**Using Twitter Fame to Advocate for EM**

An interview with social media and academic rock star Dr. Esther Choo

by Jeremy Samuel Faust, MD, MS

Esther Choo, MD, MPH (@choo_ek), is associate professor at the Center for Policy and Research in Emergency Medicine at Oregon Health & Science University in Portland. Last year, after the neo-Nazi demonstrations in Charlottesville, Virginia, she posted a series of tweets describing the unabashed racism she has experienced as a practicing Asian-American emergency physician. When that Twitter thread was retweeted by Chelsea Clinton, it went viral and was retweeted more than 25,500 times and seen by more than 4.5 million people, giving her, and her

The Contested Admission

SEE PAGE 26

**Interim Presidential Report**

ACEP President Dr. Paul Kivela is focused on communication and collaboration

Part of ACEP’s power as an advocate for emergency medicine comes from the passion and innovation of its leaders. Recently, Paul Kivela, MD, MBA, FACEP, who took over as ACEP President in October 2017, shared some of his goals for his presidential year and a few of the surprising challenges so far with ACEP Now Medical Editor-in-Chief Kevin Klauer, DO, EJD, FACEP. Here are some highlights from their conversation.

**Continued on page 13**

**Em Cases**

Infants with Congenital Heart Disease

SEE PAGE 22
ARISTOTLE (N=5502), ENGAGE-AF (N=11,200), and RE-LY (N=5882). 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 non-steroidal anti-inflammatory drugs (NSAIDs), platelet inhibitors, other anticoagulants, see Drug Interactions
- A history of traumatic or repeated epidural or spinal punctures
- A history of spinal deformity or spinal surgery
- Optimal timing between the administration of XARELTO® 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 for thromboprophylaxis.

INDICATIONS

XARELTO® is indicated to reduce the risk of stroke and systemic embolism in patients with nonvalvular atrial fibrillation (AF). There are limited data on the relative effectiveness of XARELTO® and warfarin in reducing the risk of stroke and systemic embolism when warfarin therapy is well controlled.

XARELTO® is indicated for the treatment of deep vein thrombosis (DVT). XARELTO® is indicated for the treatment of pulmonary embolism (PE). XARELTO® is indicated for the reduction in the risk of recurrence of DVT and/or PE in patients at continued risk for recurrent DVT and/or PE after completion of initial treatment lasting at least 6 months.

IMPORTANT SAFETY INFORMATION

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

A. Premature discontinuation of XARELTO® increases the risk of thrombotic events

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

B. Spinal/epidural hematoma

Epidural or spinal hematomas have occurred in patients treated with XARELTO® 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.
IMPORTANT SAFETY INFORMATION (cont’d)

CONTRAINDICATIONS
- Active pathological bleeding
- Severe hypersensitivity reaction to XARELTO® (eg, anaphylactic reactions)

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

- Risk of Bleeding: XARELTO® increases the risk of bleeding and can cause serious or fatal bleeding. Promptly evaluate any signs or symptoms of blood loss and consider the need for blood replacement. Discontinue XARELTO® in patients with active pathological hemorrhage.
- A specific antidote for rivaroxaban is not available. Because of high plasma protein binding, rivasoxaban is not expected to be dialyzable.
- Concomitant use of other drugs that impair hemostasis increases the risk of bleeding. These include aspirin, P2Y12 platelet inhibitors, other antithrombotic agents, fibrinolytic therapy, NSAIDs, selective serotonin reuptake inhibitors (SSRIs), and serotonin nonselective reuptake inhibitors (SNRIs).

- Spinal/Epidural Anesthesia or Puncture: When neuraxial anesthesia (spinal/epidural anesthesia) or spinal puncture is employed, patients treated with anticoagulants for prevention of thromboembolic complications are at risk for developing an epidural or spinal hematoma, which can result in long-term or permanent paralysis. To reduce the potential risk of bleeding associated with the concomitant use of XARELTO® and epidural or spinal anesthesia/analgesia or spinal puncture, consider the pharmacokinetic profile of XARELTO®. Placement or removal of an epidural catheter or lumbar puncture is best performed when the anticoagulant effect of XARELTO® is low; however, the exact timing to reach a sufficiently low anticoagulant effect in each patient is not known. An indwelling epidural or intrathecal catheter should not be removed before at least 2 half-lives have elapsed (ie, 18 hours in young patients aged 20 to 45 years and 26 hours in elderly patients aged 60 to 76 years), after the last administration of XARELTO®. The next XARELTO® dose should not be administered earlier than 6 hours after the removal of the catheter. If traumatic puncture occurs, delay the administration of XARELTO® for 24 hours. Should the physician decide to administer anticoagulation in the context of epidural or spinal anesthesia/analgesia or lumbar puncture, monitor frequently to detect any signs or symptoms of neurological impairment, such as midline back pain, sensory and motor deficits (numbness, tingling, or weakness in lower limbs), or bowel and/or bladder dysfunction. Instruct patients to immediately report if they experience any of the above signs or symptoms. If signs or symptoms of spinal hematoma are suspected, initiate urgent diagnostic testing and treatment including consideration for spinal cord decompression even though such treatment may not prevent or reverse neurological sequelae.

- Use in Patients With Renal Impairment:
  - Nonvalvular Atrial Fibrillation: Periodically assess renal function as clinically indicated (ie, more frequently in situations in which renal function may decline) and adjust therapy accordingly. Consider dose adjustment or discontinuation of XARELTO® in patients who develop acute renal failure while on XARELTO®.
  - Treatment of Deep Venous Thrombosis (DVT), Pulmonary Embolism (PE), and Reduction in the Risk of Recurrence of DVT and of PE: Avoid the use of XARELTO® in patients with CrCl <30 mL/min due to an expected increase in rivaoxaban exposure and pharmacodynamic effects in this patient population.
  - Prophylaxis of Deep Venous Thrombosis Following Hip or Knee Replacement Surgery: Avoid the use of XARELTO® in patients with CrCl <30 mL/min due to an expected increase in rivaoxaban exposure and pharmacodynamic effects in this patient population. Observe closely and promptly evaluate any signs or symptoms of blood loss in patients with CrCl 30 to 50 mL/min. Patients who develop acute renal failure while on XARELTO® should discontinue the treatment.

- Use in Patients With Hepatic Impairment: No clinical data are available for patients with severe hepatic impairment. Avoid use of XARELTO® in patients with moderate (Child-Pugh B) or severe (Child-Pugh C) hepatic impairment or with any hepatic disease associated with coagulopathy, since drug exposure and bleeding risk may be increased.

- Use With P-gp and Strong CYP3A4 Inhibitors or Inducers: Avoid concomitant use of XARELTO® with known inhibitors of P-gp and strong CYP3A4 inhibitors. Avoid concomitant use of XARELTO® with drugs that are known combined P-gp and strong CYP3A4 inducers.

- Risk of Pregnancy-Related Hemorrhage: In pregnant women, XARELTO® should be used only if the potential benefit justifies the potential risk to the mother and fetus. XARELTO® dosing in pregnancy has not been studied. The anticoagulant effect of XARELTO® cannot be monitored with standard laboratory testing nor readily reversed. Promptly evaluate any signs or symptoms suggesting blood loss (eg, a drop in hemoglobin and/or hematocrit, hypotension, or fetal distress).

- Patients With Prosthetic Heart Valves: The safety and efficacy of XARELTO® have not been studied in patients with prosthetic heart valves. Therefore, use of XARELTO® is not recommended in these patients.

- Acute PE in Hemodynamically Unstable Patients/Patients Who Require Thrombolysis or Pulmonary Embolectomy: Initiation of XARELTO® is not recommended acutely as an alternative to unfractionated heparin in patients with pulmonary embolism who present with hemodynamic instability or who may receive thrombolysis or pulmonary embolectomy.

DRUG INTERACTIONS
- Combined P-gp and strong CYP3A4 inhibitors increase exposure to rivaoxaban and may increase the risk of bleeding.
- Combined P-gp and strong CYP3A4 inducers decrease exposure to rivaoxaban and may increase the risk of thromboembolic events.

- XARELTO® should not be used in patients with Ccr 15 to <80 mL/min who are receiving concomitant combined P-gp and moderate CYP3A4 inhibitors (eg, erythromycin) unless the potential benefit justifies the potential risk.

- Coadministration of enoxaparin, warfarin, aspirin, clopidogrel, and chronic NSAID use may increase the risk of bleeding.

- Avoid concurrent use of XARELTO® with other anticoagulants due to increased bleeding risk, unless benefit outweighs risk. Promptly evaluate any signs or symptoms of blood loss if patients are treated concomitantly with aspirin, other platelet aggregation inhibitors, or NSAIDs.

USE IN SPECIFIC POPULATIONS
- Pregnancy: The limited available data on XARELTO® in pregnant women are insufficient to determine the risk associated with use of XARELTO® in pregnant women. Use XARELTO® with caution in pregnant patients because of the potential for pregnancy-related hemorrhage and/or emergent delivery with an anticoagulant that is not readily reversible. The anticoagulant effect of XARELTO® cannot be reliably monitored with standard laboratory testing. Consider the benefits and risks of XARELTO® for the mother and possible risks to the fetus when prescribing XARELTO® to a pregnant woman.

- Fetal/Neonatal adverse reactions: Based on the pharmacologic activity of Factor Xa inhibitors and the potential to cross the placenta, bleeding may occur at any site in the fetus and/or neonate.

- Labor or delivery: The risk of bleeding should be balanced with the risk of thrombotic events when considering the use of XARELTO® in this setting.

- There are no adequate or well-controlled studies of XARELTO® in pregnant women, and dosing for pregnant women has not been established. Post-marketing experience is currently insufficient to determine a rivaixelaban-associated risk for major birth defects or miscarriage.

- Lactation: Rivaoxaban has been detected in human milk. There are insufficient data to determine the effects of rivaoxaban on the breastfed child or on milk production. The developmental and health benefits of breastfeeding should be considered along with the mother’s clinical need for XARELTO® and any potential adverse effects on the breastfed infant from XARELTO® or from the underlying maternal condition.

- Females and Males of Reproductive Potential: Females of reproductive potential requiring anticoagulation should discuss pregnancy planning with their physician.

- Pediatric Use: Safety and effectiveness in pediatric patients have not been established.

OVERDOSAGE
- Discontinue XARELTO® and initiate appropriate therapy if bleeding complications associated with overdosage occur. A specific antidote for rivaoxaban is not available. The use of activated charcoal to reduce absorption in case of XARELTO® overdose may be considered. Due to the high plasma protein binding, rivaoxaban is not dialyzable.

ADVERSE REACTIONS IN CLINICAL STUDIES
- The most common adverse reactions with XARELTO® were bleeding complications.

Please see accompanying Brief Summary of full Prescribing Information, including boxed WARNINGS, or visit www.XareltoHCP.com/PI.

References:
XARELTO® (rivaroxaban) tablets

**WARNING: A PREMATURE DISCONTINUATION OF XARELTO® INCREASES THE RISK OF THROMBOTIC EVENTS. (See Warnings and Precautions).**

**WARNING: SPINAL/EPIDURAL HEMATOMA**

A premature discontinuation of XARELTO® increases the risk of thrombotic events.

Premature discontinuation of any oral anticoagulant, including XARELTO®, is discontinued for a reason other than pathological drug discontinuation, increases the risk of thrombotic events.

Epidermal or spinal hematomas have occurred in patients treated with XARELTO®. The risk of spinal hematoma increases with the use of other anticoagulants, epidural or spinal anesthesia, and epidural or spinal puncture.

• Indwelling epidural or intrathecal catheters should not be removed immediately postprocedural observation and monitoring, and the patient monitored for 48 hours following the procedure whenever possible.

• A second anticoagulant should be considered for a patient taking XARELTO® who is receiving neuraxial anesthesia or undergoing neuraxial procedures.

• Indwelling epidural or intrathecal catheters should be removed as soon as is feasible after the patient has been observed for the appropriate length of time (i.e., 2 half-lives of 18 hours in young patients aged 20 to 80 years).

• Indwelling epidural or intrathecal catheters should not be removed before at least 2 half-lives have elapsed (i.e., 18 hours in young patients aged 20 to 80 years).

The use of XARELTO® and neuraxial anesthesia or neuraxial procedures increases the risk of developing an epidural or spinal hematoma which can result in long-term or permanent paralysis. Consider these risks when scheduling patients treated with XARELTO® who are receiving neuraxial anesthesia or undergoing neuraxial procedures.

• If a patient who is on XARELTO® and who has developed an epidural or spinal hematoma, discontinue the drug immediately and manage the patient according to standard medical practice.

• Always perform imaging of the spine when there is clinical suspicion of a hematoma.

**INDICATIONS AND USAGE**

Reduction of Risk of Stroke and Systemic Embolism in Nonvalvular Atrial Fibrillation: XARELTO® is indicated for long-term treatment of participants with nonvalvular atrial fibrillation (NVAF) who are at increased risk of stroke and systemic embolism in patients with nonvalvular atrial fibrillation.
Table 4: Bleeding Events in Patients Undergoing Hip or Knee Replacement Surgery (RECORD 1-3) [237] XARELTO® (rivaroxaban) tablets

<table>
<thead>
<tr>
<th>Event</th>
<th>XARELTO 20 mg</th>
<th>Enoxaparin</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major bleeding</td>
<td>8 (1.0%)</td>
<td>4 (0.6%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Any bleeding event</td>
<td>105 (14.4%)</td>
<td>105 (14.4%)</td>
<td>1.000</td>
</tr>
</tbody>
</table>

Table 5: Other Adverse Drug Reactions* Reported by ≥1% of XARELTO-Treated Patients in EINSTEIN-DVT and EINSTEIN PE Studies

<table>
<thead>
<tr>
<th>Body System</th>
<th>XARELTO 10 mg</th>
<th>Enoxaparin/VKA</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allergic (rare)</td>
<td>1 (0.03%)</td>
<td>0 (0.0%)</td>
<td>1.000</td>
</tr>
</tbody>
</table>

Table 6: Other Adverse Drug Reactions* Reported by ≥1% of XARELTO-Treated Patients in RECORD 1-3 Studies

<table>
<thead>
<tr>
<th>Body System</th>
<th>XARELTO 10 mg</th>
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<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allergic (rare)</td>
<td>1 (0.01%)</td>
<td>0 (0.0%)</td>
<td>1.000</td>
</tr>
</tbody>
</table>

[237] XARELTO® (rivaroxaban) tablets
LEGISLATIVE UPDATES WITH CAPITAL MINUTE

Want a rapid rundown of what’s happening on Capitol Hill? Don’t miss this Capital Minute, where ACEP reports on testifying before Congress on opioids, new MedPAC recommendations on freestanding emergency departments, Affordable Care Act exchange information, the government-proposed conscience rule, and the very latest on #LAC18. Visit https://bit.ly/2F3CxGP.

ACEP recently led the development and draft of two emergency medicine–focused bills aimed at addressing the growing opioid epidemic. ACEP Board member Jon Mark Hirshon, MD, FACEP, participated and shared with the subcommittee a number of regulatory burdens facing emergency physicians and ACEP’s recommended solutions.

Get Accredited for Geriatric Care

ACEP has launched the Geriatric Emergency Department Accreditation Program (GEDA). GEDA recognizes that one-size ED care does not fit all patients. Older people in the emergency department have presentations, needs, dispositions, and outcomes that are specific to their age group. A geriatric emergency department may be either a separate space designated for older adults or, more likely, will integrate best practices for older adults into ED operations. Learn more about the program at www.acep.org/GEDAHome.

Representing ACEP at the National Disaster Life Support Foundation

After careful consideration of several very qualified and talented candidates, ACEP has chosen Gerald Beltran, DO, FACEP, to be the ACEP representative to the National Disaster Life Support Foundation Board of Directors. The not-for-profit foundation is dedicated to emergency medical preparedness. It is governed by a board of directors chosen by a qualifications committee. ACEP has chosen Gerald Beltran, DO, FACEP, to be the ACEP representative to the National Disaster Life Support Foundation Board of Directors.

Advocacy on the Hill and Beyond

Megan Ranney, MD, MPH, FACEP, has been named co-chair of Rhode Island’s Gun Safety Working Group by Gov. Gina M. Raimondo. Dr. Ranney is associate professor of emergency medicine at Rhode Island Hospital and the Warren Alpert Medical School of Brown University. She will lead the group with James Manni, Narragansett town manager, and that denial of emergency care or delay in providing emergency services on the basis of race, religion, sexual orientation, gender identity, ethnic background, social status, type of illness, or ability to pay is unethical.

ACEP continues its advocacy work, both with regulators and in the media, to oppose Anthem’s dangerous policy to retroactively deny coverage of emergency department visits by its policyholders that it deems “nonemergency.” ACEP’s public relations work on the issue led to a recent NBC Nightly News piece on Anthem’s actions.

ACEP submitted a response to the U.S. Department of Health and Human Services’ proposed rule enforcing so-called “conscience protections” for providers, which, as written, would allow health care providers to deny treating a patient if they had any religious or moral objections and to do so without ensuring any continuity of care or referral to another provider. ACEP strongly voiced its objection to the proposed rule in the response, noting that, both by law and by oath, emergency physicians care for all patients seeking emergency medical treatment and that denial of emergency care or delay in providing emergency services on the basis of race, religion, sexual orientation, gender identity, ethnic background, social status, type of illness, or ability to pay is unethical.

In late January when the proposed rule was first announced by the Department of Health and Human Services, ACEP and the Emergency Medicine Residents’ Association (EMRA) responded with a joint media statement.

Members in the News

Megan Ranney, MD, MPH, FACEP, has been named co-chair of Rhode Island’s Gun Safety Working Group by Gov. Gina M. Raimondo. Dr. Ranney is associate professor of emergency medicine at Rhode Island Hospital and the Warren Alpert Medical School of Brown University. She will lead the group with James Manni, Narragansett town manager, and that denial of emergency care or delay in providing emergency services on the basis of race, religion, sexual orientation, gender identity, ethnic background, social status, type of illness, or ability to pay is unethical.

The working group includes individuals from the law enforcement, mental health, not-for-profit, public policy, and education communities and is charged with developing recommendations to counteract the gun violence epidemic.
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Debunking the myths of political involvement for emergency medicine

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—Charlie Cook, editor of The Cook Report

by PETER JACOBY, MD, FACEP; AND JEANNE L. SLADE

The Changing Political Environment

Political, social, and economic changes continue to have dramatic effects on the delivery of emergency medical care in the United States. The time when we could just “practice medicine” and ignore the politics is gone.

As chairman of the National Emergency Medicine Political Action Committee (NEMPAC), ACEP’s political action committee, I am proud of the work that our Board has done to engage ACEP members in the political process. NEMPAC had one of the best fundraising years in history in 2017, despite the tumultuous political environment, raising nearly $1.29 million in contributions from ACEP members.

In my years as Chairman, I’ve seen the PAC grow in receipts and numbers of ACEP supporters. Our ACEP Board and NEMPAC Board continue to work hard to educate ACEP members about the importance of political and legislative advocacy. However, in this environment, it’s not easy.

Lately, I’ve heard a growing number of reasons from our members as to why they don’t or won’t get involved.

The choice is simple:
1. Watch from the sidelines.
2. Or stand up and make the collective voice of emergency medicine stronger to give us the opportunity to help shape legislation that is in the interests of our specialty and patients.

Why NEMPAC Should Matter to You

ACEP PACs have a significant role within the political process. In an organization with more than 37,000 members, it’s virtually impossible that everyone will share the same political viewpoints. Please try to remember that politicians from both political parties and with vastly differing ideologies have been, and continue to be, supportive of issues that affect emergency medicine.

NEMPAC must work with elected officials and political candidates to advance the interests of our specialty, regardless of personal preference or party affiliation.

Sometimes we disagree personally with some of our candidate’s positions, but their support of EM cannot be discounted. The political process works well if you continue to support NEMPAC’s efforts to advance the interests of the specialty, and in your personal life, continue to support the elected officials you feel best represent your political views. It’s important to be objective in this matter, just as you practice medicine.

Emergency physicians are results driven, but sometimes NEMPAC’s results in politics and legislative successes on Capitol Hill can be difficult to measure (See “What Have You Done for Me Lately?” for a list of legislative wins and priorities).

How Can You Get Involved With NEMPAC?

Groups whose priorities are counter to ours are coming out to support their PACs with renewed vigor. Contributing to and participating with NEMPAC is like purchasing insurance for our specialty; we all need to make it a priority.

So how can you get involved and help us drive change?
1. Give-A-Shift: Join the more than 500 physicians who contribute one of their average shifts to NEMPAC each year, and receive many benefits including opportunities to network with leaders of ACEP and attend local fundraising events on behalf of NEMPAC.
2. Talk to NEMPAC: The NEMPAC Board and staff rely on the advice and recommendations of NEMPAC donors and state chapter leaders when evaluating candidates, especially in races where there is no incumbent. Stay informed of the congressional races in your area and reach out to the NEMPAC Board and staff if you have input. All NEMPAC donors receive the NEMPAC Pulse, our quarterly newsletter filled with information about national politics, legislative initiatives and ACEP members who are making a difference through their advocacy efforts for the specialty.

Instead of burying our heads in the sand, hoping this too will pass or coming up with more “PACscuses,” we ask to work together on making the environment better for our specialty and patients.

For more information or to get involved, go to the NEMPAC website at www.acep.org/nempac.

DR. JACOBY is chairman of the NEMPAC Board of Trustees.

MS. SLADE is director of political affairs at ACEP.

The NEMPAC Process in Today’s Political Climate

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In conjunction with the 50th anniversary of the founding of ACEP, it seems appropriate to look at the 50-year evolution of the assessment and care of chest pain in the emergency department. I have not been around for all 50 years, but I have been an ACEP member since 1975 and have personally experienced the majority of the evolution of chest pain care over that time.

At an estimated 8 to 10 million visits a year, chest pain remains an everyday complaint in the emergency department and one that is associated with some significant angst on the part of clinicians. Until the recent past, missed myocardial infarction had been the leading cause of malpractice suits and even though the top position is now related to stroke care, the fear of making a mistake in the assessment of chest pain patients remains high. A recent study of closed malpractice claims by The Doctors Company (the largest physician-owned malpractice insurer) found it ranked number two on the list.

It seems the core problem with the assessment of chest pain patients, who largely turn out not to have serious disease, relates to the concept of the “acceptable miss rate.” What percentage of patients with chest pain (or an equivalent) who are discharged after a seemingly benign evaluation will actually have a major adverse cardiac outcome that may have been averted if the patient was admitted? Historically, about 2 percent of patients with an acute myocardial infarction (AMI) or sudden death were mistakenly sent home after an ED visit. Is this percentage still the case? It is hard to conceive of how we could still miss this many, given the current extraordinarily high rate of admissions for chest pain. However, the core issue is, What percentage of misses is U.S. society willing to accept, given it is impossible to get to 0 percent?

Although it is difficult to provide an exact chronology regarding the evolution of the assessment and treatment of chest pain over the last 50 years, there are some general timeframes that can be given. Henceforth, the assumption will be made that the goal of the endeavor is to exclude or make the diagnosis of ischemic chest pain and that all other diagnoses will not be considered.

1968 History and Physical Exam
By far, the emphasis at this early stage in the assessment of chest pain was the importance of the history and, to a lesser extent, the physical exam. Despite all of the advances in ED diagnostic capabilities, obtaining a careful history remains the single most important element in making the diagnosis of acute coronary syndrome (ACS).

Electrocardiograms (ECGs)
Obtaining a 12-lead ECG has been a routine part of the evaluation of potential cardiogenic chest pain. The machines were big and unwieldy, and the films were just that—sheets of radiographic film that were developed in a dark room using a variety of liquid chemicals. When the films needed to be read immediately, they were often still wet, and the term “wet reading” was commonly used for a stat interpretation.

Cardiac Enzymes
In the late 1960s, the creatinine phosphokinase (CK) enzyme was routinely measured in chest pain patients. This enzyme was found to have a variety of isoforms. Skeletal muscle expresses CK-MM (98 percent) and low levels of CK-MB (1 percent). The heart expresses CK-MM at 70 percent and CK-MB at 25 to 30 percent. A third variety is CK-BB, which is predominantly expressed in the brain and smooth muscle. The overlap between CK-MM and CK-MB allowed for multiple causes of CK elevation to be found (eg, rhabdomyolysis, muscle trauma, myocardial infarction, myositis, and myocarditis). Other causes of CK elevations included hypothyroidism, malignant hyperthermia, and neuroleptic malignant syndrome. In addition, CK elevations could occur in the myopathy associated with use of statins, which were discovered in 1976 and introduced for patient care with lovastatin in 1987. The bottom line: CK-MB elevations were not at all specific for myocardial cell damage, but they remained the standard for assessment until...
the development of the troponins. Even today, some older clinicians refer to the troponins as "enzymes" when, in fact, they are not, but the term "enzymes" was routinely used to refer to chemicals in the blood that may be elevated in the setting of cardiac cellular injury.

1975 Automated ECG Interpretation
Hewlett-Packard was among the first companies to develop the technology to measure ECG waveforms and interpret ECG readings. The machines were very large and expensive, about $5,000. In fact, they were so costly at the time that you could lease a machine just like a car ($5,000 is just what the average car cost in 1975) and make monthly payments. Now, all ECG machines have software that interprets the ECG with substantial accuracy at a much lower cost. A study by Hughes et al found that of 228 ECGs interpreted as normal by the Marquette 12SL software, only one, on over-read, was interpreted by one of two emergency physicians as requiring immediate bedding, and the patient had a normal stress test. This is not to say that there are not multiple subtle ECG patterns that are worrisome that can be missed by computerized readings. Thus, review by an experienced clinician is mandatory.

1979 Advent of Thrombolytic Therapy for STEMI-Elevation Myocardial Infarction (STEMI)
In 1978, Sol Sherry, MD, of Temple University in Philadelphia, started using streptokinase in AMI patients, and he and his colleagues began the era of "cure" versus palliation of myocardial infarction. Bed rest and he and his colleagues demonstrated that aspirin, 180 mg daily given for one month, resulted in a reduced five-week vascular mortality in AMI patients compared to standard care (9.4 percent versus 11.8 percent, a 21.3 percent relative reduction). The results produced by streptokinase alone were virtually identical to those produced by aspirin (9.2 percent versus 12 percent). As anticipated, the combination of aspirin and streptokinase was superior to either treatment alone (8 percent versus 13 percent). This study reaffirmed the efficacy of aspirin and solidified its crucial role in the treatment of all patients suspected of having cardiogenic chest pain.

1986 Widespread Use of Thrombolytic Therapy for STEMI
In 1986, the GISSI trial of 11,712 AMI patients demonstrated that compared to standard care, an IV infusion of 1.5 million units of streptokinase resulted in a 21-day mortality of 10.7 percent versus 13 percent in controls. The results of this trial prompted progressive initiation of thrombolytic therapy, but it took at least 10 years until the practice was broadly available. The gap between when thrombolytic therapy was first found to be beneficial and the widespread adoption of its use is a classic example in which knowledge translation moved very slowly. Unfortunately, many patients who could have benefited from thrombolytic did not receive it and, as a result, suffered needless mortality, recurrent myocardial infarctions, and heart failure.

1988 Demonstration of the Benefit of Both Aspirin and Thrombolysis for STEMI

References

DR. BUKATA is executive editor of Emergency Medical Abstracts and clinical professor of emergency medicine at the Keck School of Medicine of the University of Southern California in Los Angeles.

To Be Continued...
Want to learn more about the history of emergency care for chest pain in 2000 and beyond? Be sure to read Part Two of this article in the ACEP 50th Anniversary Special Issue, which will be published in September and available at ACEP18.
The sphenopalatine ganglion (SPG) is associated with the trigeminal nerve, the major nerve involved in headache (HA) disorders (see Figure 1). The mechanism behind migraines is not fully understood, but it’s thought that blocking the SPG may help relieve migraine pain.1–3

The SPG is the main source of cranial and facial parasympathetic innervation. The autonomic nerves of the SPG supply the lacrimal glands, which produce tears, as well as the sinuses, which can produce the nasal discharge or congestion associated with some migraines.4–6

**SPG’s Role in Headaches**

When a headache occurs, meninges inflammation activates pain receptors. These receptors send pain impulses through the trigeminal nerve, which then sends a signal to the brain that is perceived as pain. In cluster and migraine HA, pain signals pass through the SPG, connecting with autonomic nerves, which produce eye tearing or nasal discharge. This is called the trigeminal autonomic reflex.2–4

During a migraine, parasympathetic outflow from the SPG causes vasodilation of cranial blood vessels. This dilation allows for inflammatory mediators to activate meningeal nociceptors, which are responsible for the migraine pain. It’s theorized that a patient who experiences parasympathetic symptoms during migraines (eg, nausea, emesis, sweating, lacrimation, etc.) may benefit from SPG blocking because the SPG propagates these parasympathetic signals.3,4

A prospective, randomized and double-blinded placebo-controlled study published in JAMA in 1996 showed potential benefit for using intranasal lidocaine when compared with saline alone.5 The study included a total of 81 patients with a chief complaint of headache who fulfilled criteria for International Headache Society. The primary outcome measure found at least a 50 percent reduction of headache within 15 minutes of treatment. Fifty-five percent of patients had at least a 50 percent reduction of headache compared with 21 percent of patients in the control group, and nausea and photophobia were significantly reduced. Rescue medication for headache relief was significantly reduced in the control group. Of those patients who showed initial improvement, only 42 percent relapsed versus 83 percent in the control group.

**How to Perform an SPG Nerve Block**

The SPG can be locally accessed by several approaches, but for the purposes of quick and easy access in the ED setting, the transnasal approach is best. The materials needed include:

1. Cotton-tip applicator, 10 cm long
2. Anesthetic of your choice:
   - Lidocaine 2%, 4%, or 2% (onset 15 minutes, duration of action 30 minutes to 2 hours)
   - Bupivacaine 0.25% or 0.5% (onset 10–20 minutes, duration of action 2–4 hours)
3. 5 mL syringe and large bore needle to draw up the anesthetic
4. Plastic pill cup or any small container that can hold the anesthetic with enough depth to fully submerge the cotton-tip applicator
5. Atomizer (optional).

The technique:

1. Have the patient lie in a supine position with the head tilted up in a sniffing position.
2. Make sure the patient is on a cardiac monitor. Even though you are using less than the toxic dose, you are administering anesthetic over a highly vascular area.
3. Anesthetize the nasal passage entry by one of two ways (optional):
   - A. Inject 0.5 mL of 2% viscous lidocaine into each nostril with the open end of a 3 mL syringe (without needle). Have the patient sniff to draw the anesthetic posteriorly.
   - B. Use an atomizer to draw up 1 mL of 4% lidocaine per naris and aerosolize into each naris. Remember, you can administer a maximum of 1 mL per naris.
4. Soak one or two cotton-tip applicators in the anesthetic of your choice. If you are pre-anesthetizing the nasal passage, we recommend using only 1% or 2% lidocaine to soak the applicators, the goal being to remain well below the toxic dose.
5. Advance one cotton-tip applicator along the superior border of the middle turbinate of each nostril until the tip contacts the mucosa overlying the SPG (see Figure 2).
6. Leave the applicators in for 10 minutes or until the patient feels relief, then gently remove the applicators.2,3

Finally, remember that epistaxis is an unlikely but potential complication.

**References**


**DR. VIGURI** is associate director of ED pain management at St. Joseph’s Hospital in Paterson, New Jersey.

**DR. PAEZ PEREZ** is an emergency medicine resident at St. Joseph’s Regional Medical Center.
I think we’ve been hit with a lot of regulations. A lot of things that we’re doing that don’t add value to the patient. We’ve seen epidemic levels of burnout, and a lot of this is lack of control over our specialty and the care we provide.

My goal was really to improve communication and transparency. I think there are a lot of people who feel as though ACEP is controlled by large groups. I am in a single hospital group, and ACEP brings tremendous value to me.

PK: Pragmatically speaking, what were some of the things you thought you could do, some of the dials you could turn, or the switches you could flip to help ACEP serve its members better?

PK: The first thing that I’ve learned in my time on the Board is that there are a lot of things that ACEP does that our members don’t even realize we do, so I’ve really worked hard on trying to establish transparency and communication back to the members on the value that ACEP provides to them.

I’ve worked for over a year and a half with staff on developing a new website that will be coming out at the end of this month. Rather than force something that wasn’t ready, we took the necessary time to offer something that will be of great value with much improved function.

I think medical-legal concerns have always been among the biggest frustrations for our members. We’re really going to come forward this year with some ideas that will provide some more support for our members so that they can practice with less discomfort and less fear, while doing the right thing.

I think a lot of our members have been frustrated by mental health boarding. This is very close to my heart. We need to make sure that the emergency department is a safe environment for both our members and also the patients.

We’re moving some issues forward that should decrease boarding and improve the care to patients that see us in the emergency department. I know future presidents will continue these initiatives.

KK: Every ACEP president has important goals. However, when you get into your presidency, the issues of the day, the circumstances you encounter, are what identify some of your day-to-day priorities. What are some of the things that have come up that you never expected would be Paul Kivela’s to solve?

PK: An issue that has popped up that I never expected is communication. A great number of our members communicate via social media. I’ve really taken on the role of trying to make sure that we communicate and address issues via social media in real time. The organization has moved in the right direction. We’re going to help bring the information to you, as opposed to you looking for it.

KK: What’s one example of one of those topics that has really been very timely in social media?

PK: The issue of REBOA (resuscitative endovascular balloon occlusion of the aorta) is a great example. We had previously come up with a consensus statement with a number of other organizations that talked about some issues on REBOA that were not consistent with some of our members’ views.

Via social media, that was brought to our attention, and we quickly responded. I think that shows how our organization can respond in a very quick and efficient manner.

KK: Something you said was really an important point: meeting the members where they are. As people start to consume information differently and they go to different modalities of communicating, we have to be nimble.

PK: We are reviewing every committee objective, nearly 300, making sure each benefits the members. Many are ongoing from year to year. We want to ensure that this work is productive and meaningful.

We’re also moving forward on some issues for medical-legal reform and in psychiatric care that will hopefully translate, in a very short period of time, to improved care and make the lives of emergency physicians better.

I’ve spent time working to unify the specialty. There’s some, unfortunately, duplication within our specialty and there are many challenges that we face. I’ve reached out to work with organizations where our missions either overlap or sometimes conflict. I’ve reached out to NAEMSP [National Association of EMS Physicians], AAEM [American Academy of Emergency Medicine], SAEM [Society for Academic Emergency Medicine], and ACOPE [American College of Osteopathic Emergency Physicians]. I’m really working to coordinate the issues that we can agree on so that we are not reinventing the wheel on each one of these issues. I think I want to try and make things less competitive between the organizations and more collaborative. I think that’s hopefully moving the specialty in a better direction in the end.

ACEP dues are not an inconsequential amount of money. I hope when people look at their dues statement that every emergency physician will be able to say, “I received my value from my ACEP membership.”

KK: Every good president makes certain they have really done their very best to show the value of membership and dues dollars spent. You are certainly making good on that promise. Paul, thank you for your service and for your time. I know that you will have left ACEP in a better place following your presidency.
Thank You

ACEP proudly recognizes these groups that have ALL eligible emergency physicians enrolled as members.

For more information about how your group can participate in the 100% Club, please contact Kelly Govan at 844.381.0911 or kgovan@acep.org

Visit acep.org/grouprecognition for program details

*as of April 2018
ACEP Proposes Physician-Focused Alternative Payment Model

Introducing the Acute Unscheduled Care Model: Enhancing Appropriate Admissions

by JEFF BETTINGER, MD, FACP; AND RANDY PILGRIM, MD, FACP

The 2015 Medicare Access and CHIP Reauthorization Act (MACRA) established new opportunities for physicians to participate in alternative payment models (APMs) in Medicare. To help spur the development of new models, MACRA created an independent committee called the Physician-Focused Payment Model Technical Advisory Committee (PTAC), which reviews proposed physician-focused payment models received directly from health care providers and organizations and makes recommendations to the secretary of the U.S. Department of Health and Human Services on their consideration.

In August 2015, after MACRA passed, ACEP established a task force to help develop an APM geared toward emergency medicine. Until that point, most alternative payment arrangements were not designed to include emergency care in any meaningful way. The APM Task Force spent its first year evaluating numerous concepts centered around improving value and quality of emergency services. After intense internal work by the task force and in consultation with additional data analysis performed by a retained consultant, ACEP submitted a proposal for a new APM, the Acute Unscheduled Care Model (AUCM): Enhancing Appropriate Admissions, to the PTAC in September 2017. If recommended by the PTAC and then approved by the secretary, the AUCM will serve as an advanced APM, allowing emergency physicians who choose to participate to potentially be eligible to receive a five percent Medicare Part B payment bonus. (Advanced APMs are APMs that meet certain criteria established by MACRA, including the requirement that participants take on a nominal amount of financial risk for the services they provide under the model.)

How AUCM Works

The goal of this bundled payment model is to improve quality and reduce Medicare costs by emergency physicians accepting some financial risk for the decisions they make around discharges for certain episodes of unscheduled acute care. It uses an annual retrospective reconciliation, which compares actual spending for each episode to its target price. Target prices for select conditions are calculated based on three years of facility-specific historical claims and a specified discount percentage for the initial emergency department visit plus all costs incurred for 30 days postdischarge. The AUCM model also includes waivers that would allow emergency physicians to be more comfortable with discharge decisions by reimbursing for certain discharge-associated services that are currently unavailable. These include care coordination, postdischarge visits, and certain telehealth services.

Savings in the proposed model are generated when the actual amount spent for emergency department services and 30-day postdischarge services are below the facility-specific, targeted price for that episode. Participating emergency physicians will be able to keep these savings if they meet certain quality metrics. However, if spending for patients is more than the target for an episode, the emergency physicians would also be liable for those losses (capped at a maximum of 10 to 20 percent, depending on participation level).

For the first two to three years, the model will focus on episodes around four high-volume emergency department conditions: abdominal pain, chest pain, altered mental status, and syncope. More episodes will be added over time. Performance on a set of quality measures will determine a participant’s eligibility for savings as well as the size of a discount percentage, which is built into the target price. That discount guarantees at least some savings for the Medicare program.

The AUCM model will be flexible enough to allow the full spectrum of emergency physicians to participate, should they so choose. Ideally, participation will range from those with dedicated infrastructure and experience with reporting and meeting quality metrics and taking downside risk to smaller groups of physicians who do not have as much experience in these areas. Specifically, it will include an alternative quality-scoring methodology with more achievable standards as well as three options for risk sharing that enable emergency physicians to either take on downside risk immediately or accept more risk over time.

Next Steps

A preliminary review team within the PTAC is currently reviewing the model, and ACEP has been actively engaged in answering all of the team’s technical questions. After the preliminary review team finishes its review, the model will be considered by the full PTAC during a public meeting. The next PTAC public meeting is in June, and ACEP hopes that the AUCM model will make it on the agenda. Even if the PTAC does discuss the AUCM model in June and decides to recommend it to the secretary of Health and Human Services, there is still a long road ahead before the model would be operationalized. However, we are prepared to continue to push for this model to be developed and implemented either through the PTAC process or, if necessary, through some other legislative or regulatory vehicle.

While there is still much work to do to get this model across the finish line, we are encouraged by the considerable progress to date. Most importantly, we feel privileged to have had the opportunity to design a payment model that reflects and values the significant role that emergency physicians play in the health care system. Stay tuned for more as the process moves forward.

DR. BETTINGER and DR. PILGRIM are co-chairs of the ACEP APM Task Force.
Working from the Inside to Include Women in Leadership

A woman elected to the ACEP Board was just the beginning of greater gender equity

by PAMELA P. BENSEN, MD, MS, FACEP.

Editor’s Notes: This is part two of Dr. Bensen’s reflection on her challenging path to ACEP leadership. Part one appeared in the April 2018 issue.

In 1982, when ACEP was 15 years old, after five tries, I became the first woman elected to the ACEP Board of Directors. Until then, ACEP had been a man’s world. Administrative assistant Kathy Syke sent me the same letter she sent to all Board members to ask us to wear suits and ties to the Board meeting because our pictures would be taken. Back then, Board members might show up in jeans, shorts, or even bathing suits. In red ink, she added a smiley face and a handwritten note exempting me from the request.

I showed up in my gray flannel skirt, blue blazer, white blouse, and pearls (standard casual business attire of the day). Right before the picture, I stopped the photographer to get a Board vote on the two gaudy ties I pulled from my pocket. That Board picture shows us all smiling broadly and me in my pearls.

Chipping Away at Bias

Some gender bias was just situational blindness easily overcome by humor, casual conversation, or Board discussion, like the notorious men’s room story captured on ACEP’s anniversary film. Some bias was deliberate, insidious, cruel, and never-ending, a painful story for another day.

I was counseled not to knit at Board meetings. Knitting kept me focused and always made me feel that I accomplished something, even during the least productive meetings. So I brought my first computer and, without the benefit of the Internet, social media, or Google, often had my feet up on the table while knitting or to leaving the meeting. It was several years before even staff members had computers. I later discovered that my actions were threatening because everyone assumed I was taking copious minutes. Amazing how big some imaginations can be.

At the Board meetings, Kathy and I were the only women in attendance, and she was there to take notes. At the staff level, department heads who attended Board meetings were initially men. I was blessed with a phenomenal mentor-husband but had no female mentors on the Board. As usual, back then my capable ACEP mentor was the secretary. She who controls the minutes controls the course of the meeting, often finished my Board-assigned tasks prior to even staff members having computers. I failed to recognize at the time that there was a series of glass ceilings. I cracked one, but never President.

In 1983, when I was on the tugboat when the bank was open Monday through Friday. “him” to sign over the weekend because “he” could not break that last glass ceiling. She never became President Elect or President, a fate shared by Charlotte Yeh, MD, FACEP, who became Treasurer (1991), then Vice President, but never President.

In 1991, Nancy Auer, MD, FACEP, was elected to the Board. And in 1997, she became ACEP’s first woman President. The ACEP Board had taken another step toward gender neutrality. ACEP has had five female Presidents since.

Today, when so many women have served on the Board and as officers, some members wonder if bias still exists in ACEP. It does; differences will always generate bias.

Each of us brings a unique perspective to our specialty. ACEP needs to be the place where every emergency physician has a voice and an opportunity. To succeed, ACEP will have to seek out, listen to, and hear individuals who, though qualified to lead, choose not to, those who can join but don’t, those who should stay in the college but leave, and those we have excluded but need to include.

We need to be more aware of problems and solutions beyond our limited individual viewpoints. We need to search for emergency physicians we don’t know, listen to new ideas, broaden our horizon, and return to ACEP with a different outlook. Fifty years from now, ACEP will be glad we did. Now, as in 1982, I ask you not to vote for someone because they are ______, but don’t vote against them because they are ______. You fill in the blanks.

DR. BENSEN is president of Medical Education Programs in Buffalo Junction, Virginia.
other work, an unexpected national spotlight. I recently interviewed her over Skype.

**JF:** When did you first join Twitter and why?

**EC:** It’s been six years now. I joined on the advice of a friend from medical school, Vivek Murthy, MD, MBA, who went on to be the Surgeon General of the United States. We were having lunch, and I was like, “I don’t even know what Twitter is. I don’t get what people do there, and I don’t know how to tweet,” and he said, “Trust me, this is a powerful thing.” I was super-skeptical, but I signed up and did some things with the Doctors for America and the Obama campaign around health care messaging. I participated and then kind of forgot about it. And then this FOAM [free open access medical education] thing happened, so I dug back in and tried to participate in the ways that many people do. I met Seth Truex, MD, MPH (@MDAware), and I was in the same office as Megan Ranney, MD, MPH (@MeganRanney). A bunch of us wrote a Twitter paper about what you should do with it in academia. Those were the days when most people thought it was a waste of time.

**JF:** How many followers did you have before your legendary thread about racism, because now you have around 25,000?

**EC:** I think I had four or five thousand.

**JF:** So at that point you were a fairly well-known academic on Twitter but this moment brought you a new type of following, right?

**EC:** Yes. Up until the low 1,000s, I had a tight circle. Even if that number felt pretty tight. I knew who we were, mostly other physicians and health care providers, but it felt really rich. The beautiful thing at that range was feeling like I had colleagues internationally and a good mix of students, trainees, and people senior to me that I could learn from. When you hit that level, there’s momentum, and I felt like it became very interactive.

**JF:** You could say that you worked hard for years to become an overnight sensation.

**EC:** Exactly! I’m like, “I’ve been here the whole time!” When I look at people I admire in health care with huge followings, people like Eugene Gu, MD (@EugeneGu), Atul Gawande, MD, MPH (@AtulGawande), or Jen Gunter, MD (@DrJenGunter), all of these people have been very on-message the entire time. Jen had this amazing blog for a long time before becoming so obviously known and getting a New York Times column. Their messaging and passion have been very consistent. You don’t become somebody else to do this. You’re just who you are and then you hit a moment where your message resonates with a lot of people.

**JF:** Generally, the “tweet storm” or “thread” has become a natural outlet for you. Can you describe the mechanics of that?

**EC:** This is my favorite thing to do. I love the limited structure of Twitter actually. When you have to be brief, you have to choose your words wisely. But you miss a lot of nuance. I started doing this thing where you post something and then you respond to yourself. So when people pull it up, they see the whole string of messages, and some of them were really long because a bunch of doctors who weren’t on Twitter asked me to post [patients’ stories] in opposition to the AHCA [the American Health Care Act]. That was a nice balance between being able to say a lot but still using the thing about Twitter that makes it beautiful.

**JF:** And how did you develop the thread about the racism you’ve faced that went incredibly viral?

**EC:** It was so organic. You have no idea how spontaneous and random that was. It was a mixture of circumstance and long-brewing thought. Charlottesville [the neo-Nazi protests] happened that weekend, and it was obviously really disturbing how overt the racism had gotten in the last year. I’m in Oregon where there are a ton of white supremacists, so it’s more in my face than ever.

I’m with my family. We’re going to the park, getting ready to play, and my toddler had fallen asleep in the car. So my husband takes the other kids out to the playground, and I’m just sitting in the car doing nothing. I was bored, and of course, as we do, I opened up Twitter, and I just tossed off the thread. The rest of my day happened. I was going into an overnight, so I fell into it and went into my shift, came home, and Chelsea Clinton had retweeted it. It just went kaboom! My email box was just [sounds of explosion]. This was not premeditated or well-thought-out.

I thought there would be a few friends who would pick it up and like it, physicians who I was familiar with about racism, and it was directed at them, but I had no idea that it had general appeal. I still don’t totally understand it. I think it was timing. I think people actually do want to hear doctors’ frontline experiences with that authenticity. A lot of the responses you got were enjoyable, but you also received negative feedback from people who aren’t ready to hear these kinds of things. Could you describe what that’s been like?

**EC:** To some extent, the trolls will always come out. But there are a lot of people who said that they thought that I made it all up. There were over 2,000 comments on Twitter. There were Facebook posts that blew up even more than the Twitter posts. I really can’t go deep down into the Facebook posts because either they double down on racism or they say I made it up so I could get my “five minutes.” There’s a lot of that. Even with a bunch of other physicians chiming in and saying that it is completely believable and normal in our workplace, other people are like, “It’s too much or too extreme, and I can’t believe that happens.”

**JF:** You recently said, maybe tongue-in-cheek, that your whole life changed because of a tweet. In what way?

**EC:** For two weeks, I just got slaughtered with media requests. I had the CNN appearance, which was one of the most stressful experiences of my life because I’m not a public person, and I was trying to finish a research grant. I was ready for things to get back to normal. But I got on the map as a physician who could speak to a number of issues, and my Twitter following is now a ton of journalists. Almost every week, someone in the media reaches out about a new tweet. I’m contributing for SELF and for NBC’s new editorial site. Overall, it’s a positive thing and an opportunity to advocate for our field and portray physicians in a positive light.

**JF:** Now that you’re known as both an academic and a public figure, what do you think about the balance of FOAM, mainstream media, and peer-reviewed research publication? Which is more important, and how should the academic world respond to this?

**EC:** I’ve really come around on this. I used to be a bit of an academic snob, where the press part was this little blip at the end of research. Then I realized the power of having a big, public voice and how you can amplify your work by spending time on the public end of things. I have projects that I spent years on that eight people haven’t read even though it was published in a decent journal. I publish one thing in SELF magazine or the Huffington Post that gets thousands of reads on the first day, and so I wonder if learning how to cultivate relationships with popular press and spending time on social media should be carved-out time, with deliberate training and practice, just like we train people on how to write grants. If we really want to have translation to the public and public health, we should take that part seriously. I also have a number of hard-core academic accomplishments that have only happened because of Twitter. There’s no question that my academic work is stronger because of social media.
Are you aware of the variety of support resources available for ELIQUIS patients?

Think ELIQUIS for the treatment of DVT/PE.

DVT: deep vein thrombosis; PE: pulmonary embolism.

**INDICATIONS**

ELIQUIS is indicated for the treatment of deep vein thrombosis (DVT) and pulmonary embolism (PE), and to reduce the risk of recurrent DVT and PE following initial therapy.

**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)

**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.
  - There is no established way to reverse the anticoagulant effect of apixaban, which can be expected to persist for at least 24 hours after the last dose (i.e., about two half-lives). A specific antidote for ELIQUIS is not available.

- **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.
ELIQUIS® tablets 5mg 2.5mg

In the hospital. At discharge. At home. Consider ELIQUIS.

To learn more about transition of care resources, contact your ELIQUIS representative or call 1-855-ELIQUIS

**IMPORTANT SAFETY INFORMATION**

**WARNINGS AND PRECAUTIONS (cont’d)**

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 puncture occurs, delay the administration of ELIQUIS for 48 hours.

Monitor patients frequently and if neurological compromise is noted, urgent diagnosis and treatment is necessary. Physicians should consider the potential benefit versus the risk of neuraxial intervention in ELIQUIS patients.

- **Prosthetic Heart Valves:** The safety and efficacy of ELIQUIS have not been studied in patients with prosthetic heart valves and is not recommended in these patients.

- **Acute PE in Hemodynamically Unstable Patients or Patients who Require Thrombolysis or Pulmonary Embolectomy:** Initiation of ELIQUIS is not recommended as an alternative to unfractionated heparin for the initial treatment of patients with PE who present with hemodynamic instability or who may receive thrombolysis or pulmonary embolectomy.

**ADVERSE REACTIONS**

- The most common and most serious adverse reactions reported with ELIQUIS were related to bleeding.

**TEMPORARY INTERRUPTION FOR SURGERY AND OTHER INTERVENTIONS**

- ELIQUIS should be discontinued at least 48 hours prior to elective surgery or invasive procedures with a moderate or high risk of unacceptable or clinically significant bleeding. ELIQUIS should be discontinued at least 24 hours prior to elective surgery or invasive procedures with a low risk of bleeding or where the bleeding would be noncritical in location and easily controlled. Bridging anticoagulation during the 24 to 48 hours after stopping ELIQUIS and prior to the intervention is not generally required. ELIQUIS should be restarted after the surgical or other procedures as soon as adequate hemostasis has been established.

**DRUG INTERACTIONS**

- **Combined P-gp and Strong CYP3A4 Inhibitors:** Inhibitors of P-glycoprotein (P-gp) and cytochrome P450 3A4 (CYP3A4) increase exposure to apixaban and increase the risk of bleeding. For patients receiving ELIQUIS doses of 5 mg or 10 mg twice daily, reduce the dose of ELIQUIS by 50% when ELIQUIS is coadministered with drugs that are combined P-gp and strong CYP3A4 inhibitors (e.g., ketoconazole, itraconazole, or ritonavir). In patients already taking 2.5 mg twice daily, avoid coadministration of ELIQUIS with combined P-gp and strong CYP3A4 inhibitors.

**Clarithromycin**

Although clarithromycin is a combined P-gp and strong CYP3A4 inhibitor, pharmacokinetic data suggest that no dose adjustment is necessary with concomitant administration with ELIQUIS.

- **Combined P-gp and Strong CYP3A4 Inducers:** Avoid concomitant use of ELIQUIS with combined P-gp and strong CYP3A4 inducers (e.g., rifampin, carbamazepine, phenytoin, St. John’s wort) because such drugs will decrease exposure to apixaban.

- **Anticoagulants and Antiplatelet Agents:** Coadministration of antiplatelet agents, fibrinolitics, heparin, aspirin, and chronic NSAID use increases the risk of bleeding. APPRAISE-2, a placebo-controlled clinical trial of apixaban in high-risk post-acute coronary syndrome patients treated with aspirin or the combination of aspirin and clopidogrel, was terminated early due to a higher rate of bleeding with apixaban compared to placebo.

**PREGNANCY CATEGORY B**

- There are no adequate and well-controlled studies of ELIQUIS in pregnant women. Treatment is likely to increase the risk of hemorrhage during pregnancy and delivery. ELIQUIS should be used during pregnancy only if the potential benefit outweighs the potential risk to the mother and fetus.

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

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ELIQUIS® (apixaban) tablets, for oral use

WARNING: (A) PREMATURE DISCONTINUATION OF ELIQUIS INCREASES THE RISK OF THROMBOTIC EVENTS

(B) SPINAL/EPIDURAL HEMATOMA

(C) SPINAL/EPIDURAL HEMATOMA

DOSAGE AND ADMINISTRATION (Selected information)

Reduction in the Risk of Recurrence of DVT and PE—

Figure 1: Major Bleeding Hazard Ratios by Baseline Characteristics – ARISTOTLE Study

Table 1: Bleeding Events in Patients with Nonvalvular Atrial Fibrillation in ARISTOTLE

Table 2: Bleeding Events in Patients with Nonvalvular Atrial Fibrillation in AVERROES

Patients with Prosthetic Heart Valve

The safety and efficacy of ELIQUIS have not been studied in patients with prosthetic heart valves. Prophylaxis of Deep Vein Thrombosis Following Hip or Knee Replacement Surgery—

The safety of ELIQUIS was evaluated in the ARISTOTLE and AVERROES trials (see Clinical Trials with comparator anticoagulation) to evaluate ELIQUIS in patients with atrial fibrillation. The safety and efficacy were assessed in ARISTOTLE, the CHADS2 Score

Other Adverse Reactions

In ARISTOTLE, the results for major bleeding were generally consistent across most major subgroups including age, weight, CHADS2, CHA2DS2-VASc, history of stroke, or neurologic, or intracranial events 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 repeat epidural or spinal puncture. If thrombolytic or anticoagulant therapy is required, ELIQUIS should be temporarily discontinued.

Bleeding

ELIQUIS increases the risk of bleeding and can cause serious, potentially fatal, bleeding. (see Warnings and Precautions and Adverse Reactions)

Aspirin

Statin

Bleeding

Intracranial (ICH)‡ 52 (0.33) 125 (0.82) 0.41 (0.30, 0.57) —

Other Adverse Reactions

In AVERROES, in 1.5% and 1.3% on ELIQUIS and warfarin, respectively, and in ARISTOTLE, 1.3% and 1.4% on ELIQUIS and warfarin, respectively.

The following serious adverse reactions are discussed in greater detail in other sections of the Summary of Burden of DVT and PE—

ELIQUIS is indicated for the treatment of PE.

Intracranial (ICH)‡ 52 (0.33) 125 (0.82) 0.41 (0.30, 0.57) —

Effective therapy for deep vein thrombosis or pulmonary embolism (PE) includes 3 to 5 days of heparin or other anticoagulants followed by 2 to 3 weeks of oral anticoagulation. ELIQUIS is not indicated for the treatment of PE.

ELIQUIS is contraindicated in patients with the following conditions:

Increased risk of thrombotic events after premature discontinuation

Indications and Usage

Risk of these events may be increased by the postoperative use of indwelling epidural or spinal catheters in individuals receiving anticoagulation. There is no experience with systemic heparinization (heparin or low-molecular-weight heparin) in individuals receiving anticoagulation. Patients should be monitored closely. In the event of a major bleed, ELIQUIS should be discontinued and appropriate therapy instituted. There is no evidence with systemic heparinization (heparin or low-molecular-weight heparin) in individuals receiving anticoagulation. Patients should be monitored closely. In the event of a major bleed, ELIQUIS should be discontinued and appropriate therapy instituted.

Eligible patients were randomized to receive ELIQUIS (apixaban) or warfarin for a median duration of 1 year. Clinical trial endpoints included stroke, systemic embolism, and death from cardiovascular causes. The safety and efficacy of ELIQUIS were evaluated in clinical studies in patients with nonvalvular atrial fibrillation.

The safety and efficacy of ELIQUIS were evaluated in the ARISTOTLE and AVERROES trials (see Warnings and Precautions and Adverse Reactions). ELIQUIS is indicated for the treatment of PE.

In the ARISTOTLE study, patients received ELIQUIS (apixaban) or warfarin for 1 year. The incidence of major bleeding with ELIQUIS was similar to warfarin in the overall study population. The incidence of intracranial bleeding with ELIQUIS was lower than with warfarin. The incidence of gastrointestinal bleeding was similar with ELIQUIS and warfarin. The incidence of major bleeding in patients with CHADS2 score ≥ 2 and ≥ 3 was lower with ELIQUIS than with warfarin.
Provide your question or request based on the document content above.
Infants with Congenital Heart Disease

A simple three-step approach for time-sensitive diagnosis and treatment

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

The traditional approach to congenital heart disease (CHD) involves a detailed understanding of the pathophysiology, clinical findings, and management of each particular congenital heart defect. However, this cognitive-heavy approach is not practical for the emergency physician faced with an undifferentiated, unstable infant when decision making must be rapid. Despite improved CHD screening in recent years, a small but significant minority of these patients will be undiagnosed when they present to the emergency department.

In this EM Cases column, a simple approach is outlined, allowing the emergency physician to focus on time-sensitive, lifesaving treatments and practical management of the acutely ill infant with CHD.

The Three-Step Approach

1. Age: Younger than or older than 1 month?
   Any infant younger than 1 month old with central cyanosis or shock should be considered to have critical duct-dependent CHD until proven otherwise. This is almost always a left heart lesion such as tetralogy of Fallot, which almost always benefits from prostaglandins. Shunting or mixing lesions such as ventricular septal defect (VSD) or patent ductus arteriosus (PDA) typically present later during infancy, usually after 1 to 6 months of age.

2. Color: Do they appear pink, gray, or blue?
   Infants with undiagnosed CHD usually present to the emergency department in one of three ways:
   a. Pink: Pink-appearing infants with CHD who present to the emergency department with dyspnea should have underlying acute congestive heart failure (CHF) near the top of the differential diagnosis. They have adequate pulmonary blood flow and are relatively well perfused and oxygenated. Heart failure in these patients usually occurs due to a shunting lesion. Always consider CHF in a wheezing pink child. The most sensitive and specific clinical findings for acute CHF in infants include: 1) less than 3 ounces of formula per feed (or greater than 40 minutes per breast feed); 2) a respiratory rate greater than 60 breaths per minute (or irregular breathing); and 3) hypembolia. Other clues include poor weight gain and ventricular hypertrophy on ECG.
   b. Gray: Gray-appearing infants with CHD are usually in shock with circulatory collapse due to poor systemic flow and oxygenation due to a left-side obstructive, duct-dependent lesion. These patients will almost always benefit from fluid administration and, if younger than 1 month in age, prostaglandins.
   c. Blue: The blue appearance of central cyanosis (ie, blue discoloration of the tongue, mucous membranes, and lips) in the setting of CHD usually occurs due to a right-side obstructive duct-dependent lesion in the first month of life or a mixing lesion after one month of life. These infants, like the gray ones, almost always require prostaglandins. There are four important etiologies to always consider in infants with central cyanosis: 1) CHD; 2) sepsis; 3) respiratory disorders (such as pneumonia); and 4) hemoglobinopathies (such as polycythemia and methemoglobinemia).

3. Physical Examination and Bedside Tests
   Observing the following can provide important clues to the underlying diagnosis: the infant’s work of breathing, limb-pulse differentials, blood pressure and pulse oximetry, hyperoxia test results (see below), ECG for left ventricular hypertrophy (LVH) or right ventricular hypertrophy (RVH), and bedside cardiac ultrasound for global cardiac function, septal defects, and chamber count. After determining the infant’s color, the most important clue to CHD observed from the foot of the bed on physical exam is silent tachypnea. Tachypnea with increased work of breathing is usually due to a respiratory cause. In contrast, tachypnea without increased work of breathing—ie, silent tachypnea—is usually secondary to metabolic acidosis from a cardiac or metabolic cause.
   In addition to silent tachypnea, the hyperoxia test helps differentiate respiratory causes from cardiac causes of tachypnea. This test was originally described using the PaO2, garnered from the arterial blood gas. Although accurate, this is cumbersome, painful, and lengthy process.

A simpler modified method involves using the pulse oximeter before and after the patient receives 100 percent oxygen (or as close to a 100 percent FiO2 as possible) for five to 10 minutes and noting whether the oxygen saturation improves. If the oxygen saturation improves, the underlying cause of the oxygen desaturation favors a respiratory etiology. But if the oxygen saturation does not improve, a cardiac cause is more likely.

Proceed with caution when administering the hyperoxia test. Oxygen is a potent pulmonary vasodilator and could worsen respiratory distress in a patient with a duct-dependent lesion by decreasing pulmonary vascular resistance (PVR) and increasing pulmonary blood flow, leading to pulmonary overcirculation.

There are three physical exam maneuvers to consider involving limb-pulse differentials: 1) a pulse delay between radial (preductal) and femoral (postductal) pulses (or absence of femoral pulses); 2) a blood pressure differential between the right upper and a lower extremity; and 3) a difference in pulse oximetry of more than 3 percent between the right upper and a lower extremity. These all suggest a duct-dependent lesion.

Pediatric ECG interpretation can prove challenging for many community physicians. A simple approach with regard to CHD involves the presence or absence of ventricular hypertrophy. The ECG can suggest CHD. The ECG can suggest CHD. The ECG can suggest CHD. The ECG can suggest CHD. The ECG can suggest CHD. The ECG can suggest CHD.

When presented with gray or blue infants suspected of duct-dependent lesions in your emergency department, CHD should be in your differential diagnosis, but it is important to remember sepsis is far more common, and as such, early empiric antibiotics should be started as soon as possible. Start prostaglandin therapy for all acutely ill gray or blue infants younger than 1 month of age to keep the PDA open. Be prepared to intubate and resuscitate the neonate who receives prostaglandins because prostaglandins can cause apnea as well as severe hypotension.

Be judicious with fluids and oxygen. Consider 5–10 mL/kg normal saline boluses rather than the usual 20 mL/kg boluses in the hemodynamically unstable infant to improve preload and encourage further opening of the PDA and pulmonary blood flow through the duct.

Although the pulse oximetry goal in non-CHD patients is greater than 92 percent, in some CHD patients, aiming for this high of an oxygenation can prove deleterious to pulmonary blood flow, and it can worsen hypoxemia. Some CHD patients require a pulse oximetry of only 75 to 85 percent. Inotropes and/or vasopressors may be necessary to maintain adequate systemic perfusion and encourage pulmonary perfusion—consider them in consultation with a pediatric intensivist.

Continued on page 25
Lidocaine for Renal Colic

Should this opioid alternative be used to treat patients with kidney stone pain?

by KEN MILNE, MD, MSC, CCFP-EM, FCFP, FRRMS

The Case
A 51-year-old male presents to the emergency department with a sudden-onset, severe, left-sided flank pain radiating to his groin. It began an hour before arrival. The pain was associated with nausea, vomiting, and difficulty urinating. He does not have a history of kidney stones and is currently writhing around on the stretcher.

Background
About 1 to 5 percent of the U.S. population suffers from kidney stones. The typical ED presentation is sudden onset of pain radiating from the flank to lower abdomen accompanied by nausea, vomiting, and microscopic hematuria.

Renal colic is very painful condition. Opioids are often used for pain relief, along with intravenous nonsteroidal anti-inflammatory drugs. Alpha blockers have been repeatedly studied for use with renal colic. An excellent randomized controlled trial, published in Annals of Emergency Medicine, showed no significant difference in stone passage or time to stone passage with tamsulosin compared with placebo <1cm.1

Lidocaine may be a useful alternative, as it has been used to effectively treat visceral and neuropathic pain.2 Finding non-opioid alternatives to treat painful conditions is timely given the heightened attention on the opioid epidemic.

Clinical Question
In patients presenting to the emergency department with renal colic, is IV lidocaine as or more effective than IV opioids for pain control?

Reference

Evidence-Based Medicine Commentary

1) Renal Colic: I am unsure if the patients enrolled in this trial had renal colic. Inclusion was based on history and hematuria. Follow-up studies included a kidney-ureter-bladder X-ray and/or sonography. Neither of these diagnostic modalities are gold standard methods for diagnosing nephrolithiasis. This could have introduced diagnostic bias into the study.

2) Consecutive Patients: The authors did not explicitly state they used consecutive recruitment, only that they used Randomization.com as their randomization tool. A lack of consecutive recruitment can lead to selection bias.

3) Patient Blinding: It is possible that participants were aware of their group allocation. This is because morphine can produce side effects that could have unmasked the blinding. It is unclear if this potential bias would favor the morphine or the lidocaine group.

4) Provider Blinding: The providers may have also been unblinded, which could introduce bias into the trial. This is because differences in weight-based dosing would result in different volume administrations of medications (ie, a 100 kg patient would be dosed 550 mg [25 mL] of lidocaine but would be dosed 10 mg [the full 10 mL] of morphine).

Clinical Versus Statistical Significance: This is the key limitation of the trial. Although the results obtained statistical significance, the standard deviations of the means in every group at each measured time interval overlap and do not appear to have clinical significance.

Table 1: Comparison of the Mean Value of Pain Reduction Between Two Groups

<table>
<thead>
<tr>
<th></th>
<th>GROUP I</th>
<th>GROUP II</th>
<th>P VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>primary VAS</td>
<td>9.65±0.88</td>
<td>9.74±0.63</td>
<td>0.365</td>
</tr>
<tr>
<td>VAS0</td>
<td>3.18±2.27</td>
<td>4.45±2.16</td>
<td>0.0001</td>
</tr>
<tr>
<td>VAS10</td>
<td>1.83±1.59</td>
<td>2.89±2.07</td>
<td>0.0001</td>
</tr>
<tr>
<td>VAS30</td>
<td>1.37±1.32</td>
<td>2.55±1.52</td>
<td>0.0001</td>
</tr>
<tr>
<td>VAS60</td>
<td>1.13±1.15</td>
<td>2.23±1.57</td>
<td>0.0001</td>
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</tbody>
</table>


Key Results
The researchers enrolled 240 patients into the study, with 120 in each group. The mean age was in the mid 30s.

The trial was considered “accomplished” when either the patient had a pain score of less than 3 for 30 minutes after the last analgesic dose, or the 10 mL of solution in the syringe (either 700 mg lidocaine or 10 mg morphine) was exhausted. See Table 1 for the results.

90 percent (108/120) of patients responded to lidocaine successfully.

70 percent (84/120) of patients responded to morphine successfully.

The number of patients experiencing side effects was similar in both groups.

Clinical Versus Statistical Significance: This is the key limitation of the trial. Although the results obtained statistical significance, the standard deviations of the means in every group at each measured time interval overlap and do not appear to have clinical significance.

Bottom Line
This study does not provide good evidence for using lidocaine to treat patients presenting to the emergency department with renal colic.

Case Resolution
You perform a bedside ultrasound, and it demonstrates mild left hydro nephrosis. The patient is given ketorolac 10 mg IV and his pain resolves. He is then discharged home with analgesics, expectant management, and explicit instructions on when to return to the emergency department.

Thank you to Tony Seapaul, MD, chairman of the department of emergency medicine at the University of Arkansas at Little Rock, and Rachel Littlefield, MD, who is an emergency medicine resident at the University of Arkansas, for their help with this review.

Remember to be skeptical of anything you learn, even if you heard it on the Skeptics’ Guide to Emergency Medicine.

References
Enema or PEG?

Question 2: For fecal disimpaction of children with functional constipation, are enemas better than oral polyethylene glycol (PEG) therapy?

Rarely does a day seem to pass without having the constipation talk with the family of a child with abdominal pain. For the fecal disimpaction portion of constipation, PEG therapy has been shown to be safe in children with an optimal dosing of 1–1.5 g/kg/day.1 Regarding head-to-head trials of oral PEG therapy versus enema, two trials address this specific question.

This first study is a prospective, randomized controlled trial treating children ages 4–18 years with functional constipation and rectal fecal impaction (RFI) who presented to an outpatient clinic (n=90 total patients).2 All patients were confirmed to have a fecaloma (ie, a large amount of hard stool in the rectum) by digital rectal examination. Children were randomized to either PEG 1.5 g/kg/day for six consecutive days (n=44 patients) or a discyl sulfosuccinate enema once daily for six consecutive days (n=46 patients). After this initial disimpaction intervention, the patients performed maintenance PEG until follow-up two or more weeks later. The patients’ families recorded a bowel diary and other objective findings, such as colonic transit time, and tracked bowel motility.

The result? There was no statistically significant difference between the oral PEG and enema therapies. Disimpaction was successful (defined as no fecaloma on rectal exam at follow-up) in 80 percent of the enema group versus 68 percent in the PEG group (P=0.28). (Note: Although there was no significant difference, this study’s protocol is severely limited for ED purposes because it entailed six straight days of therapy.)

A separate prospective, randomized convenience study of children ages 1–17 years (n=80 total patients) evaluated single enema versus three days of PEG therapy in a pediatric emergency department.3 Patients were randomized to either a milk and molasses enema (41 patients at 10 ml/kg, with a max of 500 ml) or PEG (39 patients at 1.5 g/kg/day with a max of 100 g/day) for three days. When this intervention was complete, the patients began maintenance dosing PEG for another three days.

1. To assess the primary outcome of symptom improvement, patients/families were contacted by telephone on days one, three, and five to determine whether symptoms were improving, staying the same, or worsening. These subjective outcomes were compared dichotomously as improving/getting better versus worse/same. Other additional follow-up questions were assessed and dichotomously assessed as well.

Subjective outcomes are a potential limitation of this study and subject to recall bias. That said, at day one, there was a statistically significant difference in improvement when comparing the enema group with the PEG group (odds ratio 0.3; 95 percent CI, 0.1–0.8), but there was no significant difference in symptoms at days three and five. According to the authors, “Enemas produced more rapid initial symptom improvement,” but again, there was no difference at days three or five. Unsurprisingly, 54 percent of patients in the enema group were “somewhat upset” or “very upset” by the therapy at day one follow-up compared with 0 percent in the PEG group.

A more recent systematic review included only these two trials, admitting that there are a number of significant limitations to the study and concluding, “Current evidence does not allow us to conclude which intervention is more effective for treating rectal faecal impaction in children with functional constipation.”

Summary

Only limited data exist comparing a single enema in the ED setting versus home PEG therapy for disimpaction of children with constipation and fecal impaction. In a single pediatric emergency department study, an enema in the emergency department prior to discharge frequently demonstrated improvement in symptoms at day one, but was not superior to oral PEG home therapy at days three or five.

References


Tips for Negotiating Family Leave

Know what the Family and Medical Leave Act does—and does not—cover

by SARAH HOPER, MD, JD, FACEP

Imagine having a similar conversation with one of your residents who is considering their first job offer:

SH: This is a pretty good contract for your part of the country. Is your employer providing paid parental leave for the birth of your children?

Resident: Oh yeah, I’m totally covered—I have FMLA!

SH: Don’t be fooled! FMLA is not paid leave.

What Does FMLA Really Cover?

Implemented during the Clinton administration in 1993, the Family and Medical Leave Act (FMLA) is a federal law that guarantees unpaid leave for qualifying employees. The law mandates that employers with more than 50 workers allow an employee to leave for 12 weeks without fear of being fired and/or replaced, provided the employee has held that job for 12 months and logged at least 1,250 hours. Employees may be absent for 12 work weeks over a 12-month period for any of the following reasons:

• The birth of a child or placement of a child with the employee for adoption or foster care.
• To care for a spouse, child, or parent who has a serious health condition.
• For a serious health condition that makes the employee unable to perform the essential functions of the job.
• For any qualifying exigency arising out of the fact that a spouse, child, or parent is a military member on covered active duty or called to covered active-duty status.
• To care for a covered service member with a serious injury or illness when the employee is the spouse, child, parent, or next of kin of the service member (eligible for 26 work weeks unpaid).

Generally, employees need to make such requests 30 days in advance, assuming the need, such as pregnancy, is foreseeable. Some employers may require even longer notice. Employees who return from FMLA leave must be restored to their original job (or an equivalent position) with equivalent pay, benefits, and other terms and conditions of employment. Employers often tell me that when they’ve asked a prospective employer about “paid parental leave,” they’ve been told the job offers FMLA protections. I am uncertain if interviewers realize that many take this as confirmation of paid leave. Interpreted correctly, the statement means, “Yes, you may leave, but don’t expect to get paid while you’re gone.”

FMLA does not pertain to employers with fewer than 50 employees. Many small groups fall under this exemption. Therefore, these groups are not required to give any leave. In fact, some groups are small enough that they may not have enough physicians to cover the vacant shifts.

Paid Leave

Upon recently accepting a job, a physician I knew was handed a two-page contract and told to reference the faculty manual for further details. The manual, which stated that faculty members were entitled to six weeks of paid maternity leave, listed no restrictions. However, when she arrived at her new job four months’ pregnant, she was told that she was ineligible for FMLA. Her employer explained that she did not qualify because she would not have worked there for one year or 1,250 hours by the time her child would be born.

In this case, it was up to the employee to school the employer on the law. She explained that her contract and FMLA were mutually exclusive. The terms of her contract alone qualified her for paid leave.

Paid leave is most commonly found in academic and hospital contracts. It can be more difficult to find this benefit in groups whose salaries are based on relative value unit (RVU) productivity or physician staffing or contract management groups. Some productivity-based groups argue that paid leave unfairly burdens working physicians, who essentially are forced to finance another employee’s leave with the RVUs they earn during the absence. Independent contractors are unlikely to find a contract with paid parental leave. Paid leave can be negotiated. A successful negotiation often depends upon the demand for the position. A group with 10 interested people for every job is much less likely to negotiate than a group that is having a difficult time filling its positions. Physicians who are paid hourly prior to becoming a partner may be in a better position to negotiate paid leave. Presuming the RVUs a new employee bills will outpace the hourly wage. In such cases, the group can use those profits to subsidize the employee’s leave without affecting fellow physicians’ RVU compensation.

Alternatives to Paid Leave

There are alternatives to traditional paid parental leave. Some employers, most commonly staffing and physician groups, offer low-interest loans to help physicians finance family leave. Commonly, these loan payments are withdrawn directly from paychecks when the employee returns to work. Other physicians are able to “bank” paid sick leave and use their sick leave to fund their parental leave.

There are many different practice types in emergency medicine—being an independent contractor, an employee of a staffing group, or a partner in a democratic group, just to mention a few. Our varied types of practice make it difficult to have a one-size-fits-all answer to paid parental leave, which is why it is so important for physicians to discuss the parameters of parental leave with their new groups and employers and to make sure it is addressed in their contracts.

Resident: Wow, I thought I was getting paid parental leave. Do I have any power to ask for paid leave?

SH: Yes! You bring valuable skills to the table. You have many job opportunities available to you. Not every negotiation will be successful, but you shouldn’t fear negotiation. It’s part of the contracting process. If you don’t ask for paid parental leave, it won’t be given to you. The more physicians negotiate for paid parental leave, the more commonplace it will become in our contracts.

Avoid ketamine in patients suspected of CHD because it increases systemic vascular resistance (SVR), which worsens left-to-right shunting and can lead to cardiovascular collapse. Etomidate is preferred to minimize changes in hemodynamics.

Be judicious with positive-pressure ventilation. Start with a very low positive end-expiratory pressure (PEEP). Positive-pressure ventilation can increase PVR and decrease SVR as well.

Positive-pressure ventilation can increase PVR and decrease SVR as well.

Looking for signs of ventricular hypertrophy on ECG, use bedside ultrasound to determine global cardiac function, the presence of a septal defect, and the chamber count. Be judicious with fluids, oxygen, and positive-pressure ventilation. Consider pros- taglandins for all blue and gray neonates, and avoid ketamine.

If you remember these simple principles, you could save an infant’s life.

Special thanks to Dr. Gary Joubrt and Dr. Ashley Strobel for their contributions to the podcast from which this article was inspired.

References

The Contesteed Admission

Tips to reduce harmful admission delays

by SHARI WELCH, MD, FACEP

As emergency departments have struggled with inefficient admission processes, a new domain called the ED-inpatient interface (EDii) has been identified. In the December 2017 issue of Emergency Medicine Australasia, Staib et al discussed and characterized the importance of this interface.1

Meanwhile, in emergency departments across the country, the term “contesteed admissions” has been used to depict the problem of getting patients with an increasing number of comorbid conditions admitted. The contested admission refers to any discussions, testing, or consultations that delay the admission process—in other words, any answer but “yes” to the admission call.

The contested admission contributes to ED boarding, and a robust body of literature describes the ill effects produced by boarding (see the sidebar, “The Badness of Boarding”). How are facilities are reducing admission inefficiencies in general and contested admissions delays?

Three Areas of Inefficiency

Hospitals intensely focused on admission efficiency have discovered there are three areas in which inefficiencies can occur. First, bed assignment has been a source of delay, particularly in the current era of inpatient geography (ie, services with strict unit assignments) and in facilities that employ capping (ie, strict numbers of admissions allowed by services). However, many facilities have streamlined these processes with tele-tracking products and performance improvement initiatives. The admit-decision-to-departure Centers for Medicare and Medicaid Services metric currently measures the time from the admission order or bed request to departure.

Hospital services such as housekeeping and transport services can also contribute to delay. Many facilities staff environmental services (EVS) heavily on the day shift, but hospital discharges often peak in the late afternoon or early evening. This demand-capacity mismatch ensures terminal room cleaning takes more time than the 30-minute industry standard. In addition, housekeeping and transporters often lack a systematic deployment scheme, and time is wasted walking between medical center towers. Improved deployment strategies can improve both housekeeping room turnaround and transport times.

The area that currently accounts for the longest delays, however, is the time from the call to the admitting team until the admission is accepted. Emergency departments often get pushback from various services and requests for further testing and consultations. This contested admissions phenomenon at the EDii gives the impression services are trying to avoid patient admissions.

To obtain an idea of the problem’s magnitude, an informal and unpublished survey of academic emergency medicine chairs was completed. Half of the academic programs that responded to the survey were working on the problem, according to Bruce Adams, MD, chairman of emergency medicine at the University of Texas Health Science Center at San Antonio. Another unpublished study conducted at Virginia Commonwealth University showed 39 percent of admissions were contested, which added three hours to patients’ ED length of stay. Services outside the emergency department often report that additional testing is faster in the emergency department. However, Penn State Health Milton S. Hershey Medical Center’s imaging department studied the time it takes to obtain imaging studies and found studies were obtained only 15 minutes faster in the emergency department. This finding argues against holding patients in the emergency department for additional diagnostics.

Possible Solutions

To improve delays related to contested admissions in your facility, considering employing the following tactics:

Admission agreements: The first set of admission agreements we know of were the Stanford Admission Rules drafted in 2004. They were presented in a matrix and pro-

The Badness of Boarding

Studies show boarding can produce many negatives:

• The quality of care for patients with multiple conditions suffers from boarding in the emergency department.3,4
• Delays getting patients to inpatient beds have been associated with a variety of adverse events.5
• Boarding is associated with increased in-hospital death rates.4,6
• Outcomes of patients with pneumonia, acute myocardial infarction, sepsis, and trauma are less favorable when patients are boarded in the emergency department.6
• As boarding time increases, inpatient length of stay increases, and waits and delays of ambulatory discharged patients also occur.7,13
• Admitted patients boarding in the emergency department experience delays in medication administration and missed orders.8,15
• Patient experience suffers when patients are boarded in the emergency department; patients would prefer to be boarded in inpatient hallways.8,14

SHARI WELCH, MD, FACEP

is a practicing emergency physician with Utah Emergency Physicians and a research fellow at the Intermountain Institute for Health Care Delivery Research. She has written numerous articles and three books on ED quality, safety, and efficiency. She is a consultant with Quality Matters Consulting, and her expertise is in ED operations.
vide basic agreements for admissions to different services. Admission agreements can take months to years to draft and still do not anticipate every possible scenario. I recently witnessed a case of a patient on warfarin with a head injury who was neurologically intact. The ED workup revealed a ST-elevation myocardial infarction and an ischemic foot. More than four hours were spent determining the admitting service. Areas of contention included orthopedics and medicine, neurology, and neurosurgery.

Bridging orders: Bridging orders should be short-term and timed-out, allowing patients to be admitted from the emergency department to the floor while the admitting service finishes clinical or surgical work. These orders have always been endorsed by the Institute for Healthcare Improvement (IHI). They are useful in smaller facilities but can have a place in busier facilities and academia, too.

No-refusal policies: Many organizations have adopted no-refusal policies, which may be applied on the physician side and the nursing side. Such policies mean when a bed is available and a service identified for admission, there is no answer but yes. The emergency department is empowered to determine the admitting service. This model has been applied at Brown University, Washington University, Brigham and Women’s Hospital, and Carolinas Medical Center. Some sites have taken an additional step of allowing a service to refuse a patient as long as it then finds an alternative arrangement for the patient.

Shared metrics: According to Edward Jauch, MD, MS, professor and director of the division of emergency medicine at the Medical University of South Carolina, his institution has implemented shared metrics for admitted patients for the emergency department and admitting services. Shared metrics include a goal of one hour for admission to the surgical ICU. This policy originated in the C-suite and puts income at risk for not meeting shared metrics, including length of stay. It also requires professionalism and courtesy. When this policy went live, it produced a profound effect on patient flow.

Incentives for residents: The UMass Memorial Medical Center in Worcester used cafeteria vouchers to incentivize residents to increase the number of patients discharged by noon, which would open up beds for admitted ED patients.

Final Thoughts

Data surrounding contested admissions will soon be at our fingertips. Most tracking systems can now track the time from the first consultation called to admission order or bed request. The time interval between that time stamp and the admission order or bed request will more accurately capture the pain of...
the contested admission. That data will drive process changes.

By tackling the contested admission problem as a hospital, medical center, or medical school, we can improve quality, safety, efficiency, and the experience of care. Why not address your contested admissions using some of these cutting-edge strategies?

References

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Department of Emergency Medicine

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Professor and Chair
Department of Emergency Medicine, Unit 1468
The University of Texas MD Anderson Cancer Center
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Email: kalia@mdanderson.org

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FOR ADDITIONAL INFORMATION, PLEASE CONTACT:

Susan B. Promes, Professor and Chair, Department of Emergency Medicine, c/o Heather Peffley, Physician Recruiter, Penn State Health Milton S. Hershey Medical Center, 500 University Drive, PO Box 855 Mail Code A595, Hershey PA 17033, Email: hpeffley@pennstatehealth.psu.edu

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Pathogens

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Viruses</th>
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<tbody>
<tr>
<td>Escherichia coli K1</td>
<td>Cytomegalovirus (CMV)</td>
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<tr>
<td>Haemophilus influenza</td>
<td>Enterovirus</td>
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<tr>
<td>Listeria monocytogenes</td>
<td>Herpes simplex virus 1 (HSV-1)</td>
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<tr>
<td>Neisseria meningitidis</td>
<td>Herpes simplex virus 2 (HSV-2)</td>
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<tr>
<td>Streptococcus agalactiae</td>
<td>Human herpesvirus 6 (HHV-6)</td>
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<td>Streptococcus pneumonia</td>
<td>Human parvovirus</td>
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<table>
<thead>
<tr>
<th>Yeast</th>
<th>Varicella zoster virus (VZV)</th>
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<tbody>
<tr>
<td>Cryptococcus neoformans/gattii</td>
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1 Data on file at BioFire Diagnostics