gangrenosum.

The incidence of pyoderma gangrenosum is estimated to be three to 10 cases per million people per year. It is often associated with systemic disease such as inflammatory bowel disease, arthropathies, and hematologic diseases. The pathogenesis is thought to be related to neutrophil dysfunction with a component related to abnormal immune system response. Treatment is dependent on the severity of the disease. Local pyoderma gangrenosum is treated with a barrier cream and wound care to try to prevent infection. Topical steroids can also be used. If the disease is more severe, systemic corticosteroid therapy should be considered. If this does not seem to be helping, cyclosporine should be considered. Systemic steroids can result in improvement in as little as one week. It is also important to address underlying systemic disease that may be contributing to the condition.

Case Resolution

This patient was discharged on prednisone 50 mg twice a day, with a plan to taper over several weeks. It was also recommended that the patient use Vaseline with Zerol form and Kerlix for dressing changes. Cyclosporine could not be considered for this patient as he had renal disease. The patient was discharged from the hospital and was told to follow up closely with dermatology. The patient did not show up for his follow-up visits and was lost to follow-up.

DR. VENTO is an emergency medicine resident at MetroHealth in Cleveland.

DR. ROEHRS AND DR. EFFRON are emergency physicians at MetroHealth.

RESOURCES FOR FURTHER READING

MEDICAL DIRECTOR POSITIONS

- HealthAlliance Hudson Valley: Broadway Campus
  Kingston, New York
- Northeast Methodist Hospital
  San Antonio, Texas
- OSF Sacred Heart Medical Center
  Danville, Illinois
- Palms West Hospital
  West Palm Beach, Florida
- St. Mary's Medical Center
  Hobart, Indiana

ASSOCIATE DIRECTOR POSITIONS

- Community Medical Center
  Toms River, New Jersey

COAST-TO-COAST POSITIONS

- Academic • Community • Pediatric
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There is an ethical imperative for the FOAM community to establish firm safeguards, aggressively self-regulate, and promote the skepticism that is the bedrock of scientific advancement.

**ETHICAL ISSUES WITH FOAM**

The online movement should not overlook ethical standards from traditional medical education.

by NATHAN G. ALLEN, MD, FACEP; EASHWAR B. CHANDRASEKARAN, MD, MSC; REBECCA R. GOETT, MD, FACEP, FAAHPM; NICHOLAS H. KLUESNER, MD, FACEP; AND LAURA VEARRIER, MD, DBIOETHICS; ACEP ETHICS COMMITTEE

FOAM, free open access medical education, is an online movement taking place across social media, blogs, and podcasts that is challenging traditional methods of medical education. Its acronym coined in 2012, FOAM represents more than just the content of the learning resources; it is considered to be an ethos, a dedication to the learning and teaching of medicine in a collaborative environment made easily accessible by online platforms. These new educational platforms are changing the way learners engage with educational resources and how research is translated into practice. Recent studies have demonstrated that 97.7 percent of American medical residents are spending at least one hour per week supplementing their traditional academic curricula with podcasts. This rapid expansion and increasing influence of FOAM in emergency medicine suggests a need for ethical analysis. Pros and cons of FOAM from an ethical perspective are outlined in Table 1. The ensuing discussion elaborates on these key issues emergency physicians should consider when utilizing or participating in FOAM.

**Patient Confidentiality**

Our responsibility to protect patient privacy and health care information takes on new complexity in the FOAM and social media environment. The risks, especially when discussing clinical case vignettes or sharing radiographic or electrocardiographic content, are well described. Attention to “de-identification” of publicly shared content requires not only removal of key patient details (eg, names, ages, and birth dates) but also more subtle identifiers such as unique conditions, events, locations, and time lines. When participating in FOAM, emergency physicians should follow the guidelines established in ACEP’s forthcoming policy on use of social media.

**Conflicts of Interest**

The disclosure of FOAM authors’ conflicts of interest should follow the same standards recognized by traditional peer-reviewed journals, namely that all professional and financial conflicts be fully disclosed to readers. In FOAM, this expectation should apply not only to content authors but also commentators. A high-traffic website, social media page, or Twitter feed has significant potential value for advertising revenue. A complete ban on industry sponsorship or ad placement is not a tenable solution, as there can be substantial costs in the creation of high-quality FOAM content. Disclosing these conflicts is crucial. Trans- parent and reduced-bias funding sources for FOAM, such as grants, may mitigate but not reduce the risk posed by conflicts of interest. Similar to the publication bias of journals, there may be selection bias for content that will be of interest to and shared by users. This constant pressure for innovative and engaging posts, particularly in concert with the limitations in peer review, may create content that misrepresents standard practices by emphasizing new techniques and studies. Further- more, the lack of verification of the identity and credentials of commenters clouds the reliability of information and opens the door to covert industry infiltration, astroturfing, and other malicious intents. Social media platforms common to FOAM, as well as social media users in general, have proven to be extremely vulnerable to manipulation and dissemination of false information. There is an ethical imperative for the FOAM community to establish firm safeguards, aggressively self-regulate, and promote the skepticism that is the bedrock of scientific advancement.

**Peer Review**

Attempts have been made to implement formal peer review in FOAM. The blog Academic Life in Emergency Medicine (ALEIM) introduced an “expert peer review” process for providing feedback, edits, and commentary on published articles while still not delaying their release. Furthermore, there are emerging structured mechanisms to evaluate non-traditional educational sources (eg, AIR Series and AIR Score, Medical Education Translational Resources: Impact and Quality [METRIQ], and Social Media Index). The open-access nature of commenting on FOAM resources does allow for a uniquely real-time appraisal. This interactive process gathers the insights of multiple practice backgrounds and experiences and is a form of ground-truthing that is not similarly available in print media. On the contrary, for FOAM in general and for comments in particular, users may be left to assess the authenticity of a statement by either the reputation of the author or the perceived accuracy of a post in a manner that is vulnerable to bias and error.

**Eminence Versus Evidence**

One criticism of FOAM is that it threatens a return to the time of “eminence-based” medicine rather than the evidence-based medicine that underpins modern practice. The validity of this criticism is undermined by the fact that eminent voices continue to have an amplified influence in traditional educational venues. The presence of individuals with unique access to platforms and followings that allow dissemination of information is not exclusive to online communities and can be found in academic and scientific circles as well. FOAM could be considered, in part, a reaction to the crowded space of traditional medical education and may be an attempt to democratize the process of information generation and dissemination. Although the gold standard for medical learning remains the personal review of primary source materials, the time in which practitioners could read all of the literature relevant to their practice has long passed. Reliance on trusted sources to summarize and sort the wheat from the chaff is no longer optional. However, like any educational tool, participation in FOAM without a curricular roadmap can neglect and even create substantial and dangerous knowledge gaps. There is no equivalent to PubMed for FOAM. Reliance on search engines like Goog-
Knowledge Translation Time

FOAM has the ability to decrease the time from knowledge discovery to knowledge integration into clinical practice, though this process continues to be less regulated than traditional methods.1 The traditional methods of inquiry and assimilation of research findings into medical practice can take decades, with an estimated lag from time of inception to clinical practice of as much as 17 to 23 years.13 Traditional medical journals are incorporating FOAM techniques through partnering strategies, The Annals of Emergency Medicine and ALiEM.com have collaborated on online journal clubs; one such encounter had 1,440 readers and 33,229 Twitter audience impressions.16 Also, FOAM is by definition free, and eliminating the cost barrier gives it another advantage.

However, the increased speed of knowledge translation raises the question, How fast is too fast? Significant changes in clinical practice could occur before further study can verify and confirm exciting new findings.6 Future important directions for FOAM are to establish the real-time ability to publish and discuss not only new ideas but also the structure necessary to evaluate them in standardized trials with subsequent peer review.

Distinguishing FOAM from Crowdsourcing

Crowdsourcing is the solicitation of real-time clinical input from others over an electronic platform. Distinguishing crowdsourcing from FOAM may be difficult because crowdsourcing isn’t FOAM, but it may occur in the same space as FOAM activities. For example, a Twitter discussion about the best agent for blood pressure control in aortic dissection is likely FOAM, whereas tweeting, “Help!!! What should I do for my patient with an aortic dissection?” is crowdsourcing. The social media platform SERMO advertises crowdsourcing as a benefit of its network. Crowdsourcing is a seductively appealing and convenient method for patients to seek assistance from others in an online community. FOAM contributors also must keep patient confidentiality paramount and disclose commercial interests or involvement.

Conflicts of interest Community is required to actively participate to maintain the peer-review process and protect the FOAM movement has created collaborative communities capable of ultra-rapid dissemination of information and remote interaction between learners and educators. These pioneering advancements must be coupled with new responsibilities for both educators and learners. The flood of available information must be consciously processed and methodically vetted by those learning through FOAM to maintain the peer-review process and promote evidence over eminence. In addition to this responsibility, FOAM contributors also must keep patient confidentiality paramount and disclose commercial interests or involvement. FOAM will likely continue to grow and expand over time, and attention and research are needed to focus on how FOAM can best integrate with, augment, or supplant more traditional existing resources. Emergency physicians are forerunners in medical education and should continue this leadership role to ensure FOAM evolves responsibly.

References


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<th>TABLE 1: PROS AND CONS OF CHARACTERISTICS OF FOAM</th>
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<tr>
<td>CHARACTERISTIC</td>
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<tr>
<td>Decrease in knowledge translation time</td>
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<td>Low barrier of entry</td>
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<td>Lack of traditional peer review</td>
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<td>No curriculum</td>
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<td>Worldwide access</td>
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<td>Self-promotion</td>
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<td>Variable quality of content</td>
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<td>Forced brevity due to social media platforms</td>
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<td>Professionalism and anonymity</td>
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<td>Ability to crowdsource as an informal consultation</td>
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| CHARACTEBRIC | PRO |
|-----------------------------------------------|
| Conflict of interest | Community is required to actively participate in the identification and disclosure of conflicts of interest |

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| The authors are members of the ACEP ethics committee. |
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CLEVIPREX® (clevidipine) Injectable Emulsion is contraindicated in patients with:

- Allergies to soybeans, soy products, eggs, or egg products;
- Defective lipid metabolism seen in conditions such as pathologic hyperlipemia, lipid nephrosis, or acute pancreatitis if it is accompanied by hyperlipidemia; and
- Severe aortic stenosis.

CLEVIPREX® is intended for intravenous use. Use aseptic technique and discard any unused product within 12 hours of stopper puncture.

Hypotension and reflex tachycardia are potential consequences of rapid upward titration of CLEVIPREX®. If either occurs, decrease the dose of CLEVIPREX®. There is limited experience with short-duration therapy with beta-blockers as a treatment for CLEVIPREX®-induced tachycardia. Beta-blocker use for this purpose is not recommended.

CLEVIPREX® contains approximately 0.2 g of lipid per mL (2.0 kcal). Lipid intake restrictions may be necessary for patients with significant disorders of lipid metabolism.

Dihydropyridine calcium channel blockers can produce negative inotropic effects and exacerbate heart failure. Monitor heart failure patients carefully.

CLEVIPREX® is not a beta-blocker, does not reduce heart rate, and gives no protection against the effects of abrupt beta-blocker withdrawal. Beta-blockers should be withdrawn only after a gradual reduction in dose.

Patients who receive prolonged CLEVIPREX® infusions and are not transitioned to other antihypertensive therapies should be monitored for the possibility of rebound hypertension for at least 8 hours after the infusion is stopped.

There is no information to guide use of CLEVIPREX® in treating hypertension associated with pheochromocytoma.

Most common adverse reactions for CLEVIPREX® (>2%) are headache, nausea, and vomiting.
Brief Summary

CLEVIPREX® (clevidipine) injectable emulsion, for intravenous use

Brief Summary of Prescribing Information

SEE PACKAGE INSERT FOR FULL PRESCRIBING INFORMATION

INDICATIONS AND USAGE

CLEVIPREX is a dihydropyridine calcium channel blocker indicated for the reduction of blood pressure when oral therapy is not feasible or not desirable.

CONTRAINDICATIONS

Known Allergy - CLEVIPREX is contraindicated in patients with allergies to soybeans, soy products, eggs, or egg products.

Defective Lipid Metabolism - CLEVIPREX is contraindicated in patients with defective lipid metabolism such as pathologic hyperlipidemia, lipid nephrosis, or acute pancreatitis if it is accompanied by hyperlipidemia.

Severe Aortic Stenosis - CLEVIPREX is contraindicated in patients with severe aortic stenosis because afterload reduction can be expected to reduce myocardial oxygen delivery.

WARNINGs AND PRECAUTIONS

Need for Aseptic Technique - Use aseptic technique and discard any unused product within 12 hours of stopper puncture (see Storage and Administration).

Hypotension and Reflex Tachycardia - CLEVIPREX may produce systemic hypotension and reflex tachycardia. If either occurs, decrease the dose of CLEVIPREX. There is limited experience with short-duration therapy with beta-blockers as a treatment for CLEVIPREX-induced tachycardia. Beta-blocker use for this purpose is not recommended.

Lipid Intake - CLEVIPREX contains approximately 0.2 g of lipid per mL (2.0 kcal). Lipid intake restrictions may be necessary for patients with significant disorders of lipid metabolism. For these patients, a reduction in the quantity of concurrently administered lipids may be necessary to compensate for the amount of lipid infused as part of the CLEVIPREX formulation.

Negative Inotropy - Dihydropyridine calcium channel blockers can produce negative inotropic effects and exacerbate heart failure. Monitor heart failure patients carefully.

Beta-Blocker Withdrawal - CLEVIPREX is not a beta-blocker, does not reduce heart rate, and gives no protection against the effects of abrupt beta-blocker withdrawal. Beta-blockers should be withdrawn only after a gradual reduction in dose.

Rebound Hypertension - Patients who receive prolonged CLEVIPREX infusions and are not transitioned to other antihypertensive therapies should be monitored for the possibility of rebound hypertension for at least 8 hours after the infusion is stopped.

Pheochromocytoma - There is no information to guide use of CLEVIPREX in treating hypertension associated with pheochromocytoma.

ADVERSE REACTIONS

The following risk is discussed elsewhere in the labeling: Hypotension and Reflex Tachycardia (see Warnings and Precautions (5.2)).

Clinical Trials Experience

CLEVIPREX clinical development included 19 studies, with 99 healthy subjects and 1307 hypertensive patients. Infusions resulted in at least one dose of clevidipine (1406 total exposures). Clevidipine was evaluated in 15 studies in hypertensive patients: 1099 patients with perioperative hypertension, 126 with severe hypertension and 82 patients with essential hypertension.

The desired therapeutic response was achieved at doses of 4-6 mg/hour. CLEVIPREX was infused for ≤24 hours in the majority of patients (n=1199); it was infused as a continuous infusion in an additional 93 patients for durations between 24 and 72 hours. Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice.

Perioperative Hypertension

The placebo-controlled experience with CLEVIPREX in the perioperative setting was both small and brief (about 30 minutes). Table 2 shows treatment-emergent adverse reactions and the category of “any common adverse event” in ESCAPE-1 and ESCAPE-2 where the rate on CLEVIPREX exceeded the rate on placebo by at least 5% (common adverse reactions).

Table 2. Common adverse reactions in placebo-controlled perioperative studies.

<table>
<thead>
<tr>
<th></th>
<th>ESCAPE-1</th>
<th>ESCAPE-2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CLV N=53%</td>
<td>PB0 N=51%</td>
</tr>
<tr>
<td>Any common adverse event</td>
<td>27 (51%)</td>
<td>21 (41%)</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>5 (9%)</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Nausea</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Three randomized, parallel, open-label studies called ECLIPSE, with longer exposure in cardiac surgery patients define the adverse reactions for patients with perioperative hypertension. Each ECLIPSE study compared CLEVIPREX (n=750) to an active comparator: nitroglycerin (NTG, n=278), sodium nitroprusside (SNP, n=283), or nicardipine (NIC, n=193). The pooled mean maximum dose in these studies was 10 mg/hour and the mean duration of treatment was 8 hours.

There were many adverse events associated with the operative procedure in the clinical studies of CLEVIPREX and related to lipid infusion as well as CLEVIPREX lower blood pressure. Thus, the ability to differentiate the adverse event profile between treatments is limited. The adverse events observed within one hour of the end of the infusion were similar in patients who received CLEVIPREX and in those who received comparator agents. There was no adverse reaction that was more than 2% more common on CLEVIPREX than on the average of all comparators.

Serious Adverse Events and Discontinuation – Perioperative Hypertension Studies

The incidence of adverse events leading to study drug discontinuation in patients with perioperative hypertension receiving CLEVIPREX was 4.9% versus 3.2% for all active comparators. For patients receiving CLEVIPREX and all active comparators the incidence of serious adverse events within one hour of drug infusion discontinuation was similar.

Severe Hypertension

The adverse events for patients with severe hypertension are based on an uncontrolled study in patients with severe hypertension (VELOCITY, n=126).

The common adverse reactions for CLEVIPREX in severe hypertension included headache (6.3%), nausea (4.9%), and vomiting (3.2%). The incidence of adverse events leading to study drug discontinuation for CLEVIPREX in severe hypertension was 4.8%.

Less Common Adverse Reactions in Patients with Severe or Essential Hypertension

Adverse reactions that were reported in <1% of patients with severe or essential hypertension included:

- Cardiac: myocardial infarction, cardiac arrest
- Nervous system: syncope
- Respiratory: dyspnea
- Post-Marketing and Other Clinical Experience

Because adverse reactions are reported voluntarily from a population of uncertain size, it is not always possible to estimate reliably their frequency or to establish a causal relationship to drug exposure. The following adverse reactions have been identified during post-approval use of CLEVIPREX: increased blood triglycerides, ileus, hypersensitivity, hypotension, nausea, decreased oxygen saturation (possible pulmonary shunting) and reflex tachycardia.

DRUG INTERACTIONS

Clevidine and its major dihydropyridine metabolite do not have the potential for blocking or inducing any CYP enzyme.

USE IN SPECIFIC POPULATIONS

Pregnancy: Pregnancy Category C - There are no adequate and well-controlled studies of CLEVIPREX use in pregnant women. In animal studies, clevidipine caused increases in maternal and fetal mortality and length of gestation. CLEVIPREX should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

There was decreased fetal survival when pregnant rats and rabbits were treated with clevidipine during organogenesis at doses 0.7 times (on a body surface area basis) the maximum recommended human dose (MRHD) in rats and 2 times the MRHD in rabbits.

In pregnant rats dosed with clevidipine during late gestation and lactation, there were dose-related increases in maternal mortality, length of gestation and prolonged parturition at doses greater than or equal to 1/6 of the MRHD based on body surface area. When offspring of these dams were mated, they had a conception rate lower than that of controls. Clevidipine has been shown to cross the placenta in rats [see Nonclinical Toxicology (13.3)].

Labor and Delivery: CLEVIPREX in the labor and delivery setting has not been established as safe and effective. Other calcium channel blockers suppress uterine contractions in humans. Pregnant rats treated with clevidipine during late gestation had an increased rate of prolonged parturition.

Nursing Mothers: It is not known whether clevidipine is excreted in human milk. Because many drugs are excreted in human milk, consider possible infant exposure when CLEVIPREX is administered to a nursing woman.

Pediatric Use: The safety and effectiveness of CLEVIPREX in children under 18 years of age have not been established.

Geriatric Use: Of the 1406 subjects (1307 with hypertension) treated with CLEVIPREX in clinical studies, 620 were ≥65 years of age and 232 were ≥75 years of age. No overall differences in safety or effectiveness were observed between these and younger patients. Other reported clinical experience has not identified differences in responses between the elderly and younger patients. In general, for an elderly patient doses should be titrated cautiously, usually starting at the low end of the dosing range, reflecting the greater frequency of decreased hepatic, renal or cardiac function, and of concomitant disease or other drug therapy.

OVERDOSAGE

There has been no experience of overdosage in human clinical trials. In clinical trials, doses of CLEVIPREX up to 106 mg/hour or 1153 mg maximum total dose was administered. The expected major effects of overdose would be hypotension and reflex tachycardia.

Discontinuation of CLEVIPREX leads to a reduction in antihypertensive effects within 5 to 15 minutes [see Clinical Pharmacology (12.2)]. In case of suspected overdosage, CLEVIPREX should be discontinued immediately and the patient’s blood pressure should be supported.

Please see Full Prescribing Information at www.clevprex.com.

References:


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BY KENNETH TOTZ, DO, JD, FACEP

On March 27, 2018, the Supreme Court of Pennsylvania affirmed the Pennsylvania Peer Review Protection Act (PRPA), and held that the emergency medicine contracting group, UPMC Emergency Medicine, Inc. (ERMI), did not qualify as a “professional health care provider” under the PRPA and was thus not given the evidentiary privilege afforded by the state’s peer-review statute.

The court ruling stems from the medical malpractice action of Reginelli v Boggs. The clinical story is a seemingly familiar one in emergency medicine, whereby a patient presents to the emergency department with epigastric discomfort, returning later with a misdiagnosed myocardial infarction. The issues soon turned from the basic elements of negligence to the discoverability of peer-review material. During a deposition, it became apparent that Brenda Walther, MD, the ED director, maintained a “performance file” on the physicians in the group, including the defendant, Marcellus Boggs, MD. The plaintiffs filed a motion to compel production of the performance file, which was allowed by virtue that “ERMI, as an independent contractor, is not an entity enumerated in the PRPA as being protected by peer review privilege.”

The court suggested that peer-review protections would have been afforded to ERMI and Dr. Boggs had Dr. Walther been a designated and documented member of the hospital’s peer-review committee. Alternatively, the court asserted that the evidentiary privilege could have been afforded had the staffing hospital established a formal written affiliation with ERMI as an outside entity to conduct its peer-review processes for the emergency department. Although the court record alluded to an existing quasi-contractual peer-review arrangement at the trial phase of the proceedings, the agreement was not provided in a timely fashion to the court and thus became inadmissible on appellate review.

While it is a tough pill to swallow, the ruling in this case should prompt the majority of emergency medicine groups to reassess the protections their peer-review processes have in the context of the statutes in the states where they operate.

Lessons Learned
1. Review your state’s peer-review statute to ensure strict compliance.
2. Confirm written affiliation between the hospital and the ED group/director to specifically perform the peer-review process.
3. Consider making the ED director or other group designee a member of the hospital peer-review process/committee.
4. Identify all peer-review personnel, materials, and processes as affirmatively falling within the peer-review statutory authority.
5. Periodically update the hospital/group agreement using language commensurate with that of the protecting peer-review statute.
6. Mark all peer-review correspondences in any form (written, oral, electronic, etc.) as protected peer-review material.

Reference
1. Pa Stat § 425.2.

DR. TOTZ is facility medical director at First Choice Emergency Room at Adeptus Health in Texas.

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Peers Review Paranoia

Pennsylvania case weakens peer review protections

ERMI did not “qualify as a health care provider under the PRPA, because it is not approved, licensed, or otherwise regulated to practice or operate in the health care field in Pennsylvania, and it did not become one because one of its employees (Walther) conducted an evaluation of another of its employees (Boggs).” The court suggested that peer-review protections would have been afforded to ERMI and Dr. Boggs had Dr. Walther been a designated and documented member of the hospital’s peer-review committee. Alternatively, the court asserted that the evidentiary privilege could have been afforded had the staffing hospital established a formal written affiliation with ERMI as an outside entity to conduct its peer-review processes for the emergency department. Although the court record alluded to an existing quasi-contractual peer-review arrangement at the trial phase of the proceedings, the agreement was not provided in a timely fashion to the court and thus became inadmissible on appellate review.

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Caring for ED Sexual Assault Victims

Common federal and state law requirements

by RALPH J. RIVIELLO, MD, MS, FACEP; and HEATHER V. ROZZI, MD, FACEP

The Case
You are working a Saturday night when a 21-year-old female comes in for alcohol intoxication. Her vital signs are stable, and she appears visibly intoxicated. She appears withdrawn and tearful, and shortly after evaluation, she confides in you that she was raped at a college party. She doesn’t want the police called but does want a rape kit collected. She continues to insist on no police involvement. You are unsure about collecting a kit without law enforcement involvement and do not have a sexual assault nurse examiner (SANE) team at your hospital. What do you do?

It’s Not Unusual
Sexual assault is a common complaint in the emergency department. A 2010 study showed that one in five women and one in 71 men will experience rape, which the authors defined as “forced attempted or completed penetration.”1 The rates of sexual violence other than rape, including sexual coercion, unwanted sexual contact, and non-contact unwanted sexual experiences, are experienced by one in two women and one in five men sometime during their life. And these national statistics do not adequately reflect the experience of populations in which rape and sexual violence occur in much higher numbers.

How patients are cared for in the emergency department can have profound effects on their physical, psychological, and emotional healing. Well-functioning, victim-centered programs utilizing SANE teams have been perceived as helpful, caring, compassionate, and supportive by sexual assault victims. Survivors are left feeling supported, believed, heard, respected, safe, reassured, in control, informed, and well cared for post-assault.2–4 In contrast, a negative ED experience following sexual assault produces the opposite effects.

What Does the Law Require?
Emergency physicians are often held to federal and/or state laws when caring for victims of crime, including sexual assault survivors. Several of these laws come into play in the above case as well as in the overall care of intoxicated sexual assault patients.

First, many state statutes mandate law enforcement notification when patients who are victims of certain crimes present to the emergency department. These include gunshot wounds, stab wounds, battery, assaults with weapons, child abuse, elder abuse, domestic abuse, and sexual assault. For these crimes, ED personnel are classified as mandated reporters.

But just because a state law mandates reporting does not mean the patient has to speak to police. It is well within the rights of the patient to refuse to cooperate with law enforcement. Police understand this discrepancy between the law and the victim’s autonomy. Police officers may also be able to better explain to the patient all of their options regarding sexual assault reporting and investigation as well as the benefits and potential downsides of reporting. Sometimes officers develop a rapport with patients and may convince them to report the crime.

States may have victims’ rights laws, which provide protections and rights to crime victims. Some states require the patient be given the option of having a rape crisis advocate present during their examination. The hospital must inform the patient of this right and notify the advocate for the patient. In most jurisdictions, communication between the victim and police can be difficult. Some patients may want to cooperate with law enforcement, while others may not.

Emergency departments should have clear and concise policies for managing unconscious, incapacitated, or intoxicated victims.

CONTINUED on page 31
ED Inefficiency Drives Poor Quality

The data shows how we can reduce the number of patients who leave before completing treatment

by JAMES J. AUGUSTINE, MD, FACEP

Emergency departments face significant challenges in patient management and concomitant increases in regulatory and reporting requirements. Some regulatory requirements are matched to transparency mandates for items that will be reported to the public, such as the Hospital Compare measures from the Centers for Medicare and Medicaid Services. And notably, the issues of timely care, flow through the emergency department, and safety incidents in the ED waiting room have activated regulators, The Joint Commission, and the general media.

ED leaders have tackled the critical imperative to reduce the number of people who enter the emergency department but leave prematurely. The Emergency Department Benchmarking Alliance (EDBA) has worked with other organizations to develop definitions that provide consistency in reporting. With the input of ED leaders who recognize the temptation to cheat on this reporting element, it was necessary to develop an inclusive term that would incorporate all patients who leave before they are supposed to and would not provide gaps for patient encounters to be missed in the ED reporting systems.

The participants in the third Performance Measures and Benchmarking Summit have published the results of the sessions in which ED definitions were developed, with incomplete ED patient encounters termed “left before treatment complete” (LBTC). This single definition provides the most complete accounting for all patients who leave the emergency department before they are supposed to, and it includes patients who leave before or after the EMTALA-mandated medical screening examination, those who leave against medical advice (AMA), and those who elope (ie, simply walk out without speaking to anyone).

The EDBA uses this single statistic to compile the annual number of patients who are recognized by the emergency department but leave prior to completion of treatment. This provides the most complete accounting for all incomplete encounters, and it delivers a great comparison statistic.

The Stats

A number of studies have related ED walkaway rates to flow rates, and across a 14-year time frame, the EDBA data have found walkaway rates relate to volume, ED type, time to first contact with a licensed provider, and overall ED flow. The LBTC rate has trended lower across EDBA hospitals over the prior 14 years (see Figure 1).

For the past eight years, the EDBA study has evaluated various time intervals and their potential contribution to the LBTC rate. Table 1 shows the eight-year data on LBTC and median door-to-provider and door-to-decision times for admitted patients. Despite a consistent drop in median door-to-provider times, the LBTC rate has gone up, possibly because during those eight years the door-to-decision time has crept higher. That time is generally under the control of the emergency physician, and it offers an opportunity to focus on efficiencies that will move critical information to emergency physicians so they can make a quality decision.

In 2017, the LBTC rate was 2.7 percent across all emergency departments, but it varied significantly across the various cohorts that represent different types of emergency departments and different ED volumes. The LBTC rate by cohort appears in Figure 2. Table 2 shows the EDBA time intervals and LBTC rate. That data suggests a correlation between processing all patients, the door-to-decision time for admitted patients, and overall walkaway rates. And it clearly reflects the stresses on ED operations when volume and patient acuity increase.

The overall complexity of processing patients who ultimately are admitted is reflected in the amount of time it takes across all cohorts to arrive at the decision to admit. Even in the smallest emergency departments, the median time to that decision was 164 minutes. The other ED cohorts reported a time range of roughly 180–228 minutes, except for the group of emergency departments in the 100,000–120,000 volume range, where the time to decision averages 273 minutes. Long time intervals to decision to admit or discharge are associated with higher LBTC rates.

Conclusions

We can see emergency physicians have an opportunity to reduce walkaway rates by improving decision timing. Many emergency departments have reduced door-to-provider time, but we also need a timely effort to perform diagnostic testing and get the results to the responsible emergency physician, initiate any necessary treatment and evaluate the results, conduct necessary conversations with primary care or other consulting physicians, and complete a review of pertinent medical

FIGURE 1: PERCENTAGE OF PATIENTS WHO LBTC, 2004–2017

TABLE 1: DOOR-TO-PROVIDER, LENGTH-OF-STAY, AND LBTC TRENDS, 2010–2017

<table>
<thead>
<tr>
<th>YEAR</th>
<th>MEDIAN DOOR-TO-PROVIDER TIME (MINUTES)</th>
<th>MEDIAN LENGTH OF STAY, DOOR-TO-DECISION FOR ADMITTED PATIENTS (MINUTES)</th>
<th>LBTC</th>
</tr>
</thead>
<tbody>
<tr>
<td>2010</td>
<td>33</td>
<td>151</td>
<td>1.9%</td>
</tr>
<tr>
<td>2011</td>
<td>30</td>
<td>185</td>
<td>2.0%</td>
</tr>
<tr>
<td>2012</td>
<td>32</td>
<td>168</td>
<td>2.2%</td>
</tr>
<tr>
<td>2013</td>
<td>30</td>
<td>176</td>
<td>2.3%</td>
</tr>
<tr>
<td>2014</td>
<td>28</td>
<td>186</td>
<td>2.2%</td>
</tr>
<tr>
<td>2015</td>
<td>28</td>
<td>189</td>
<td>2.6%</td>
</tr>
<tr>
<td>2016</td>
<td>27</td>
<td>185</td>
<td>2.7%</td>
</tr>
<tr>
<td>2017</td>
<td>25</td>
<td>186</td>
<td>2.7%</td>
</tr>
</tbody>
</table>
advocate are privileged. Rape crisis center advocates understand the many ways sexual assault can impact a person and their family and are there to provide support and information so that the survivor may make informed, critical decisions. Advocates may also intervene or act on behalf of the survivor and assist with navigating the processes within the medical, law enforcement, and court systems.

Some states may have emergency contraception (EC) laws that require hospitals to provide emergency contraception to sexual assault survivors. If EC is not provided on site, such laws require the hospital have alternative plans for patients to obtain the medication. Some states require hospitals to register with the state and provide patient notification of plans for patients to obtain the medication. All of those items combined will give the emergency physician an opportunity to make a quality disposition decision and initiate transition of care.

Decision to admit by an emergency physician begins a cascade of events that should end in a timely movement of the patient to the inpatient unit. A boarding time discussion took place in a 2016 ACEP Now article, but unfortunately the EDBA boarding time numbers in 2016 and 2017 remained consistent at about 117 minutes.

Despite ED volume and acuity increases that challenge ED providers, improved operations have been evident in many emergency departments. However, the LBTC rates relate to ED efficiency and processing, and improving those areas requires coordinated utilization of staff, equipment, processes, and documentation.

Bottom line: Opportunities to improve patient flow do exist, and improved patient flow will reduce the rate of incomplete patient encounters.

References

TABLE 2: 2017 EMERGENCY DEPARTMENT BENCHMARKING ALLIANCE DATA SURVEY RESULTS

<table>
<thead>
<tr>
<th>ED TYPE</th>
<th>MEDIAN LENGTH OF STAY, ALL ED PATIENTS (MINUTES)</th>
<th>MEDIAN DOOR-TO-DECISION TIME, ADMITTED PATIENTS (MINUTES)</th>
<th>LBTC</th>
</tr>
</thead>
<tbody>
<tr>
<td>All EDs (N=1,717)</td>
<td>181</td>
<td>186</td>
<td>2.7%</td>
</tr>
<tr>
<td>Under 20K volume</td>
<td>134</td>
<td>164</td>
<td>1.6%</td>
</tr>
<tr>
<td>20–40K</td>
<td>164</td>
<td>173</td>
<td>2.2%</td>
</tr>
<tr>
<td>40–60K</td>
<td>200</td>
<td>209</td>
<td>3.1%</td>
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<tr>
<td>60–80K</td>
<td>222</td>
<td>213</td>
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<tr>
<td>80–100K</td>
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<td>229</td>
<td>4.3%</td>
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<tr>
<td>100–120K</td>
<td>245</td>
<td>273</td>
<td>4.8%</td>
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<tr>
<td>Over 120K volume</td>
<td>228</td>
<td>228</td>
<td>4.9%</td>
</tr>
<tr>
<td>Pediatric EDs</td>
<td>149</td>
<td>187</td>
<td>1.6%</td>
</tr>
<tr>
<td>Adult EDs</td>
<td>247</td>
<td>225</td>
<td>4.2%</td>
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CONTINUED on page 33
5-Step Approach to the Agitated Patient

Stick to your plan to “keep it chill”

by ANTON HELMAN, MD, CCFP(EM), FCFP

We’re all familiar with the spike in cortisol levels we feel when faced with agitated patients in the emergency department. That’s not only because of our hard-wired fight-or-flight response but also because we know that these patients are high-risk to themselves, us, and our ED staff. Agitation or agitated delirium is not a diagnosis but rather a cardinal presentation. Pathology, such as psychiatric, medical, traumatic, and toxicological diagnoses, is lurking beneath; it is imperative that we safely and rapidly calm these patients so we can assess and manage their underlying diagnoses. Here is a five-step approach to managing agitated patients.

Step 1: Categorizing Agitation as Mild, Moderate, or Severe

It is helpful to categorize the level of agitation to better target sedation. The mildly agitated patient is able to converse and is cooperative without being disruptive, while the moderately agitated patient is disruptive to your emergency department without imminent danger to themselves or your ED staff. However, the severely agitated patient is imminently dangerous to all. This last category includes patients with excited delirium syndrome, a true emergency with a very high mortality rate. Excited delirium syndrome has several distinctive features that include unusual superhuman strength, imperviousness to pain, severe metabolic acidosis, inability to maintain attention, and hyperthermia.

Step 2a: Nonpharmacologic De-escalation for Mildly or Moderately Agitated Patients

Verbal de-escalation is often effective in the mildly to moderately agitated patient, but it requires a calm and deliberate approach. Some key elements of effective de-escalation include environmental awareness and self-awareness, such as delegating one person to speak to the agitated patient, ensuring a quiet room, modulation of your own emotional and physiologic responses to remain calm, avoiding clenched fists, and having your hands visible.

The SAVE mnemonic outlines scripted responses that may be helpful when faced with a violent patient:

- **Support:** “Let’s work together…”
- **Acknowledge:** “I see this has been hard for you.”
- **Validate:** “I’d probably be reacting the same way if I was in your shoes.”
- **Emotion naming:** “You seem upset.”

Step 2b: “Code White” for Moderately and Severely Agitated Patients

Consideration should be given to calling a “code white” for the patient who is an immediate physical threat to you or your staff. A common pitfall is to call a code white as a threat to an uncooperative patient, which can inadvertently increase agitation. Consider calling a concealed code white, directly to security, rather than using an overhead page for the moderately agitated patient who is not posing an imminent danger.

Step 3: Safe and Effective Physical Restraints

There is ongoing debate as to whether physical restraints should be used at all in the management of the agitated patient in the emergency department. If you are going to use physical restraints, the goal should be to use them only as a last resort as a bridge to chemical restraint, which should take no longer than five to 15 minutes with appropriate dosing. Prolonged use of physical restraints may result in active resistance of restraints, which may lead to electrolyte abnormalities or dysrhythmias and put the patient at further risk for rhabdomyolysis.

One option is to avoid the use of physical restraints and instead hold the patient down by security for the few minutes it takes for the calming medications to take effect. The other option is to place the physical restraints on the patient, immediately administer intramuscular (IM) calming medications, and release the restraints as soon as the patient is calm. Physical restraints should always be followed by immediate sedation.

When used properly, physical restraints can be quite safe. However, improper use can be lethal. In one study, 26 deaths were presumed to be the direct result of improper physical restraints. Avoid covering the agitated patient’s mouth and/or nose with a gloved hand. This can lead to asphyxia, metabolic acidosis, and death. Use an oxygen mask to prevent the patient from spitting on staff. This may also serve to improve oxygenation. With the patient in the supine position with about 30 degrees head elevation, use four-point restraints tied to the bed frame (rather than the rails), with one arm above the head and the other below the waist.

Step 4: Chemical Restraint or Sedation

The goal of calming medications is to enable rapid stabilization of the acutely agitated patient and to enable the expeditious search for potential life-threatening diagnoses. The choice of route depends on how agitated your patient is. For cooperative mildly agitated patients, offer oral or sublingual medications first. For uncooperative moderately and severely agitated patients, the safest option is to start with IM medications.

Calming medication options include ketamine, benzodiazepines, and antipsychotics. Whenever possible, tailor the therapy to the underlying diagnosis (eg, psychotic psychiatric disorder, alcohol withdrawal, drug intoxication, etc.). While the evidence for one regimen over another is lacking, current ev ev-idence-based recommendations are summarized in Table 1.

<table>
<thead>
<tr>
<th>LEVEL OF AGITATION</th>
<th>FIRST-CHOICE DRUG, DOSE, ROUTE</th>
<th>ALTERNATIVE OR ADJUNCT DRUG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Lorazepam 1–2 mg sublingual</td>
<td>Oral antipsychotic that has previously been effective for that patient</td>
</tr>
<tr>
<td>Moderate</td>
<td>Midazolam 2–5 mg IM</td>
<td>Haloperidol 5–10 mg IM</td>
</tr>
<tr>
<td>Severe</td>
<td>Ketamine 5 mg/kg IM</td>
<td>Haloperidol 10 mg IM and midazolam 10 mg IM</td>
</tr>
</tbody>
</table>

Dr. Helman is an emergency physician at North York General Hospital in Toronto. He is an assistant professor at the University of Toronto, Division of Emergency Medicine, and the education innovation lead at the Schwartz/Reisman Emergency Medicine Institute. He is the founder and host of the Emergency Medicine Cases podcast and website (www.emergencymedicinecases.com).

CONTINUED on page 34
The Emergency Group, Inc.
Honolulu, Hawaii

The Emergency Group, Inc. (TEG) is a growing, independent, democratic group that has been providing emergency services at The Queen’s Medical Center (QMC) in Honolulu, Hawaii since 1973. QMC is the largest and only Level 1 Trauma Center in the state and cares for more than 65,000 ED patients per year. QMC opened an additional medical center in the community of West Oahu in 2014, which currently sees 60,000 ED patients annually.

Due to the vastly growing community in the West Oahu area, TEG is actively recruiting for EM Physicians BC/BE, EM physicians with Pediatric Fellowship who are BE/BC and an Ultrasound Director. Physicians will be credentialed in both facilities and will work the majority of the shifts at the West Oahu facility in Ewa Beach, Hawaii.

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EMERGENCY MEDICINE FACULTY


The Department of Emergency Medicine at East Carolina University Brody School of Medicine seeks BC/BE emergency physicians and pediatric emergency physicians for tenure or clinical track positions at the rank of assistant professor or above, depending on qualifications. We continue to expand our faculty to meet the clinical needs of our patients and the educational needs of our learners. We envision further program development in clinical education, emergency ultrasound, EM-critical care, pediatric EM, and clinical research. Our current faculty possesses diverse interests and expertise leading to extensive state and national level involvement. The emergency medicine residency includes 12 ED and 2 ICU rotations per year. We treat more than 130,000 patients per year at our level one ED at Vidant Medical Center. VMCH is a 90-bed Level 1 trauma center and regional referral center for cardiac, stroke, and pediatric care. Our tertiary care catchment area includes more than 1.5 million people in eastern North Carolina. Additionally, we provide clinical coverage at two community hospitals within our health system. We are responsible for medical direction of East Carolina’s integrated mobile critical care and air medical service, and multiple county EMS systems. Our pediatricians ED is the largest ED in 2012 and serves approximately 25,000 children per year. Greenville, NC is a university community offering a pleasant lifestyle and excellent cultural and recreational opportunities. Beautiful North Carolina beaches are nearby.

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In alcohol-intoxicated patients, beware of respiratory depression with benzodiazepines and place them on a cardiac monitor, ideally with end-tidal CO2 monitoring for early detection of respiratory depression.

Haloperidol should be considered an adjunct to benzodiazepines for moderate and severe agitation and may be appropriate as monotherapy in moderately agitated intoxicated patients who cannot be placed on a monitor when resources are limited. Be aware that haloperidol has a longer half-life than midazolam, which can result in the patient staying in your emergency department for much longer than would be necessary otherwise. Although haloperidol prolongs the QTc, this effect is very unlikely to be clinically consequential at the doses typically used for emergency agitation. Nonetheless, caution is advised in patients who are already taking multiple QTc-prolonging agents. Consider obtaining a baseline ECG first in these higher-risk patients.

Ketamine is an N-methyl-D-aspartate receptor antagonist, providing rapid sedation and analgesia. Of the available options, its time to sedation is the fastest, usually less than five minutes with appropriate dosing. Current evidence for the effectiveness and safety of ketamine in calming the severely agitated patient is promising but not definitive.

**Step 5: Treating Immediate Life Threats and Pursuing Underlying Diagnosis as Soon as Calming Medications Take Effect**

For the mildly to moderately agitated patient, corroborating the history with a head-to-toe physical exam with the patient completely disrobed is essential. Consider a broad differential diagnosis, including space-occupying central nervous system lesions as well as toxicological, psychiatric, traumatic, and metabolic causes.

For the severely agitated patient, it is important to be organized in your approach, which can be divided into the first few minutes, the next few minutes, and the next hour.

In the first few minutes, place the patient in a resuscitation room and apply cardiorespiratory monitoring, capnography, and oximetry. Initiate one to two large-bore peripheral IVs and assess for and start to treat the four Hs: hypoxia, hyperthermia, hypovolemia, and hypoglycemia. If a definitive airway is required, consider delayed sequence rather than rapid
sequence intubation, including hyperventilation and sodium bicarbonate in the peri-intubation period as the patient may be severely acidotic. In the next few minutes, obtain electrolytes and blood gas and treat for hypokalemia and acidemia. Consider a head CT scan. In the next hour, consider primary diagnoses such as sepsis, neuroleptic malignant syndrome, thyrotoxicosis, and meningitis/encephalitis. In addition, it is important to assess for consequences of agitation (eg, rhabdomyolysis and traumatic injuries).

Having a simple approach to the agitated patient in the emergency department will not only buffer your cortisol levels but also give you the tools you need to safely and efficiently uncover and manage a life-threatening diagnosis.

Special thanks to Dr. Margaret Thompson and Dr. Reuben Strayer, the guest experts on the podcast from which this column was inspired.

References
DOCUMENTING WOUND DEBRIDEMENT

by HAMILTON LEMPERT, MD, FACEP, CEDC

Question: How do I code for a wound debridement?

Answer: Current Procedural Terminology (CPT) provides several different coding options for reporting wound debridement service, depending on the type of debridement performed. The most common codes used in the emergency department are 97597 (0.68 relative value units [RVU], $24 for Medicare) for debridement involving the epidermis and/or dermis and 11042 (1.78 RVU, $64 for Medicare) for debridement of subcutaneous tissue. The deepest layer of tissue debrided determines which code to use. Each of these codes is for 20 cm² or less, and there are additional codes to use when more than 20 cm² is debrided, 97598 and 11045, respectively. Ideal documentation for debridement should include the depth of tissue (layers) debrided as well as the total surface area of the wound. There are also debridement codes for muscle or fascia (11043) and bone (11044) when performed by the emergency department provider.

Brought to you by the ACEPT Coding and Nomenclature Committee.

DR. LEMPERT is chief medical officer, coding policy, at TeamHealth, based in Knoxville, Tennessee.

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