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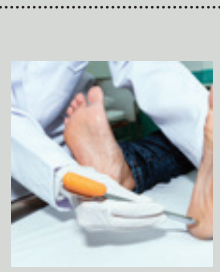

EM CASES

6-Step Approach to Acute Motor Weakness

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Pain Relief in Palliative Care

Tips for treating pain and increasing comfort for dying patients

by HALEY VERTELNEY, MD; MICHAEL RAY GARCIA, MD; AND ERIC ISAACS, MD, FACEP

Emergency physicians are proficient in recognizing and caring for the dying medical or trauma patient but often have limited training in managing patients who are actively dying from a terminal illness, organ failure, or frailty.[1] These patients frequently present to emergency departments and have a predictable set of symptoms that can be managed. Emergency physicians should understand the treatments available and how to use them for patients arriving from hospice or transitioning to comfort care in the emergency department. This article will re-

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SKEPTICS' GUIDE TO EMERGENCY MEDICINE

Steroids for Cardiac Arrest

Does administering them lead to better neurological outcomes?

by KEN MILNE, MD

The Case

A 54-year-old man is admitted to the observation unit to rule out an acute coronary syndrome. While waiting for further testing, he has a cardiac arrest, and a code blue is called. CPR and advanced cardiac life support protocols are initiated. The resident asks if we should be administering corticosteroids because of an old trial she remembers reading.

Clinical Question

Is there better survival with a good neurological outcome in patients with cardiac arrest who are treated with corticosteroids?

Background

There are high morbidity and mortality rates with both in-hospital cardiac arrests (IHCAs) and out-of-hospital cardiac arrests (OHCAs). Improving outcomes for patients with cardiac arrest has been challenging. Various treatment modalities, such as therapeutic hypothermia, intravenous versus

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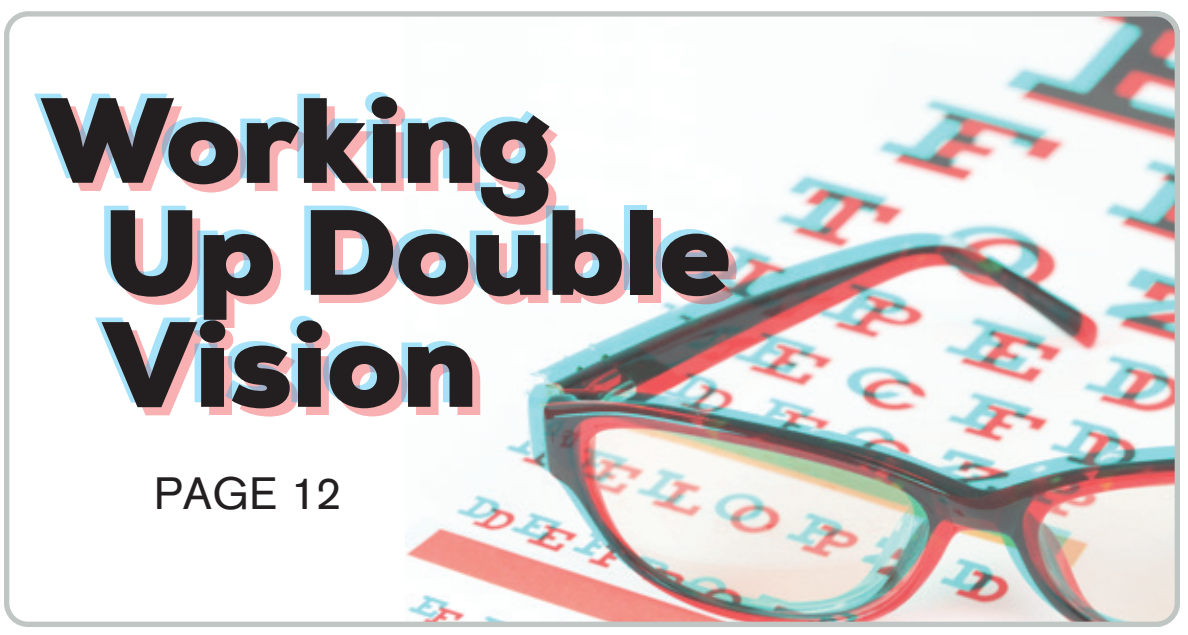

Working Up Double Vision

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NEWS FROM THE COLLEGE

UPDATES AND ALERTS FROM ACEP

Introducing Our New Medical Editor in Chief

ACEP Now is pleased to announce that Cedric Dark, MD, MPH, FACEP, is our new Medical Editor in Chief. Dr. Dark is a longstanding columnist (PolicyRx) for *ACEP Now* and a member of the Editorial Advisory Board. He's also an attending physician, assistant professor, and health policy scholar at Baylor College of Medicine in Houston. Learn more about Dr. Dark in the August 2021 issue, and turn to page 4 to read parting words from our current Medical Editor in Chief Jeremy Faust, MD, MS, MA, FACEP.

ACEP Calls for UnitedHealthcare to Abandon Retroactive ED Coverage Denials Policy

When UnitedHealthcare revealed that a new policy would allow the insurer to retroactively deny ED claims it determines are nonemergent, ACEP immediately responded with a strong public rebuke of this dangerous policy. This announcement kicked off six days of aggressive advocacy that resulted in UnitedHealthcare delaying its policy until at least the end of the public health emergency for COVID-19.

Shortly after this delay was announced, ACEP and 32 organizations representing patient advocates, hospitals, and physicians across medical specialties called on UnitedHealthcare to permanently abandon its planned policy to retroactively deny patients' emergency care claims. To protect the nation's 150 million yearly emergency patients, ACEP is resolute that a delay is insufficient—UnitedHealthcare needs to scrap the policy altogether. Read the full recap at www.acep.org/advocacy-uhc-reversal.

Giving COVID-19 Vaccines in the ED

As an emergency physician, you can help increase the number of people who are vaccinated. On June 11, ACEP President Mark Rosenberg, DO, MBA, FACEP, spoke with Centers for Disease Control and Prevention Director Rochelle Walensky, MD, MPH, about ED vaccination programs during a live-streamed White House Town Hall. Dr. Rosenberg was one of five health care leaders chosen to discuss innovative approaches to patient outreach and vaccine programs with moderators Anthony Fauci, MD; Dr. Walensky; Vivek Murthy, MD, MBA; Marcella Nunez-Smith, MD, MHS; and Bechara Choucair, MD.

ACEP encourages you to consider working with your institution to provide vaccines to appropriate patients who will be discharged from the emergency department. We have resources to help you get started at www.acep.org/vaccinationprogramsintheED.

Teaching Fellowship Opportunity Coming Up

Improve your skills as a professional medical educator or explore your options to become one. Don't miss your chance to register for the ACEP Teaching Fellowship and Resident Teaching Fellowship—classes are filling fast. For faculty, Class 1 is Aug. 9–13, and Class 2 is Aug. 10–14. The class for residents is Aug. 13–15. Register at www.acep.org/teaching-fellowship.

Louisiana Gets Important Scope-of-Practice Win

In early May, HB 495, a bill that would give full practicing authority for advanced practice registered nurses, was progressing through the Louisiana legislative process and looked poised to become state law. If passed, some advanced practice registered nurses would have been permitted to practice independently without physician oversight. But thanks to a passionate deluge of lobbying from physicians and concerned constituents throughout the state, the legislation period ended without the bill being brought to vote in the state senate.

"Louisiana physicians of all specialties came together and basically bombarded the senate with calls, emails, and pleas to stop this assault on patient safety. The senators openly discussed how many calls they were getting about this bill. They were hit from both sides," said Deborah Fletcher, MD, FACEP, an emergency physician in Shreveport, Louisiana, who testified at the hearings about HB 495. "In the end, they heard that Louisiana physicians do not endorse this bill." Read more at www.acep.org/LA-scope-of-practice-win.

New Checklist Helps Vet Employment Contracts

Employment contracts are complex and often difficult to navigate. ACEP's new employment contract checklist (www.acep.org/employmentcontract) is designed to help you consider all the right questions when reviewing a contract. Review all the resources to make the best decision for your career and feel confident you're protected before you sign on the dotted line. This and additional career resources are available on demand in ACEP's new Career Center at www.acep.org/careers.

Celebrating Accomplishments and New Beginnings

A message from *ACEP Now*'s Medical Editor in Chief

by JEREMY SAMUEL FAUST, MD, MS, MA, FACEP

SUSAN R. SYMONDS FOR MAINFRAME PHOTOGRAPHICS

Dr. Jeremy Samuel Faust

I wrote my first column for *ACEP News* as a fourth-year medical student in 2012. By allowing me to keep contributing columns during my early residency years, Bob Solomon, MD, FACEP, then the editor of *ACEP News*, in effect gave me my first recurring "job" as a writer. I could write about whatever I wanted, whether it was light-hearted, serious, or edgy. What a gift that was.

When, under the visionary leadership of Kevin Klauer, DO, EJD, FACEP, *ACEP News* became *ACEP Now* in 2014, I stayed on as a columnist, thanks to some backroom support from ACEP staff members Nancy Calaway and Darrin Scheid. (I never forget, and I always celebrate, those who have helped me along the way. Thanks, you two!) I soon joined the magazine's editorial advisory board. Kevin had me writing dispatches about social media and medicine. It was service journalism, sure, but it kept me in the game and provided me with a front-row seat as a great magazine was being built and maintained. (It also freed me up to write about larger issues for other media outlets like *Slate*, where I soon became a regular contributor, and which led to my more recent work as a frequent contributor to the *Washington Post*, *The New York Times,* and elsewhere.) It's no exaggeration then to say that *ACEP Now* has been an integral and formative part of my life as an emergency physician and writer, from the beginning of my career through today.

Naturally, I was thrilled to be offered the Medical Editor in Chief role for *ACEP Now* when Dr. Klauer left the position to become the Chief Executive Officer of the American Osteopathic Association. When I started in the role, I already understood the strengths of the magazine and also knew the areas where we could improve. Thanks to the professionalism and dedication of the team at Wiley, led by Dawn Antoline-Wang, our fearless, brilliant, incredibly well-organized, and somehow still fun and cheerful editor there; the fine craftwork, intelligent instincts, and good humor of Chris Whissen, our art director; plus ACEP's passionate staff members Nancy Calaway and Jordan Grantham, we accomplished in two years what I thought would take at least five. The goal was to make sure that the magazine better reflected the College's membership, had a modern look and feel, and captured the Herculean efforts of our physician members and full-time staff in Dallas and Washington, DC.

Our Achievements

Here is just some of what we have accomplished:

- My first priority was to create new and recurring columns that better reflected the diversity of our membership and the outstanding advocacy by ACEP's membership and its professional staff. I am proud that the very first recurring column I created was "The Equity Equation." Curated by Dara Kass, MD, and Uché Blackstock, MD (and now by Jenice Baker, MD, FACEP), the column represents a major step in our commitment to covering gender issues, racial equity, and racism in medicine on a continuing and regular basis.
- We next focused on emerging voices in our field, creating "Resident Voice" and "Residency Spotlight." These spaces are consciously meant to establish loyalty and excitement about the College from the people who will be our future colleagues and who will inherit the legacy we create today. Recently, to bolster the "Resident Voice" column, we announced our first Resident Fellow, who will steward this column and keep it current and lively (read more on page 17).
- "FACEPs in the Crowd" is another a feature I initiated to promote our FACEP members and their diverse talents both in and outside of medicine. The idea was to highlight creative wellness opportunities, to promote our own members, and to provide another incentive for members to apply for FACEP status.
- "Medicolegal Mind" draws on expertise from Gita Pensa, MD, and Eric Funk, MD, who write about litigation concerns and challenging cases and documentation, respectively. Dr. Pensa's columns destigmatize litigation—a substantial risk to all practicing emergency physicians—so readers are educated about its realities and so physicians facing legal proceedings may feel less isolated and less shame. In this way, we created a column that simultaneously empowers and informs readers while subtly offering wellness and coping strategies to overcome what is a significant source of stress for many of our colleagues. Similarly, the case reviews by Dr. Funk provide summaries of challenging clinical scenarios, seamlessly blending medical education on evolving standards of care, quality, and metacognition into a succinct and powerful format.
- In "By the Numbers," another new section, we have devoted space to facts and figures on gun violence, suicide, the opioid epidemic, intimate partner violence, injury prevention, COVID-19, and more in clear and easy-to-digest data snapshots. The numbers speak for themselves, arming our members with data to understand and, where appropriate, advocate effectively for our profession and public health in the public space. I am proud that we have found a way to cover these important issues without politicizing them.
- Last year, we inaugurated a column to cover COVID-19 research to help readers stay updated on a wide array of emerging knowledge.
- More recently, we added the new column "Practice Changers," written by Lauren Westafer, DO, MPH, an expert in research knowledge translation. This column provides more of the evidence-based cutting-edge medical education that members value.
- While we attracted new talent, we also kept our valued long-time contributors active so that we have a combination of new and established voices.
- We also improved our coverage of ACEP's advocacy, making "ACEP4U" a regular staple of the magazine, written by ACEP staff member Jordan Grantham. Helping our members understand the advocacy done by the professionals at ACEP in Dallas and Washington, DC, has been a central theme of my tenure, as I felt that most members (especially those of "my generation" and the next) do not understand the full extent and implications of these efforts, which, in my opinion, deserve greater attention and appreciation.
- During my tenure, we added one new member of the *ACEP Now* editorial advisory board, Cedric Dark, MD, MPH, FACEP. Dr. Dark's column, "PolicyRx," began prior to my editorship, although the idea to recruit Dr. Dark to create this particular column was mine. I was pleased that my predecessor, Dr. Klauer, was wise enough to inaugurate that column and that it continued during my editorship. The fact that Dr. Dark has been named as my successor as Medical Editor in Chief of *ACEP Now* is a source of great satisfaction for me. More on that later.

By making these changes and improvements and others, our team at *ACEP Now* has engaged more members and taken concrete steps to begin addressing our profession's long-standing challenges with inclusion, diversity, and representation. While more work remains to be done in this area, our feature contributors and regular columnists are more diverse by any measure than at any time in the history of the magazine.

In sum, I believe we have raised the quality of the magazine beyond even the truly excellent standards established by my predecessors. The magazine continues to win industry APEX Awards for Publication Excellence and remains among ACEP members' most valued benefits. When you see the magazine online or in your real-life snail-mail mailbox, I know that many of you do, in fact, pick it up and read it. "Where have I seen you?" I am often asked by someone I've just met at ACEP Scientific Assembly or another emergency medicine event. "You've probably seen my face either on your desk or your bathroom floor," I sometimes joke. I play it for laughs, but behind it is a genuine message: I'm proud that this magazine reaches people who I respect and admire in our field.

A New Chapter

What's next? I'm pleased to share with you some details of my next chapter. Over the past several months, I've been working with a major partner on launching a new newsletter which will be published at least twice per week. I'll be writing about all areas of medical news and research, though of course as I am a practicing emergency physician, the lens will always be one that you'll recognize. I hope you'll join me when we go live. That's all I am permitted to say for now, but if you follow me on Twitter @JeremyFaust or Instagram @JeremySamuelFaust, you'll be the first to know when we are up and running.

Lastly, I know you're in great hands. There's no one I would rather pass the baton to than my successor-designate, Dr. Cedric Dark. Dr. Dark is not only a wise and passionate advocate for emergency medicine—by which I mean that he is fiercely committed to helping both physicians and our patients—but he is a good friend. I've called on him for advice many times during my time in this role, and I know how he thinks. He'll be a rigorous and fair steward of this magazine and help us navigate the important and complicated issues that we face today. The renewed energy he is already bringing to *ACEP Now* will take the magazine, and therefore all of us associated with the College, to new heights.

Farewell! And thanks to vaccines, I will see you at ACEP21 here in Boston!

DR. FAUST is Medical Editor in Chief of *ACEP Now*, an instructor at Harvard Medical School, and an attending physician in the department of emergency medicine at Brigham and Women's Hospital in Boston.

RESIDENCY SPOTLIGHT

WELLSTAR KENNESTONE EMERGENCY MEDICINE RESIDENCY

Twitter:
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Location:
Marietta, Georgia

Year founded:
2017

Current Number of residents:
14

Program length:
Three years

What makes your program stand out?

We are a high-volume tertiary care community hospital with only four other residency programs in the hospital. That means when patients come in, they are managed fully by our residents, not off-service consultants competing to get their share of procedures. We are a certified advanced stroke center and a STEMI center with emergent coronary artery bypass graft available. In our very first year, from July 2018 to June 2019, our first-year EM residents performed more than 100 intubations. That said, we are a very well-resourced hospital; we have all major specialties available in house, and we do not typically stabilize and transfer patients elsewhere. As the main receiving facility for the Wellstar system (11 hospitals in the Atlanta area), we take all comers. This combination of volume and acuity, ED resident independence, and outstanding and advanced clinical resources provides a unique and dynamic learning environment. We also just opened a brand-new emergency department built to handle an eventual volume in excess of 200,000 patients per year.

What unique benefits does your city have to offer?

Kennestone is in a suburb 20 miles north of downtown Atlanta. If you go another 20 or 30 miles outside of Marietta, you get to the North Georgia mountains. We are uniquely positioned between a major metropolitan area with an incredible cultural landscape and easy access to outdoor activities including hiking, tubing, and mountain biking. Marietta is a small community, but it has a variety of neat neighborhoods. The diversity of patients we see is pretty remarkable.

What is an interesting fact about your city?

Atlanta is one of the fastest-growing and most rapidly diversifying cities in the country. Insiders know about our amazing dining scene as well as a world-class entertainment culture.

What is an interesting fact about your program?

We recruit residents from all over the country, and many have fascinating pasts—some are on second (or even third) careers. We have former professional musicians, presidential aides, and many others with fascinating stories. We pride ourselves on our diversity and look for candidates who bring something unique to the program!

—Ted Stettner, MD, emergency medicine residency program director

IN THE NEWS

Pediatric Emergency Doctors Absent from Rural America

by RONNIE COHEN

Vast regions of the rural United States and three states have no pediatric emergency specialists, a new study shows.[1]

The number of clinically active physicians who are board certified in both pediatrics and emergency medicine nearly doubled to 2,403 from 2008 to 2020, the study found. But they worked almost exclusively in urban areas, and not even one was practicing in Montana, South Dakota, or Wyoming last year.

Christopher Bennett, MD, MA, the study's lead author and assistant professor of emergency medicine at Stanford University in California, described the absence of a single pediatric emergency physician in three states and hardly any in a band of states from North Dakota to Texas as "a little startling." Parents sometimes have to drive hundreds of miles or more to be with a critically ill child who was helicoptered to a medical center, he said.

"It is a situation that does require a fresh look given that it's not great now, and in the coming years it will likely be worse," Dr. Bennett told Reuters Health in a phone interview.

Dr. Bennett and colleagues at Stanford and at Harvard Medical School in Boston examined the American Medical Association Physician Masterfile database on March 11, 2020. They found that pediatric emergency physicians in rural areas were significantly older than those in urban areas, likely compounding the problem when the limited rural workforce retires.

While roughly one-quarter of patients seen in U.S. emergency departments are children, pediatric emergency physicians comprise less than 5 percent of the emergency medicine workforce, Dr. Bennett's team reported. More than 85 percent of the 30 million children who visit emergency departments are seen in facilities designed to treat adults.

Moreover, nearly one-fourth of the doctors working in emergency departments had no form of emergency board certification.

Training in pediatric emergency care began in the 1980s, according to an accompanying commentary by Gillian Schmitz, MD, FACEP, President-Elect of the ACEP.[2] Residency-trained emergency physicians are capable of caring for children in emergency settings, she says, but few practice in rural areas.

It would be impractical for a pediatric emergency medicine physician to staff every emergency department, Dr. Schmitz said. "Future models of health care delivery will need to embrace new ways of providing access, specialty consultation, and emergency care for pediatric patients," she writes.

References

1. Bennett CL, Espinola JA, Sullivan AF, et al. Evaluation of the 2020 Pediatric Emergency Physician Workforce in the US. *JAMA Netw Open*. 2021;4(5):e2110084.
2. Schmitz GR. Addressing shortages in pediatric emergency care-the evolution of health care access. *JAMA Netw Open*. 2021;4(5):e2110115.

view the recommended medical treatments for patients requiring end-of-life comfort care in the emergency department.

When choosing medications to address symptomatic care in the acutely dying patient, emergency physicians should take into account half-life, dosing, route, and onset of action. It is important to consider whether the patient has already been receiving these medications prior to arrival; if not, multiple doses may be necessary to achieve symptom relief. The intravenous route is preferred, as it is easily titratable with a fast onset. However, buccal, nasal, subcutaneous, oral, and intramuscular routes are also frequently acceptable.[2–4] See Table 1 for a summary of palliative medical management options for various symptoms.

The Case

A 56-year-old male with end-stage pancreatic cancer is brought to your emergency department after his partner was unable to control his symptoms with his prescribed pain regimen. He arrives complaining of diffuse abdominal pain and shortness of breath.

Vitals:

- Temperature: 98º F
- Heart rate: 105
- Blood pressure: 110/55
- Respiratory rate: 29
- Oxygen saturation: 95 percent

Physical exam is significant for a distended abdomen with diffuse moderate abdominal tenderness with nausea. He is short of breath. He and his partner understand that his cancer is terminal and are ready for home hospice. Hospice staff will arrive in the morning, but the pain and discomfort are unbearable now.

How will you provide aggressive palliative resuscitation in the emergency department until the hospice team arrives in the morning?

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Dyspnea

Dyspnea is prevalent in terminally ill patients and is a frequently distressing symptom that drives patients to the emergency department at the end of life. **Opioids are the first-line treatment for palliation of dyspnea.**[5] They decrease the chemoreceptor response to hypercapnia, thereby depressing the central respiratory drive and mitigating anxiety.[5] In the opioid-naive patient, low doses of oral or IV morphine can provide relief. In patients who are already taking opioids regularly, administer 5 percent of their total daily morphine dose to manage dyspnea.[2–4] Oxycodone or hydromorphone can also be used, particularly in patients with a morphine allergy.

Other nonpharmacological alternatives include positioning a fan to blow cool air toward the face, repositioning the patient for better oxygenation, and administering supplemental oxygen.[2–4] Admission to the hospital or hospice inpatient unit should be considered when a patient is experiencing refractory dyspnea despite initial palliative resuscitation with appropriate medications, alternative medical interventions, and the ruling out of any reversible causes of dyspnea. Management of a terminally ill patient with refractory symptoms should be escalated to a palliative physician for reassessment of end-of-life care in an acute setting.

Pain

Opioids are also the principal treatment for pain in terminally ill patients. Intravenous administration of opioids is generally best for controlling severe nociceptive pain that is new or escalating, whereas oral medication can be better for chronic mild or moderate pain if the patient tolerates oral administration.[2–4] Given the rapid peak plasma concentration (six minutes) and short half-life of IV opioids, patients should be reassessed every 15 minutes for repeated doses. The half-life of morphine and hydromorphone is approximately two hours. The duration of action of fentanyl is only 30 minutes to an hour.[6]

When assessing a patient who presents to the emergency department with an acute pain exacerbation despite the use of their home medications, emergency physicians should calculate the 24-hour dose of their prescribed opioids and use an equianalgesic dosage conversion calculator so the patient is appropriately medicated. When converting from an oral morphine equivalent to another opioid in a patient with a high opioid requirement, emergency physicians should reduce the total dosage of the new opioid medication by 25 to 50 percent. Be cautious in terminally ill patients who may have renal impairment and avoid morphine, as this can lead to opioid-induced neurotoxicity. Use hydromorphone or fentanyl as the opioid of choice if there is known renal impairment.

Infusions Versus Push Dose IV

The dose of opioids for pain management in the dying patient should be increased by 50 to 100 percent regardless of the starting dose until pain is adequately controlled. Infusions are less effective in the short-term emergency setting, as they require four to five half-lives to reach steady plasma concentration, which can

Table 1: Palliative Medical Management of Symptoms in the Acutely Dying Patient

Symptom	Drug	Dose	Tips
Pain	Opioids	**Morphine** Initial bolus: 4 mg IV x 1 AND IV bolus q15 minutes PRN **Hydromorphone** Initial bolus: 1 mg IV x 1 AND 1 mg IV bolus q15 minutes PRN **Fentanyl** Initial bolus: 25 mcg IV x 1 AND 25 mcg IV bolus q15 minutes PRN	If pain is unrelieved for 30 minutes, increase bolus dose by 50–100%. Repeat q30 minutes as necessary. If patient is not opioid-naive, give 50% of usual breakthrough pain dose or up to 20% of total daily dose.
Dyspnea	Opioids		If patient is not opioid-naive, give 5% of total daily morphine dose.
Nausea/Vomiting	Dopamine antagonist 5-HT3 antagonist Dopamine antagonist Dopamine antagonist	Metoclopramide 10 mg q4 hours Ondansetron 4 mg q4 hours Haloperidol 0.5 mg q6 hours Chlorpromazine, prochlorperazine 10 mg q4 hours	
Anxiety/Agitation/Delirium		Haloperidol 1 mg IVP q1 hour PRN Lorazepam 1 mg PO or SQ q1 hour PRN	SL>IV>SQ Monitor for paradoxical agitation
Terminal Secretions		Glycopyrrolate 0.1 mg IV or 0.2 mg PO	Prevents new secretions. Reduces respiratory gurgling. Does not cross blood-brain barrier compared to other agents. Decreased risk of delirium compared to the use of atropine or scopolamine.

take up to 10 hours.[6] Bolus doses outlined in Table 1 are recommended, and infusions can be considered as a supplement if long-term pain management is necessary.

The Principle of Double Effect

The "principle of double effect" is an ethical doctrine that indicates that an action with both intended and unintended outcomes is justified if the intended benefit significantly outweighs the unintended harm.[7] This has historically been applicable to the concern that opioids may depress respiratory drive and therefore accelerate the dying process. While the use of opioids is widely considered justified by the principle of double effect, there is evidence that opioids do not actually hasten death when used appropriately for pain in the actively dying patient, particularly at low doses.[8–10] One study found that opioid use titrated to comfort in a palliative setting does not significantly alter $PaCO_2$, PaO_2, or overall survival. It does, however, manage pain and reduce dyspnea, thereby significantly increasing comfort at the end of life.[1,9,10]

One notable exception of the double effect is in conscious patients with imminent airway loss. These patients require rapid and large boluses of opioids or benzodiazepines, propofol, ketamine, barbiturate, etc. The ultimate result would be palliative sedation, which is necessary in this instance to mitigate suffering from significant dyspnea.[8]

Nausea

The pathophysiology underlying nausea in a dying patient is often multifactorial and can include drugs, organ failure, metabolic disorder, obstruction, constipation, gastroparesis, inflammation, and tumors, to name a few. Nausea is usually treated based on suspected etiology; however, empiric antiemetic therapy has been shown to be equally effective.[11,12]

Secretions

The "death rattle" refers to the sound that is made when air passes through a dying patient's pooled secretions within the posterior oropharynx. The rattle is not harmful to the dying patient but is often the most distressing symptom to the family. Proper positioning can facilitate drainage of secretions, but atropine and glycopyrrolate (a quaternary amine that does not cross the blood-brain barrier and therefore does not contribute to terminal delirium) are appropriate drugs in the acute setting to decrease secretions and mitigate noisy respiration.[5] **Aggressive suctioning should be avoided in the dying patient** as comfort of the patient is the goal.

Anxiety, Agitation, and Delirium

Haloperidol has the best data in treating agitation or delirium in this patient population. Droperidol may also be effective, but there is less evidence to support its use. Benzodiazepines can cause paradoxical agitation in elderly patients and should be used as a second-line option with close monitoring of the patient.[5,8] If terminal delirium is refractory to initial medications, consult a palliative physician to assist with palliative sedation.

Case Conclusion

You are able to find a quiet room for the patient and his family. You place a nasal cannula for comfort and review the patient's opioid home regimen as 60 mg extended-release morphine sulfate BID with a rescue dose of 20 mg every four hours for a total oral morphine equivalent of 240 mg that can be converted to 80 mg (3:1 PO:parenteral) in a 24-hour period. Because the patient is not opioid-naive, you give his typical home dose with a 50 percent reduction (10 mg IV) and write for an additional 50 percent of his breakthrough pain dose (3 mg IV) to be given every 30 minutes PRN for severe pain. This dose of opioids will also help manage his dyspnea. For nausea, you select ondansetron 4 mg. Physicians should use an online opioid conversion calculator, such as https://clincalc.com/opioids or https://opioidcalculator.practicalpainmanagement.com, or consult their ED pharmacist when converting oral to IV opioid formulations.

After your interventions, the patient appears much more calm and comfortable. His heart and respiratory rates come down. The patient dies comfortably with family at bedside less than an hour later. They are very appreciative of the care and support provided by the emergency team.

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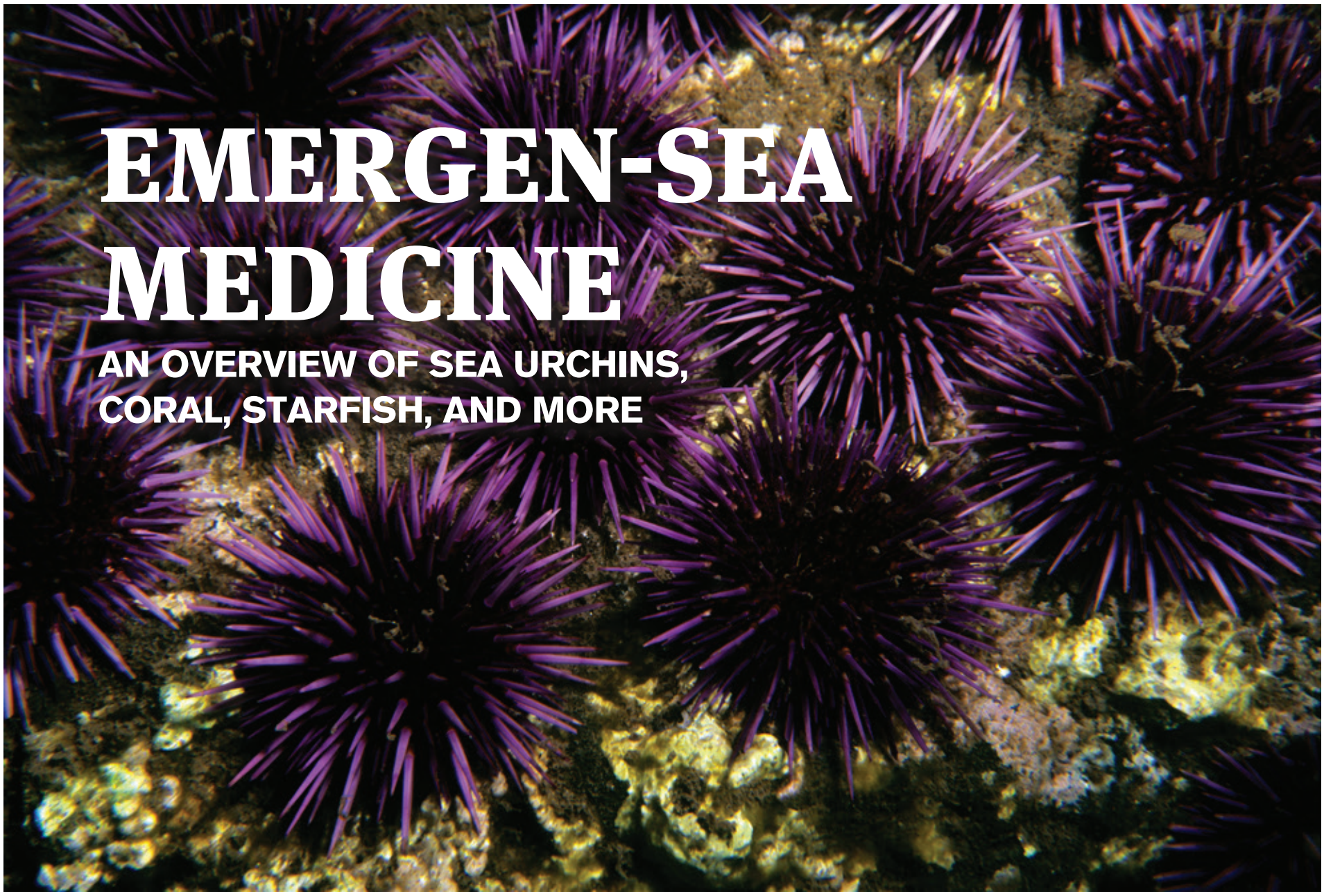

GETTY IMAGES

EMERGEN-SEA MEDICINE

AN OVERVIEW OF SEA URCHINS, CORAL, STARFISH, AND MORE

by CHRISTOPHER HAUGLID, DO; JOHN KIEL DO, MPH; AND ANDREW SCHMIDT, DO

Editor's Note: This is Part 2 of a 3-part series on managing marine envenomations. Last month we reviewed some general tips and strategies for jellyfish stings. In August, we'll examine other chordates.

PHYLUM: CNIDARIA

Class: Cubozoa (Cubozoa species produce the highest morbidity and mortality of all Cnidaria.[1])

Sea Wasp or Marine Stinger *(Chironex fleckeri)*

Location: Indo-Pacific Ocean, southeast United States (rare)

Epidemiology: Ninety-two percent of stings occur during "stinger season" (Australian summer, Oct. 1–June 1) between 3 and 6 p.m., with 83 percent occurring in <1 m of water.[2]

Appearance: The sea wasp has a large bell (body) measuring an average of 25 to 30 cm in diameter. Each of its four corners contains about 15 tentacles that contain millions of "stinging cells" called nematocysts (ie, cnidocytes) and can measure up to 3 m in length.[1]

Pathophysiology and Symptoms: Contact with the tentacles causes rapid onset of a pruritic/burning, erythematous maculopapular rash with a characteristic "ladder-rung" pattern. Severe pain is the most common complaint and can last for several hours due to sustained muscle contractions caused by myotoxins within the venom.[3] This can result in rhabdomyolysis, depending on the duration and intensity of the contractions. Although uncommon, cardiovascular collapse can occur due to a combination of dysrhythmias (due to hyperkalemia) and osmotic dysregulation of endothelial and cardiac tissues from pore-forming toxins within the venom.[4] This ultimately leads to cardiogenic pulmonary edema, severe hypotension, and death in as few as 30 seconds.[5] Additional complications include altered mentation, dizziness, ataxia, and hemolysis.

Management: Pain should be managed with vinegar, hot water immersion, local lidocaine (infiltration or topical), and/or Stingose solution (pain relief has been shown within five seconds of application).[6] Compressive dressings are controversial and recent recommendations are *against* their use in the management of all Cnidaria envenomations due to the demonstrated increase in venom release from nematocysts.[1,7,,8] Obtain an ECG, serial cardiac biomarkers, and chemistry panel to assess for electrolyte abnormalities. Box jellyfish antivenom, when available, has been shown to prevent all toxicity in animal studies when administered prophylactically.[9] However, current beachside (ie, Surf Life Saving Australia's beach lifeguard group) dosing recommendations of three vials intramuscularly at three separate sites may be too small a dose and too slow a route. Early IV administration of antivenom in large doses—initial treatment of one to three vials diluted 1:10 with saline, along with IV magnesium sulfate (0.2 mmol/kg, max 10 mmol in adults) bolused over 5–15 mins—is recommended if there are any signs of severe toxicity, intractable pain, or cardiac arrest. But even this may be too small a dose, and further research is needed.[10,11] The current maximum dose of antivenom is six vials undiluted and should be given via rapid IV push if the patient is in cardiac arrest.[10]

Caveats: Contrary to previous studies in mice, verapamil has been shown to be ineffective and possibly detrimental; it exacerbated cardiovascular symptoms and increased mortality in pigs.[8,9,12–15] Felodipine and magnesium sulfate may actually worsen the effects of the venom, but further research is needed. *C. fleckeri* stings can be deadly, however, recent data suggest the fatality rate is actually much lower than the roughly 20 percent that was previously reported.[5,12,16] Morbidity and mortality are dose- and time-dependent, which explains why the majority of deaths have been in children.[11,17] Avoid treating with fresh water, alcohol, methylated spirits, and urine, as these have also been shown to increase nematocyst firing.[18]

Class: Hydrozoa

Fire Corals *(Millepora alcicornis)*

Location: Worldwide (excluding Hawaii) in reefs and shallow waters

Appearance: White to yellow-green seaweed-like growths fixed to rocks and coral. They possess tentacles that extend upward and are roughly 2 m in length.

Pathophysiology and Symptoms: Contact with tentacles causes painful, urticarial lesions that may become hemorrhagic and ulcerate. Symptoms usually resolve within 90 minutes, but they can last up to 72 hours with skin hyperpigmentation that can last several weeks. Rarely, patients will present with mild systemic symptoms (eg, nausea, vomiting, myalgias, dyspnea, anxiety, abdominal pain, headaches, etc.).

Management: Pain is best managed with vinegar. Steroid creams and oral antihistamines can be used for mild urticaria. If severe, oral steroids may be warranted.

Class: Anthozoa

Sea Anemones

Location: Worldwide in deep and coastal waters, often attached to coral or rock

Appearance: Anemones vary in appearance. Most are a single polyp with a cylindrical body. Their mouths are surrounded by cnidocyte-containing tentacles.

Pathophysiology and Symptoms: Anemone venom contains multiple enzymes including cytolytic/hemolytic toxins, neurotoxins, cardiotoxins, and protease inhibitors, which cause symptoms ranging from erythema, pruritis, and blisters to fevers, chills, fatigue, myalgias, and syncope. Skin changes can become permanent in the form of hyper-/hypopigmentation and keloid formation.[19]

Management: Pain is managed with vinegar. Other symptoms are managed with supportive care.

PHYLUM: ECHINODERMATA

Class: Echinoidea

Sea Urchins

Location: Worldwide in both shallow and deep waters

Appearance: Composed of spherical, hard shells called

"tests" that measure up to 4–5 inches in diameter which are covered in calcified spines. Venom is contained within these spines, as well as their pedicellarie (ie, pincers), which are more difficult to remove from human skin and contain more venom.

Pathophysiology and Symptoms: Contact causes an erythematous rash with localized burning, pruritis, myalgias (lasting approximately 24 hours), and edema. Symptoms rarely progress to nausea, vomiting, paresthesias, weakness, abdominal pain, hypotension, and syncope. Spines commonly break off, causing hyperpigmentation of the skin, and can lead to granuloma formation, secondary infection, and synovitis if the joint is involved.

Management: Pain control is generally the biggest concern with these injuries and is best achieved with hot-water immersion and local lidocaine. Attempts to remove spines are often futile. The spines are very fragile and tend to crumble in the skin. To further complicate the removal process, areas where no spine remains may still have the appearance of a foreign body from "tattooing" of the skin. Operative exploration should be considered if there is joint involvement. Granulomas may also need surgical exploration because spines often crumble and are hard to find.

Class: Asteroidea

Crown-of-Thorns Starfish *(Acanthaster planci)*

Location: Indo-Pacific Ocean, Red Sea, east coast of Africa, and west coast of Central America

Appearance: A central disk with radiating arms (usually more than 15–20 arms), densely covered with spines. Adults are often dull brown to green colored (although some have bright colors to warn predators) and normally range from 9–14 inches in diameter.

Pathophysiology and Symptoms: Spines pierce the skin and cause severe pain (usually lasting ≤3 hours) with local inflammation. The spines are coated with a slime that is extremely toxic and, in severe cases, can cause paralysis, hemolysis, and hepatotoxicity. Additional symptoms include paresthesias, nausea, vomiting, and secondary infection.

Management: Same as Echinoidea/sea urchins (see above).

PHYLUM: PORIFERA

Class: Demospongiae

Fire Sponge *(Tedania ignis)*, Poison-Bun Sponge or Touch-Me-Not Sponge *(Neofibularia nolitangere)*, Red Moss or Red Beard Sponge *(Clathria prolifera)*, and Australian Stinging Sponge *(Neofibularia mordens)*

Location: Worldwide

Pathophysiology and Symptoms: These sponges produce crinotoxins (dermal irritants) that cause a maculopapular rash with local edema, bullae formation, paresthesias, and possible joint swelling that resolves spontaneously in approximately seven days. More severe reactions may cause fevers, chills, fatigue, nausea, and myalgias with delayed immunologic responses that manifest as erythema multiforme or dyshidrotic eczema. Additionally, sponges may also be colonized with Cnidaria species and cause a necrotic skin reaction (ie, sponge divers' disease).[20] *Note:* Rewetting a dried sponge can cause it to regain its toxicity, even after several years.

Management: Control pain with vinegar. Topical steroids and oral anthistamines are used for mild symptoms, and oral steroids for erythema multiforme or dyshidrotic eczema.[20–22]

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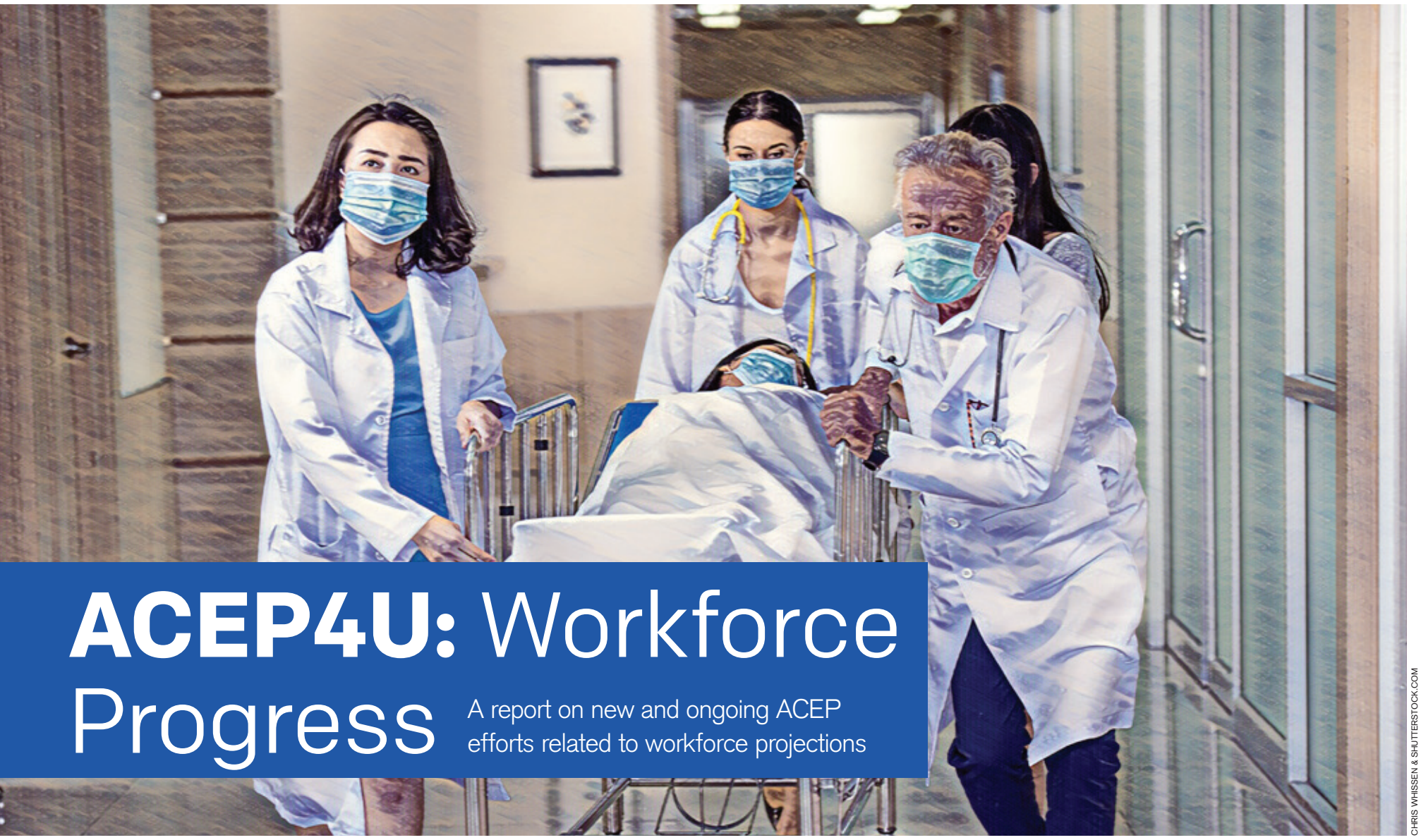

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ACEP4U: Workforce Progress

A report on new and ongoing ACEP efforts related to workforce projections

As we await publication of the "Emergency Medicine Physician Workforce: Projections for 2030" research, ACEP is leading efforts down many important paths to favorably influence the future of the EM workforce.

Define EM Residency Standards for the Future

- ACEP met in early June with the Association of Academic Chairs of Emergency Medicine, Emergency Medicine Residents' Association, Council of Emergency Medicine Residency Directors, American College of Osteopathic Emergency Physicians, Society for Academic Emergency Medicine (SAEM), and SAEM Residents and Medical Students to begin reviewing and developing recommended program requirement changes to the Accreditation Council for Graduate Medical Education (ACGME) for the 2022 review cycle.
- The stakeholder representatives looking at the ACGME guidelines will meet every two weeks. ACEP President Mark S. Rosenberg, DO, MBA, FACEP, appointed members including Kelly Gray-Eurom, MD, MMM, FACEP; Christopher S. Sampson, MD, FACEP; and Laura Oh, MD, FACEP, to this effort.

Ensure Business Interests Do Not Supersede Education and Patient Care

There are pressures to start residency programs from for-profit hospitals, nonprofit hospitals, and academic health systems. There are also some state laws that reward expansion of graduate medical education.

- ACEP leaders will reach out to hospitals, physician groups, and other stakeholders to set up meetings to ensure they understand the history and growth of graduate medical education, the research from the Workforce Task Force about the potential future impact on the EM workforce, and the role the groups and health systems play.
- Discussions also include legal, legislative, and regulatory options that tie Centers for Medicare & Medicaid Services (CMS) funding of graduate medical education to specialties and geographic areas of need as well as how ACEP might be able to encourage needed changes. Findings will be reported.
- An objective has been added to the Academic Affairs Committee to conduct research with residency programs with respect to employment models, funding sources, staffing models, physician assistant (PA)/nurse practitioner (NP) training programs, and policies for prioritizing critical care to EM residents.

Protect Unique Role of the Emergency Physician and Fight Independent Practice

ACEP believes that emergency physicians provide the highest-quality care of patients with undifferentiated illnesses. We fight for physician-led teams and robust guardrails to maintain the scope of practice for NP/PAs.

- ACEP recently became a member of the American Medical Association (AMA) Scope of Practice Partnership (SOPP) and continues to attend regular meetings to leverage the house of medicine on ways to ensure physician-led teams nationwide as well as to utilize and promote shared resources among chapters.
- In early June, a proposal in Louisiana that would have allowed NPs to have independent practice was withdrawn with the help of the Louisiana ACEP chapter and its members. National ACEP is working to support other chapters facing similar threats of scope creep and other states, including Texas and Colorado, with an eye toward similar victories in those state legislatures.
- Alarmed by the implications of independent practice for PAs, ACEP vehemently opposed the decision by the American Academy of Physician Assistants to change its professional title to physician "associate." ACEP issued a statement denouncing the name change and advocated that the AMA address this through its SOPP efforts, including its truth in advertising campaign.
- ACEP President Dr. Rosenberg established a task force to research and potentially establish an ED accreditation program that would define nationally recognized standards to provide the highest-quality patient care. Joseph Adrian Tyndall, MD, FACEP, was named the chair of this task force, which will meet regularly and, by ACEP21 in Boston, will offer a proposed direction about pursing an accreditation program.

Support Emergency Physicians in All Communities, Especially Rural

- ACEP leaders have met with CMS staff to problem-solve rural hospital closings and will continue to explore ways to ensure access to quality emergency care, led by emergency physicians, no matter a patient's ZIP code.
- The Rural Emergency Medicine Task Force report closely aligns with efforts to influence change in the EM workforce of the future. ACEP leaders are working with the Rural EM Task Force members and the Rural EM Section members to ensure our next steps are coordinated.
- ACEP is compiling and will promote a list of current opportunities within the federal government, Indian Health Service, Veterans Affairs, and other locations that have incentives for hiring emergency physicians to practice in rural and underserved areas.
- To get better data-driven research, ACEP is helping to conduct a resident census about job availability and challenges year over year. This survey should be distributed within a few weeks and could become an annual effort to provide a sense of the hiring landscape over the long-term.

Increase Demand and Meet Evolving Needs of Our Communities

We recognize emergency physicians' skill sets are not defined by a physical location. Emergency medicine will continue to evolve to meet the changing health care landscape and delivery models as we emphasize our value, expertise, and necessity.

- ACEP leaders continue to explore adding fellowships to expand emergency medicine's brand. Successful models are being considered for replication. Potential areas for growth include geriatrics, hospice and palliative care, home health monitoring and post-acute care, disaster medicine, administrative medicine/executive leadership, and more. Recommendations are forthcoming.
- ACEP is continuing to gather member profiles to highlight those who have successfully expanded their roles, with a goal to crowdsource and research ways to bring nontraditional EM practice areas (observation, acute psychiatric, EMS, telehealth) under the EM umbrella as viable career pathways.

Identifying Patients with High COVID-19 Risk in North Carolina

Q&A with EMF grantee Dr. John D. Purakal

Since its launch in 1972, the Emergency Medicine Foundation (EMF) (www.emfoundation.org) has awarded grants totaling more than $17 million to support research projects developed to improve and advance the field of emergency medicine. Recently, the organization awarded $163,967 in grants to fund four active COVID-19 research projects led or co-led by U.S.-based emergency physicians.

Among these grantees is John D. Purakal, MD, MSc, assistant professor of emergency medicine at Duke University School of Medicine in Durham, North Carolina. Dr. Purakal received a $50,000 COVID-19 research grant award from the EMF to support his project "Social Determinants of Health and COVID-19 Infection in North Carolina: A Geospatial and Qualitative Analysis." To learn more, we spoke with Dr. Purakal about the impetus behind the project and the short- and long-term goals that he and his team set out to achieve.

Dr. John D. Purakal

EMF: Tell us about your EMF grant project.

JP: This project is designed to identify the highest at-risk patients in areas surrounding our hospital system so we can target our education and resources and develop a community-based strategy to address these patients' needs. First, we look at particular census track data from the North Carolina Department of Health and Human Services and the maps showing where these social determinants of health are most disproportionate or affecting people the most. Second, we create a geospatial map of our COVID-19 outbreaks. Third, we put the two maps together and figure out which of the social determinants of health are most highly associated with outbreaks and may be contributing to COVID-19 clusters or hot spots. So, in the first part of our study we create a map of hot spots or clusters to help us identify patients in the highest-risk areas.

On the back end though, we explore this further with qualitative analysis or interviews with those identified patients to learn about their personal experience with COVID-19. In one of our early interviews, the patient said that only when they tested positive did they start talking about COVID-19 with others in their community. It took them coming forward first before anybody else would share their information. We can see how that would affect disease spread—asymptomatic spread especially—if people don't feel the need to share their information until other people talk about it. We're hoping to identify more such interesting findings through our interviews.

EMF: Why did you choose this topic?

JP: I'm interested in social emergency medicine, social determinants of health, and how they impact the patients that present to the emergency department. We know that minority patients and underserved patients live disproportionately in areas with high social determinants of health that impact their day-to-day life and their health outcomes. This project makes sense to me because it takes an acute need—the novel studying of a virus that's happening in real time—and identifies problems utilizing information available here in North Carolina to determine which areas are of highest risk. In so doing, we prepare clinicians to risk-stratify patients from these areas.

EMF: What are the goals of your research?

JP: Our short-term goals for this project are to identify COVID-19 outbreak areas in our community and in the patients that we serve at Duke University and to determine which social determinants of health are most frequently or highly associated with those outbreaks. Long term, we're hoping that this information will provide a blueprint for health care workers and for community organizers, as well as state or local government officials, so they can be more prepared for future pandemics and other outbreaks that may require education, community resources, and community-specific approaches. We will know which areas might be hardest hit sooner and be able to target them right away so these outbreaks among people who may be underinsured or underserved don't happen at such fast or disproportionate rates. By looking at transportation needs, socioeconomic needs, and the like, we can identify patients who would be highest risk if we sent them home with a positive COVID test and/or be able to better inform them about having family members tested, social distancing, and things of that nature.

EMF: How did your EMF grant help advance your career in emergency medicine?

JP: It means the world to me that EMF believed in our team's project and vision. It's especially impressive to me that, during an unfolding pandemic, EMF was able to provide additional funding for people who were interested in exploring it in real time.

EMF grants are essential for researchers at all stages in their careers—without them, the field of emergency medicine would advance at a much slower pace. Everyone needs to start somewhere, and I think that this grant is going to help me get my foot in the door in terms of conducting research at a higher level. I'm very appreciative of this opportunity and for the faith that EMF has shown in our team, my mentors, and collaborators to accomplish this project. We're very grateful that the organization was able to help us fund our project and others like ours, as we all work to improve and advance the field of emergency medicine.

By the Numbers

Overdose Cardiac Arrests

IN 2020

42%

increase in EMS-observed overdose cardiac arrests nationally compared to baseline of 42 per 100,000 EMS calls (average of 2018 and 2019)

INCREASES BY RACE

50%
for Black patients

50%
for Latinx patients

38%
for White patients

INCREASES BY ZIP CODE TYPE

44%
High-poverty ZIP codes

46%
Medium-poverty ZIP codes

31%
Low-poverty ZIP codes

THE PACIFIC REGION HAD THE LARGEST INCREASE AT 64%, WHILE NEW ENGLAND DECREASED BY 4%

INCREASES WERE HIGHEST IN MAY 2020 (98%) AND LOWEST IN DECEMBER 2020 (21%)

Compiled by Joseph Friedman, MPH, an MD/PhD trainee at UCLA Center for Social Medicine and UCLA Medical Informatics PhD program in Los Angeles.

Visit **ACEPNow.com** for the sources of these statistics.

Working Up Double Vision

How to evaluate diplopia and spot life-threatening etiologies

CHRIS WHISSEN & SHUTTERSTOCK.COM

by JONATHAN STRONG, MD, MPH

Diplopia, colloquially referred to as "double vision," is a challenging chief complaint in the emergency department. The etiologies are vast. Acute life-threatening causes include stroke, aneurysm, or increased intracranial pressure. Less urgent chronic causes can include decompensated phoria (ie, misalignment of the eyes) or slowly evolving microvascular disease. Life- and organ-threatening causes should be the top concern in the emergency department, and emergency physicians must be prepared to identify any related conditions.[1]

Clinicians must first determine whether the patient has monocular or binocular diplopia. Monocular diplopia is confirmed when covering the opposite eye fails to resolve the diplopia. This strongly suggests etiologies that are nonemergent but should be referred for outpatient ophthalmologic evaluation. (Note: Some exceptions such as multiple sclerosis, seizures, or various forms of encephalopathy will be apparent by other aspects of the patient's presentation.)[2] The remainder of this article discusses binocular diplopia (henceforth referred to as diplopia), which occurs due to misalignment of the visual axes.

Neuroanatomy

A brief review of the neuroanatomy of eye movement is helpful to recognize patterns of extraocular muscle weakness, identify cranial nerve (CN) palsies, and localize structural lesions. Proper eye alignment and movement depend on extraocular muscles innervated by CNs III, IV, and VI (see Table 1 and Figure 1). These CNs originate in the midbrain and pons, course through the subarachnoid space near important structures such as the posterior communicating artery, travel through the cavernous sinus, enter the orbit through the superior orbital fissure, and finally innervate their target extraocular muscle(s) at the neuromuscular junction. Pathology at any of these locations may cause diplopia.

A complete CN III palsy impairs eye supraduction (vertical upward glance), infraduction (vertical downward glance), and adduction. This causes the eye to rest in a "down and out" position. An incomplete CN III palsy will present with varying degrees of weakness in each of the affected extraocular muscles. Additionally, CN III contains motor fibers that control the levator muscle of the eyelid and parasympathetic fibers located on the periphery of the nerve trunk that control pupil constriction. Compressive lesions such as aneurysms will usually (but not always) result in pupil dilation with some degree of oculomotor dysfunction and/or ptosis.

CN IV palsy causes upward deviation and rotational misalignment; however, this can be difficult to identify due to the compensatory effect of other intact extraocular muscles. To diagnose an isolated unilateral CN IV palsy, the examiner should assess whether the upward deviation in the affected eye worsens with gaze toward the contralateral side and with the head tilted toward the ipsilateral side. If the exam is not consistent with these findings, one should consider alternative diagnoses.

CN VI palsy is easier to identify: Patients present with outward deviation most apparent when looking toward the affected side. It is important to note that CN VI is particularly susceptible to traction caused by structural lesions or elevated intracranial pressure, a cause particularly suggested by the presence of a bilateral CN VI palsy.

Figure 1: Eye Movements and the Related Cranial Nerves (CNs) and Extraocular Muscles

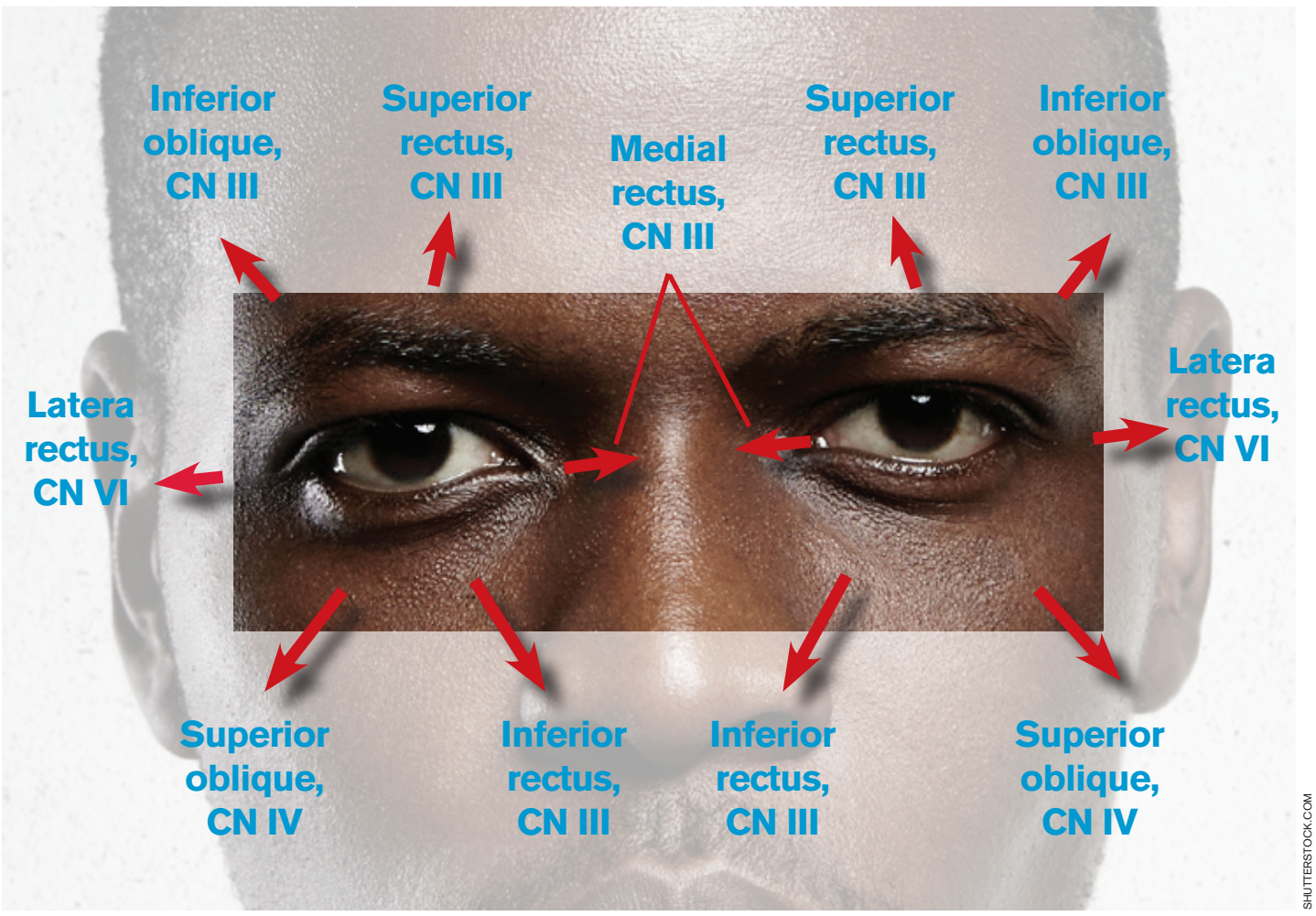

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Table 1: Eye Movements and Corresponding Extraocular Muscles

Eye Movement	Primary Extraocular Muscle	Secondary Extraocular Muscle(s)
Adduction	Medial rectus	Inferior rectus, superior rectus
Abduction	Lateral rectus	Inferior oblique, superior oblique
Supraduction	Superior rectus	Inferior oblique
Infraduction	Inferior rectus	Superior oblique
Intorsion (rotation toward the patient's nose)	Superior oblique	Superior rectus
Extorsion (rotation toward the patient's ear)	Inferior oblique	Inferior rectus

Considerations for the History and Exam

Emergency physicians must first determine whether the patient has isolated diplopia or diplopia with additional neurological deficits suggestive of a posterior circulation stroke. Patients presenting with acute-onset diplopia in combination with altered mental status, bulbar weakness (such as difficulty with speech and swallow, facial muscle use, or emotional volatility), vertigo, or "crossed" brain stem signs (ie, ipsilateral CN deficits with contralateral limb weakness or sensory loss) should be considered to have a brain stem stroke until proven otherwise. The clinician should activate institutional stroke protocols, obtain neurology consultation, and obtain neuroimaging (including an MRI after a screening CT has been obtained) as soon as possible.

After a posterior circulation stroke has been excluded, clinicians should gather a thorough history as well as perform comprehensive neurological and eye exams. It is important to determine the orientation of the diplopia (horizontal, vertical, or oblique) and whether the diplopia worsens in a particular direction of gaze. This will help to identify the extraocular muscles and/or cranial nerves affected. The onset and timing of diplopia can also aid in diagnosis. Intermittent diplopia that worsens with fatigue and later in the day is suggestive of myasthenia gravis. Slowly progressive diplopia may suggest a compressive cause (eg, inflammatory, infectious, neoplastic, or vascular lesions), whereas intermittent diplopia may be due either to a decompensating phoria

or myasthenia gravis.

Pain associated with diplopia can be seen in a variety of conditions. Pain localizing to the eye or retrobulbar region—particularly pain that worsens with eye movement—suggests pathology of the cavernous sinus, orbit, and/or extraocular muscles such as Tolosa-Hunt syndrome (a condition believed to be caused by inflammation of either the cavernous sinus or the superior orbital fissure), thyroid eye disease, infection, or malignancy. Microvascular ischemia and optic neuritis can also cause pain in a similar distribution. Diplopia accompanied by headache can occur in giant cell arteritis, cavernous sinus thrombosis, pituitary apoplexy (bleeding), intracranial aneurysm, cervical artery dissection, and a variety of other conditions. As always, severe or thunderclap headache suggests aneurysmal subarachnoid hemorrhage, which may be associated with aneurysmal compression of cranial nerves, most commonly CN III.

However, painless diplopia does not exclude serious neurological conditions, such as aneurysms, as pain is an unreliable finding in many conditions.[3] Rarely, giant cell arteritis may present with diplopia in the absence of headache or other systemic symptoms. Accordingly, all patients over the age of 55 with double vision should undergo laboratory screening with erythrocyte sedimentation rate, C-reactive protein, and complete blood count.[4,5]

Internuclear ophthalmoplegia (INO) is an ocular motility disorder of horizontal conjugate gaze that is important to recognize. INO occurs due to a lesion in the medial longitudinal fasciculus of the brain stem, most commonly from multiple sclerosis or ischemic stroke. When a patient with INO attempts to look away from the affected side, there is impaired ipsilateral eye adduction and nystagmus of the contralateral eye on abduction. This may occur with conjugate gaze toward one or both sides. Convergence is usually preserved.

Diplopia occurring after head trauma may be due to decompensated phoria, CN palsies due to stretch injury or skull fracture, intracranial hemorrhage, direct extraocular muscle damage, extraocular muscle entrapment due to orbital fracture, orbital compartment syndrome resulting from a retrobulbar hematoma, or carotid-cavernous fistula. A noncontrast head CT should be the first neuroimaging obtained in trauma patients presenting with diplopia.

The patient's past medical history can also suggest potential causes for diplopia. Patients with a history of malignancy should be suspected to have diplopia due to perineural spread or lesions in the orbit or brain. Patients suffering from chronic alcoholism may develop Wernicke encephalopathy characterized by altered mental status, gait ataxia, and oculomotor dysfunction. A history of cardiovascular risk factors is supportive of microvascular ischemia or vertebrobasilar insufficiency, though the presence of cardiovascular risk factors does not exclude other important causes discussed here.[4,6]

Localization and Suggested Imaging and Workup

Findings that suggest a cortical or brain stem lesion include altered mental status, weakness or numbness in the extremities, ataxia, vertigo, and/or bilateral cranial nerve deficits. MRI of the brain with and without gadolinium should be obtained.

Bilateral cranial nerve deficits can also occur due to pathology in the subarachnoid space such as infectious or carcinomatous meningitis. In addition to neuroimaging, select patients may require lumbar puncture.

In contrast, multiple unilateral cranial nerve deficits suggest a brain stem, cavernous sinus (CNs III, IV, V_1, V_2, VI), or orbit (CNs III, IV, V_1, VI) lesion. Cavernous sinus and orbit lesions may also cause proptosis. MRI with and without gadolinium of the orbit is the preferred imaging modality for orbital lesions. CT or magnetic resonance (MR) venography is helpful for diagnosing cavernous sinus thrombosis, whereas CT or MR angiography is helpful for diagnosing a carotid-cavernous fistula.

All patients with isolated CN III palsy should undergo urgent MR or CT angiography of the brain to assess for intracranial aneurysm. Several studies have demonstrated that the presence of vascular risk factors and/or physical exam findings such as pupil involvement do not reliably distinguish between subacute causes such as microvascular ischemia and serious acute neurological disease.[3,4,6]

Most, if not all, patients with isolated CN IV or VI palsy should also undergo an urgent MRI of the brain with and without gadolinium. Historically, urgent neuroimaging was deferred in selected patients with isolated CN IV or VI palsy lacking high-risk features. However, this practice has been called into question by recent studies that have shown a significant proportion of patients have structural lesions evident on MRI that dictate changes in early management.[4]

Conclusion

Knowing the differential diagnosis and how to distinguish among the diverse causes of diplopia should enhance your confidence in your ability to assess important emergency neurological conditions. A thorough history and exam as well as appropriate neuroimaging and diagnostic testing can clinch the diagnosis, thereby saving a patient's sight and maybe even their life.

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TWI in aVL

Subtle ECG findings evolving to STEMI

by YOUSTINA MICHAEL, DO; BRENDA SOKUP, DO; AND JORDAN JEONG, DO

An inverted T wave occurring in an isolated electrocardiographic region is referred to as an isolated T wave inversion (TWI) or isolated T wave negativity.[1] Such TWIs can occur in healthy individuals with no evidence of heart disease but are also seen in patients with coronary artery disease. In asymptomatic patients without cardiovascular history or risks, isolated TWIs are unlikely to indicate undiagnosed advanced coronary artery disease.

TWIs can be transient. Transient causes include hypokalemia, early stages of hypertrophic cardiomyopathy, hyperventilation, and transient ischemia from coronary vasospasm (people of Asian ethnicity also are more likely to have this latter finding).[1] Even anxiety and fear have been described in association with transient TWI.

However, a study by Farhan et al assessing patients with chronic stable angina demonstrated that TWI in aVL correlated highly with significant left anterior descending artery (LAD) lesions, typically mid segment.[2] Alarmingly, of the 14 percent of ECGs in this study that demonstrated this isolated finding, *all* were read as normal by referring physicians. But this finding should not be viewed as normal in the setting of chest pain, as it may be the first objective finding in an evolving ST-elevation myocardial infarction (STEMI).

Case Report

A 51-year-old man with asthma, hypertension, and hyperlipidemia presented with crushing substernal left-sided chest pain radiating down his left arm that started 10 minutes prior to arrival. He was seen immediately upon arrival by the triage physician, and his initial ECG (obtained at 7:50 p.m.) demonstrated isolated TWI in aVL (see Figure 1). In the setting of significant left-sided chest pain and the ECG finding, the patient was moved to the critical area of emergency department.

In the critical area of the department, the patient was clutching his chest and was diaphoretic. A repeat ECG (obtained at 8:03 p.m.) demonstrated ST elevation in V1–V3 with reciprocal ST depression in II and aVF (see Figure 2).

A "Code STEMI" was initiated. Bedside echo showed preserved left ventricular ejection fraction with akinesia of the antero-septal segment. The initial troponin was negative. Tenecteplase 50 mg was given at 8:16 p.m., with aspirin 325 mg, clopidogrel 300 mg, heparin 4,000 U bolus, and rosuvastatin 40 mg. Next, a heparin 12 U/kg/hr infusion was initiated. A subsequent ECG (obtained at 8:41 p.m) showed greater than 50 percent resolution of the prior V1–V3 ST elevations. The patient reported that his chest pain improved by 8:56 p.m. The patient was transferred to a percutaneous coronary intervention center. Catheterization was completed within 24 hours, demonstrating 99 percent occlusion of the LAD (see Figure 3).

Discussion

An isolated TWI in aVL is not a well-recognized early sign of occlusion across the relevant medical specialties. In a survey by Hassen et al, only 25 percent of physicians identified an isolated TWI in aVL as abnormal, although emergency physicians were better than other specialties at recognition.[3] This study demonstrated that angiograms done specifically during the evaluation for STEMI revealed mid-LAD lesion association with TWI with a sensitivity of nearly 88 percent, and positive predictive value (PPV) of 81 percent for mid-LAD lesions greater than 50 percent. Patients with similar ECG findings who underwent coronary angiography for other reasons demonstrated a sensitivity of 65 percent, PPV of 83 percent, and specificity of 67 percent for mid-LAD lesions 70 percent or greater. The LAD supplies a large portion of the heart and renders a large area of myocardium at risk in the setting of an occlusion, making this subtle ECG change important to identify.

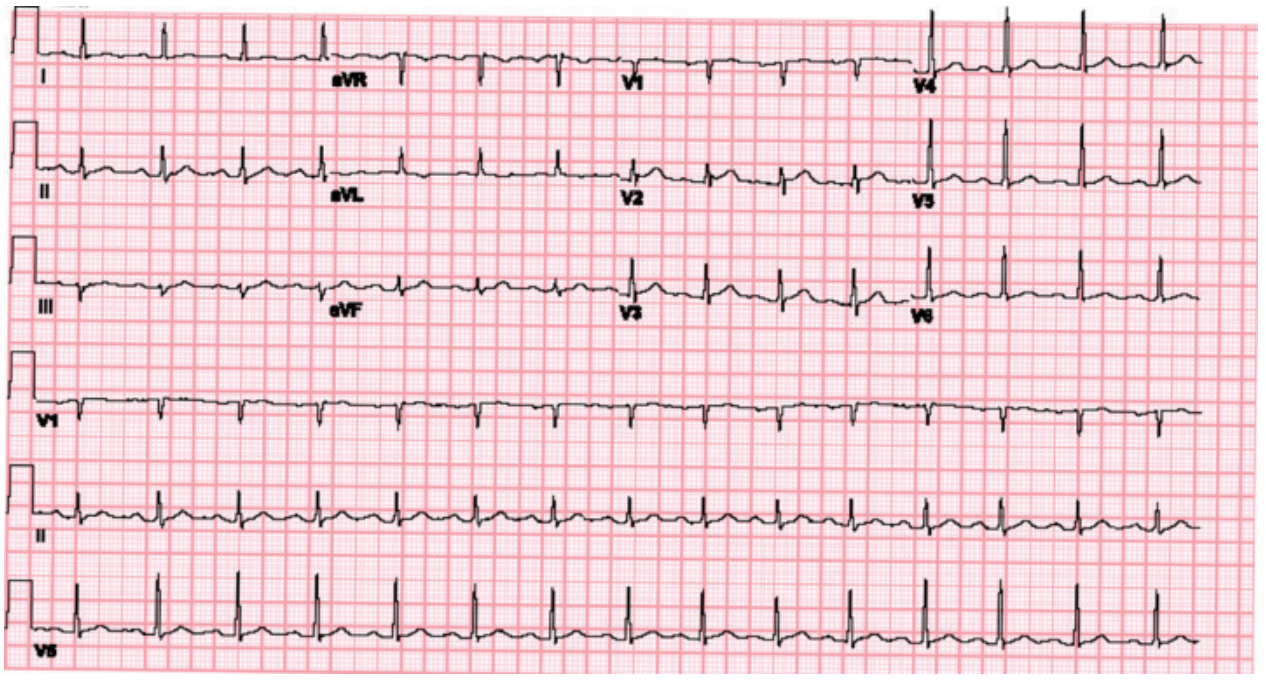

FIGURE 1: The patient's initial ECG demonstrated isolated T wave inversion in aVL.

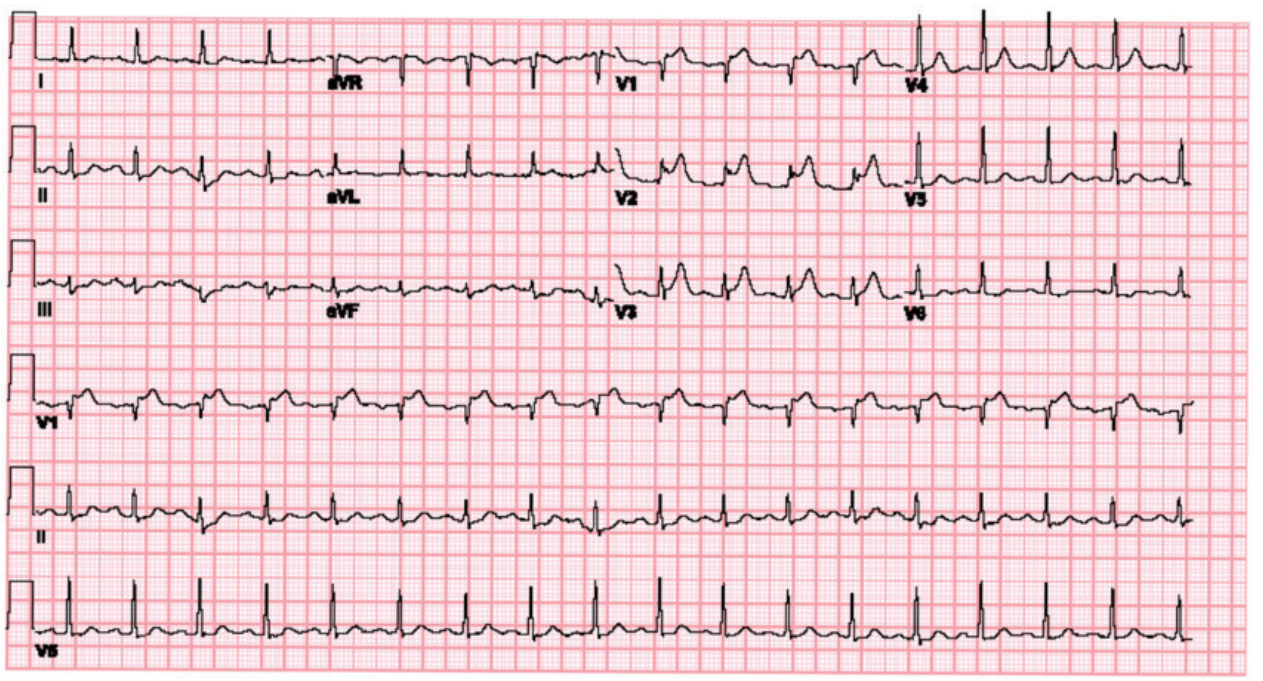

FIGURE 2: The patient's repeat ECG demonstrated ST elevation in V1–V3 with reciprocal ST depression in II and aVF.

For hospitals that do not have an on-site catheterization lab, thrombolytics may be required as a bridge to definitive therapy. ("Time is myocardium.") Patients who receive thrombolytic therapy may have improvement in chest pain and ECG findings. However, these patients require cardiac catheterization within 24 hours of presentation. Early recognition and treatment of patients with concerning presentations and an isolated TWI in aVL may save a life.

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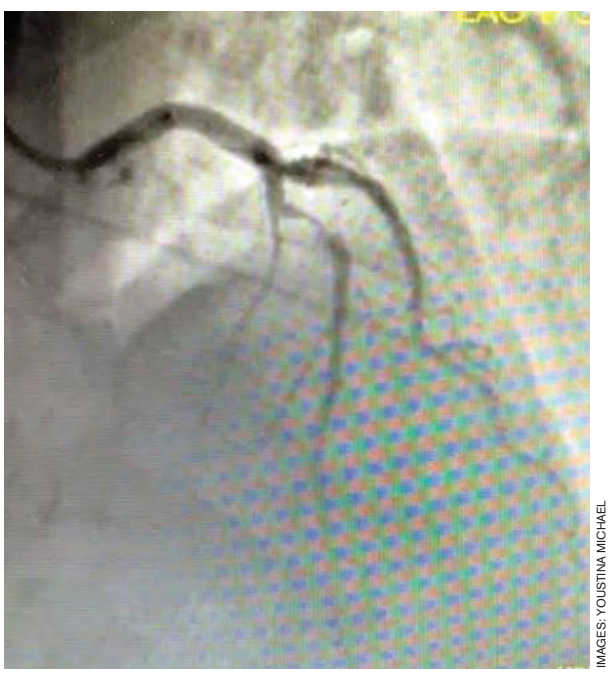

IMAGES: YOUSTINA MICHAEL

FIGURE 3: Catheterization showed 99 percent occlusion of the left anterior descending artery.

BE A MEDICAL DETECTIVE—BONE UP ON YOUR FORENSIC SKILLS

FORENSIC FACTS

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DR. ROZZI is an emergency physician, director of the Forensic Examiner Team at WellSpan York Hospital in York, Pennsylvania, and chair of the Forensic Section of ACEP.

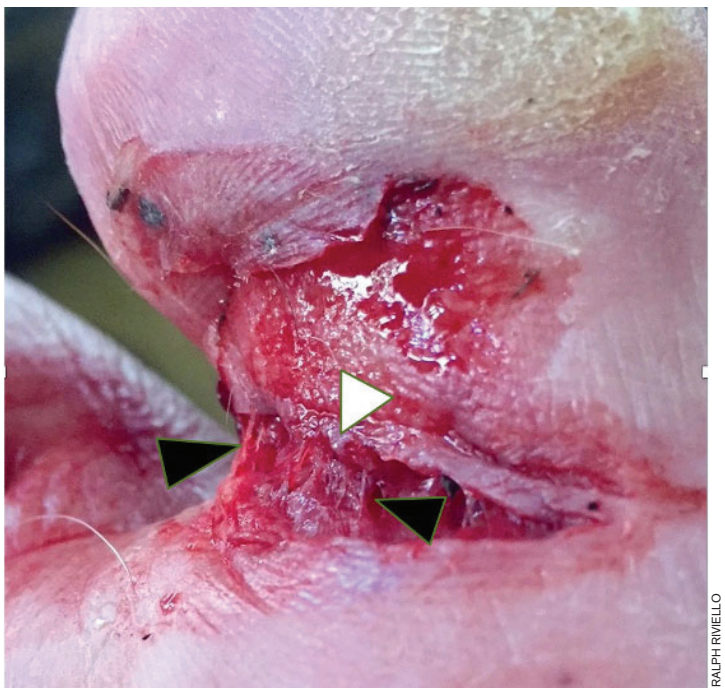
Wound Terminology

Bridges are the clue to correct wound interpretation

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

The Case

A 45-year-old man is brought to the emergency department with a toe injury. The patient states he was at a cookout drinking alcohol when he developed pain in his great toe and noticed bleeding while walking. He stumbled and thinks he stepped on a broken piece of glass. He denies any other injuries or loss of consciousness. He is intoxicated with a Glasgow Coma Score 14–15 (for slight verbal disorientation at the time), is slightly agitated but redirectable, and has stable vital signs. Examination of his toe shows tenderness to palpation without deformity, and there are no signs of neurovascular compromise. His injury is shown in Figure 1. A radiograph is negative for fracture or foreign body. What do you suspect happened?



RALPH RIVIELLO

FIGURE 1: Wound on right great toe depictinged irregular wound edge (white arrowhead) and tissue bridging (black arrowheads).

KEY POINTS

- Not all traumatic open skin injuries are lacerations. Use the correct terminology.
- Lacerations are blunt trauma with irregular, bruised edges; incised wounds or cuts are sharp force trauma with linear, sharp, smooth edges.
- Tissue bridging is seen in the majority of lacerations.
- Comparison of wound length and depth can determine the type of sharp force trauma sustained.
- Wounds should be thoroughly documented including size (measurements), shape, location, and distinguishing characteristics.
- Documentation of wounds should include words, drawings, and images. Digital documentation provides accurate information for future users.

Describing and Classifying Wounds

From a forensic and mechanistic perspective, traumatic injuries can be divided into two categories: blunt trauma and sharp force trauma. Blunt trauma results from an impact with a dull, firm surface or object. Injuries can be patterned (ie, the characteristics of the wound suggest a particular type of blunt object) or nonspecific. Blunt trauma is typically caused by contact with a solid, firm surface (floor or ground) or with an object (such as a fist, bat, or bottle). Sharp force trauma describes an injury produced by an instrument with a thin edge or point (ie, sharp cutting implements).

Blunt trauma injuries include abrasions, bruises/contusions, hematomas, avulsions, fractures, and lacerations. Sharp force injuries include stab wounds, incised wounds, and cuts. Even though lacerations and cuts both involve a break in the skin, they are not synonymous, and the terms should not be used interchangeably. Most emergency physicians and clinicians use the term "laceration" incorrectly to mean any break in the skin, especially one that requires closure, but it really only applies to those skin breaks that are the result of blunt force trauma or contact with an object. Though they may appear similar, especially if they are linear, they are mechanistically different. Here's why being precise with these terms, and using the strict definitions, can help documentation, especially for forensics purposes.

Lacerations are sustained via two mechanisms: crushing and tearing. Crush lacerations occur when tissue is compressed between two objects with enough force to cause skin breakage. Lacerations are usually full thickness and are seen when the skin and soft tissue are crushed between the impacting object and underlying bone. Lacerations are often linear but can be irregularly shaped. Blood vessels and nerves can be exposed. The wound edges of a laceration are frequently irregular, bruised, or macerated. Such wounds may be contaminated. Delicate tissue bridges can be seen within the depth of the wound. In fact, tissue bridging is the hallmark finding in lacerations. In cuts, these tissue bridges are disrupted by the sharp edge of the weapon.

Split lacerations occur when tissue is stretched at two points resulting in splitting or tearing at the weakest point in between. This mechanism is classically seen in genital trauma following sexual assault. The wound edges of split lacerations may not exhibit damaged wound edges and may not include tissue bridging.

Sharp force wound edges are characteristically sharp, clean, unbruised, unabraded, and inverted. There is no tissue bridging, and the injuries tend to be more linear. The deeper tissues are cut cleanly in the same plane. Stab wounds are deeper than their length or width and occur when a sharp object is thrust into the skin. Incised wounds and cuts are greater in width and/or length than depth and occur when a sharp implement is dragged over the skin surface.

Documentation Tips

When describing open wounds in the emergency department, there are some important historical key points that need to be considered:

- Mechanism of injury
- Type of injury: blunt trauma, sharp force trauma, or both
- Area of the body injured
- Object causing injury: type, size, other characteristics (ie, clean or contaminated)
- Social factors: consistency of wounds or injuries seen with the history provided

After obtaining a thorough history, each wound should be closely inspected and documented. The more precise the wound description, the clearer it will be to future chart readers. Wound descriptions should include the precise location, relationship to key body landmarks, size, shape, directionality, patterned appearance, and any other unique characteristics. The presence of any debris, bleeding, or foreign matter should be noted. When documenting the size of a wound, a ruler or other measurement device should be used. Most cotton tip applicator packages have a ruler imprinted on the packaging. Whenever possible, at least two measurements should be documented, length and width, length and depth, or all three. Some implements used for stab wounds have unique characteristic patterns that can be imparted into the skin surface. Any of these hallmark patterned wounds should be described. Forensic experts may be able to match the wound pattern to the weapon that caused it.

Wounds should be documented using both narrative descriptions and graphic representations using electronic medical record (EMR)–embedded body diagrams and/or uploaded digital photographs of the patient's injuries. EMR-embedded body diagrams are often used to highlight wound location and shape but may make accurate wound representation difficult compared to written charts. Digital photographs, on the other hand, provide an accurate representation of the wound and memorialize findings for future review by other clinicians and experts, plus they may be useful in any potential legal proceedings. Most current EMRs allow images to be uploaded securely, creating easy storage and access.

Case Resolution

Based on your examination, you feel the wound is consistent with a laceration, although the mechanism is uncertain. The patient's wife later arrives and states he was walking down the patio steps when he stumbled, struck his toe against the edge of the step, cried out in pain, and began bleeding. The wound is irrigated, and its characteristics are documented. The wound is sutured, and the patient is discharged with return precautions and follow-up instructions for suture removal.

RESOURCES FOR FURTHER READING

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intraosseous access, supraglottic airways, crowdsourcing CPR, and mechanical CPR, have been tried with mixed results.[1–6]

A randomized controlled trial (RCT) published in 2013 investigated a protocol of vasopressin, steroids, and epinephrine (VSE) for IHCAs.[7] It reported a better odds ratio for return of spontaneous circulation (ROSC) and survival to discharge, with good neurological outcome with the protocol. This was an interesting finding, but a validation study replicating the results has apparently not been published.

An older RCT looked at the role of the corticosteroid dexamethasone in OHCA.[8] That trial failed to demonstrate improvement in survival to hospital discharge. Liu et al performed a systematic review and meta-analysis on the use of corticosteroids after cardiac arrest.[9] They found there was an increase in ROSC and survival to discharge but were limited by the availability of adequately powered high-quality RCTs.

Reference: Shah K, Mitra AR. Use of corticosteroids in cardiac arrest-a systematic review and meta-analysis. *Crit Care Med*. 2021;49(6):e642-e650.

- **Population:** RCTs and observational studies of patients with IHCA or OHCA
  - » **Exclusions:** Single-arm studies, case reports/series, narrative reviews, and studies irrelevant to the focus of this article
- **Intervention:** Corticosteroids as adjunct therapy in cardiac arrest
- **Comparison:** Patients who did not receive corticosteroids in cardiac arrest
- **Outcomes:**
  - » **Primary Outcomes:** Good neurological outcome (measured using the Glasgow-Pittsburgh Cerebral Performance Category score), survival to hospital discharge, and survival at equal to or greater than one year
  - » **Secondary Outcomes:** ROSC, ICU and hospital length of stay (LOS), duration of vasopressor and inotropic treatment, and blood pressure including mean arterial pressure during CPR and after ROSC

Authors' Conclusions

"Our study found that there are limited high-quality data to analyze the association between corticosteroids and reducing mortality in cardiac arrest, but the available data do support future randomized controlled trials. We did find that corticosteroids given as part of a vasopressin, steroids, and epinephrine regimen in in-hospital cardiac arrest patients and for postresuscitation shock did improve neurologic outcomes, survival to hospital discharge, and surrogate outcomes that include return of spontaneous circulation and hemodynamics. We found no benefit in in-hospital cardiac arrest or out-of-hospital cardiac arrest patients receiving corticosteroids only; however, a difference cannot be ruled out due to imprecision and lack of available data."

Results

There were five RCTs and two observational studies included in this systematic review and meta-analysis (see Table 1). The total cohort consisted of 6,199 patients with a cardiac arrest (90 percent of the cohort came from one retrospective observational study conducted in Taiwan).

- **Secondary Outcomes:**
  - » **ROSC:** Four RCTs, relative risk 1.32 (95% CI, 1.16–1.50)
  - » **ICU and hospital LOS:** One RCT, no statistical difference
  - » **Duration of vasopressor and inotropic treatment:** No studies
  - » **Hemodynamic:** Two studies in supplemental material
  - » **Safety:** Three studies with no statistical differences

Evidence-Based Medicine Commentary

1. Few Studies: There were only five RCTs with a total of 530 patients included in this systematic review and meta-analysis, despite cardiac arrest being a common event with high morbidity and mortality.

2. High Risk of Bias: Only four of the five RCTs could be assessed for bias using the Cochrane Risk of Bias Assessment 2 Tool. Three of those four were at high risk of bias. The lack of high-quality studies weakens any conclusions that can be drawn from these data.

3. Single Research Group: The vast majority of the RCT data for IHCA (92 percent) came from two trials by the same group of authors in Greece. One trial was published in 2008 and the other in 2013. This raises the issue of external validity to 2021 and other health care systems.

4. VSE Protocol: The largest RCT (n=268) assessing corticosteroids for cardiac arrest was part of a VSE protocol for IHCA. The protocol included vasopressin, corticosteroids, and epinephrine rather than corticosteroids alone. This means there has not yet been an RCT that addresses corticosteroids alone for IHCA, though equipoise would be difficult to justify in many care settings. The one RCT for OHCA was from 1984 and failed to show a patient-oriented benefit for dexamethasone when given in addition to standard American Heart Association protocols.

5. Replication: The RCT from Greece assessing VSE in IHCA has, to my knowledge, not been replicated. The lack of replication is a common problem in science.[10]

Bottom Line

There is weak direct evidence to support the use of corticosteroids in IHCA as part of a VSE protocol and no evidence to support the use in OHCA.

Case Resolution

ROSC is achieved, and the patient is transferred to the ICU. The patient eventually goes for an angiogram where a stent is place in his left anterior descending coronary artery. The patient makes a full recovery and is discharged home within a week. You decide to do a journal club with your EM residents to discuss the use of corticosteroids for IHCAs and consider whether your hospital should add a VSE protocol.

Thank you to Dr. Ryan Stanton, who is a community emergency physician with Central Emergency Physicians in Lexington, Kentucky, a member of the ACEP Board of Directors, and host of the ACEP Frontline Podcast.

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

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Should steroids be part of your cardiac arrest protocol?

Table 1: Trial Characteristics

PRIMARY OUTCOME	NUMBER OF STUDIES	RELATIVE RISK (95% CI)
Good neurological outcome	4 RCTs	2.85 (95% CI, 1.39–5.84)
Survival to hospital discharge	4 RCTs	2.58 (95% CI, 1.36–4.91)
Survival of at least 1 year	1 RCT	2.34 (95% CI, 0.83–6.54)

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THE NEXT GENERATION OF EM

RESIDENT VOICE

DR. BORELLI is an emergency medicine resident at the University of Texas Health Science Center at San Antonio and *ACEP Now* resident fellow.

Where Medicine and Literature Meet

Bringing a literary perspective to medicine with the voice of residents

by CARA BORELLI, DO

I am delighted to have been selected as the inaugural *ACEP Now* resident fellow. As the incoming fellow, I would like to introduce myself and describe my vision for the fellowship.

Currently, I am a third-year resident at the University of Texas Health Science Center at San Antonio. My interests specific to emergency medicine include toxicology, prehospital care, psychiatric emergencies and disorders, and addiction medicine. Complementing my experience as an emergency medicine resident, I have a background in research and medical editing that I am eager to bring to this new position. Outside of work, my interests and hobbies include playing the accordion, reading medieval literature, and finding the best hiking trails throughout rural Texas.

Medicine and the humanities, literature in particular, have long been passions of mine. Their intersection in my life has driven me throughout my education and career. As an undergraduate, I majored in literature and spent a year studying English literature at the University of Oxford. At the same time, I was involved in neuroscience research, linguistics, and its relationship to neural processing of language. That work invoked and inspired fundamental questions around interiority (the focus on inner life and identity), subjectivity, and individual experience. This liberal arts perspective formed the core of my perspective.

Enriching Medicine with the Humanities

Humanities and sciences are often taught as distinct and unconnected fields of study. But as the long history of physician-authors demonstrates, the art and practice of medicine can be enriched by literary exploration. For doctors who also write, illness is more than a literary device; illness and the treatment of illness are parts of a meditation on what it means to be human. History has seen physician-authors including Sir Arthur Conan Doyle, author of the Sherlock Holmes series; American poet William Carlos Williams; and Khaled Hosseini, author of *The Kite Runner*. Even James Joyce, the famous Irish modernist, briefly studied medicine. The complex and varied relationships between these authors and the field of medicine are too vast to capture, but I will quote Williams, who viewed writing about his experiences in medicine as a self-reflective experience: "There's nothing like a difficult patient to show us ourselves...I would learn so much on my rounds, or making home visits...I heard words, lines, saw people and places—and used it all in my writing." He saw the process as a "descent into [him]self."[1]

Writing about medicine can both examine the exterior experience and probe into the interior. As emergency physicians, the use of and reflection on personal narrative can help us to incorporate our complex and challenging experiences into our sense of self, to articulate a sense of purpose, and to manage the inevitable difficulties of our work.

I plan to incorporate elements of this perspective into the empirical, scientific discipline of medicine through an examination of both the exterior and the interior human experience, specifically through reflections provided by emergency medicine residents. In unprecedented times where emergency medicine residents are completing their training during a pandemic, the voices of emergency medicine residents are of particular importance. As residents, we are part of the future of the profession, and our reflections during the time of COVID are of both personal and professional significance.

I would like to use this introduction as a space to invite residents to submit to the "Resident Voice" column of *ACEP Now*. I hope you will join me as I seek to incorporate the intersection of the humanities and medicine into this column. I also would like to encourage creativity in topics and themes. Please feel free to submit writing proposals or pieces to the email borelli@uthscsa.edu and follow the Twitter account @ACEPNow.

I am enthusiastic to be selected for this role, and I look forward to resident submissions for the "Resident Voice" column and the introspection that I hope these works will inspire.

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BRINGING YOU CANADA'S BRIGHTEST MINDS IN EM

EM CASES

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6-Step Approach to Acute Motor Weakness

Narrowing your differential diagnosis

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

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Physicians increasingly depend on laboratory tests and imaging, sometimes at the expense of a careful history and physical, to rule in and rule out one diagnosis or another.[1] Perhaps no presentation in medicine depends more on careful history taking and physical examination than acute motor weakness. Here, I'll outline a practical and efficient six-step approach to help narrow your differential diagnosis for the patient who presents to the emergency department with acute motor weakness. It is my hope that by using this approach, you will arrive at a diagnosis well before any tests are considered.

Step 1: Determine if Weakness Represents a True Loss of Motor Power

The chief complaint of "weakness" is often vague and can mean malaise or fatigue or anhedonia to some people and true loss of motor power to others.[2] It is incumbent upon the emergency physician to elucidate whether a complaint of weakness represents a true loss of motor power. Some history-taking tips include asking about what activity of daily living or specific function has been lost or made more difficult. For example, patients may complain of being unable to pick up and control a coffee cup, or they may say they had difficulty standing up from a seated position. The word "power" is preferred when communicating with patients and other physicians, as it is more specific to neuromuscular strength than the word "weakness."

Step 2: Find Pattern of Motor Power Loss

Outside of diffuse weakness, there are five patterns of loss of motor power that each correlate with a specific neuroanatomical source. Diffuse loss of motor power may be caused by systemic problems such as hypo- or hyperkalemia or thyrotoxicosis as well as polymyositis and dermatomyositis.[2] Paraplegia suggests a lesion in the thoracic or lumbar spinal cord or peripheral nerves, quadriplegia suggests a lesion in the cervical spine, and hemiplegia suggests a lesion in the contralateral cerebral cortex. A bilateral ascending pattern that starts at the feet and progresses toward the head suggests a peripheral polyneuropathy such as Guillain-Barré syndrome or a spinal cord lesion such as transverse myelitis, while a bilateral descending pattern suggests neuromuscular junction disease such as myasthenia gravis or a presynaptic disease such as Lambert-Eaton syndrome.[3]

Step 3: Investigate Timing, Course, and Fatigability

The onset of loss of power may help narrow the differential diagnosis. An abrupt onset should be considered an acute vascular event such as an ischemic stroke until proven otherwise. That said, small vessel lacunar infarcts may present in a gradual stuttering fashion over hours or even days; thus, a gradual onset should not rule out the possibility of a stroke.[4] An onset over minutes to hours could also be caused by metabolic abnormalities or toxic exposures. Peripheral neuropathies, neuromuscular junction disease, and myopathies tend to develop over many hours to days. A fluctuating or relapsing course of illness suggests myasthenia gravis, multiple sclerosis, or periodic paralysis, while transient motor loss may be caused by peripheral nerve entrapment or a complex hemiplegic migraine. Finally, fatiguability or worsening motor weakness with repeated muscle contraction, such as chewing or talking, that seems to be more difficult through the day suggests a neuromuscular junction disease such as myasthenia gravis.[5]

Step 4: Explore Associated Findings

There are five key associated findings that may accompany motor power loss that can help localize the lesion and narrow the differential diagnosis. Absence of reflexes suggests a peripheral neuropathy such as Guillain-Barré syndrome. Bladder dysfunction associated with loss of motor power is invariably caused by a spinal cord lesion. Bulbar symptoms such as diplopia, dysarthria, and dysphagia point to a brain stem lesion. To distinguish a left versus right cerebral cortex lesion, dysphasia suggests the former, while hemispatial and/or sensory neglect suggest the latter. Hemispatial neglect is sometimes obvious based on observation alone, but visual field testing confirms the finding.[6] The "double extinction test" assesses for the presence of hemisensory neglect. This test is performed by tapping on the patient's forearm on one side while asking which side they are able to feel the tap, right or left.[7] The test is then repeated on the other side. Finally, both forearms are tapped simultaneously. The patient with hemisensory neglect will report sensation only on the non-neglected side.

Step 5: Distinguish Upper Versus Lower Motor Neuron Weakness

Clinical examination can distinguish between upper and lower motor neuron pathology. A Babinski reflex and increases in reflexes are seen with upper motor neuron lesions, while decreased or absent reflexes are associated with lower motor neuron lesions. An assessment of the degree and speed of movement may help distinguish upper from lower motor neuron disease.[8,9] Lower motor neuron disease weakness is generally more pronounced than upper motor neuron disease weakness. For example, the loss of power one gets as a result of a peripheral neuropathy, such as a foot drop, is generally more severe than the loss of power one gets from a central nervous system stroke. Another distinguishing feature is the speed of movement: While lower motor neuron lesions do not affect the speed of motor movement, upper motor neuron lesions usually result in slow movements, sometimes referred to as corticospinal tract slowness. To test motor speed, ask the patient to tap their foot or roll their forearms around one another at an increasing tempo.

Step 6: Differentiate the Types of Lower Motor Neuron Lesions

Peripheral neuropathies tend to present with sensory deficits before motor ones, with the longest nerves typically affected first, as is common in patients with Guillain-Barré syndrome.[10] In contrast, neuromuscular junction diseases such as myasthenia gravis as well as myopathies usually present with pure motor deficits. Myopathies are painful, with proximal muscles being affected most (difficulty getting out of a chair, climbing stairs, or brushing one's hair) as opposed to neuromuscular junction diseases, which tend to be painless, and peripheral neuropathies, which tend to attack the distal nerves first.[3]

Quick Screen Motor Exam

A detailed motor exam is often not practical in the emergency department given time constraints. However, there are six quick screening physical exam maneuvers that, if normal, can influence the need for a detailed segmental motor exam in all except patients in whom you suspect a spinal cord lesion. Two of them are familiar to emergency physicians: facial symmetry/power testing and arm pronator drift. The other four, although less familiar, are well-validated and can be time-saving.

1. The forearm roll test: Ask the patient to make fists with both hands and roll their forearms around each other quickly

for 10 seconds in both directions.[11,12] With an upper motor neuron lesion, the affected arm will be noticeably slower than the unaffected arm.

2. If the forearm roll test is normal (equal speed between the arms), ask the patient to roll their index fingers around one another. Again, the affected side will be slower than the unaffected side.
3. Many emergency physicians are unaware of the lower extremity drift test (the "Mingazzini maneuver," see Figure 1), where the supine patient flexes the hips so their thighs are at right angles to the trunk and the lower legs are flexed so they are horizontal to the stretcher.[12,13] The patient is asked to hold this position for 30 seconds. The affected thigh will lower toward the stretcher, and the leg will lower in the patient with an upper motor neuron lesion.
4. Foot tapping test: The seated patient is asked to repeatedly tap their foot at a quick, steady tempo, and the clinician observes progressive slowing of the tempo. This suggests an upper motor neuron lesion.

Next time you evaluate a patient in the emergency department with the chief complaint of limb weakness, this six-step approach employing a careful history and this four-item quick screening motor exam will help you home in on the diagnosis before any tests are considered. When it comes to acute motor weakness, the history and physical are a not-so-secret weapon.

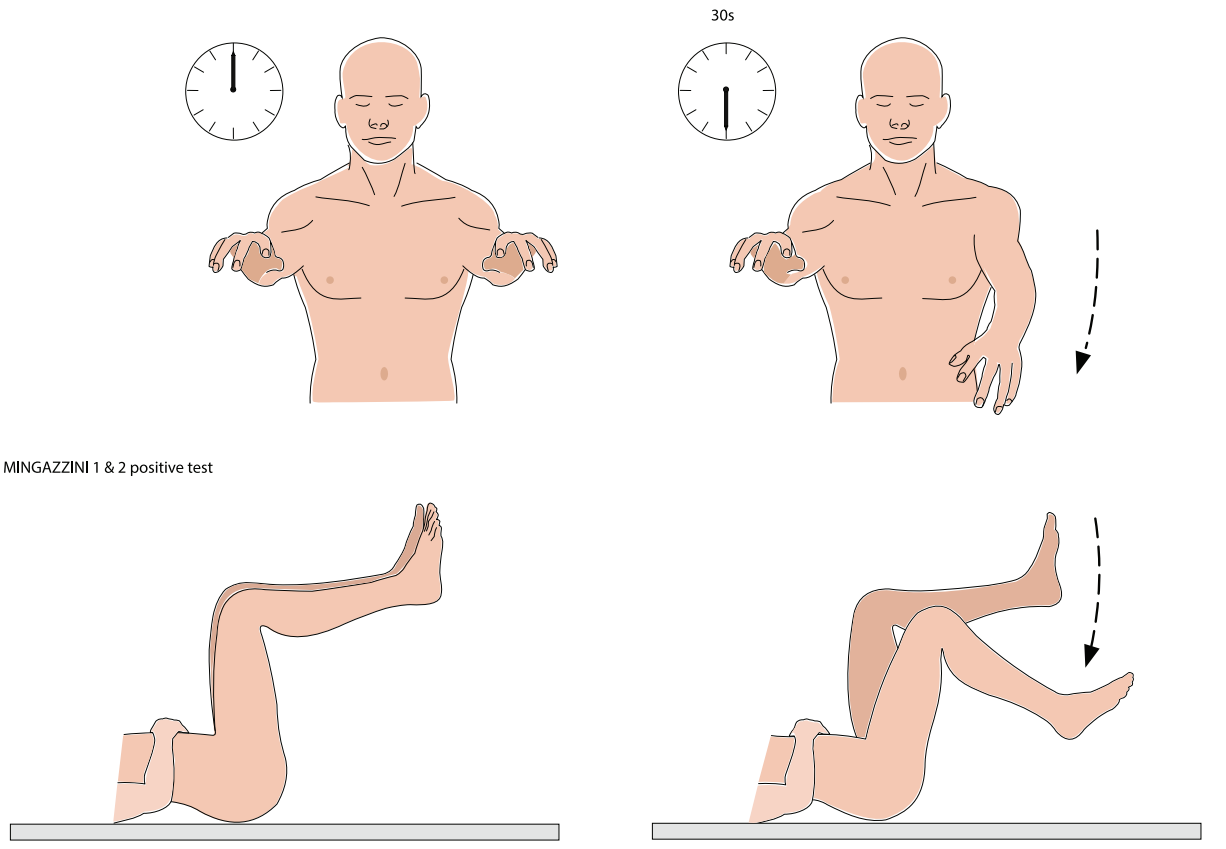

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Figure 1: In the Mingazzini sign (top), a standing or seated patient raises their arms with fingers spread and holds the position for 30 seconds. The affected arm will lower. In the Mingazzini maneuver (bottom), a supine patient flexes the hips so their thighs are at right angles to the trunk and the lower legs are horizontal and holds the position for 30 seconds. The affected thigh will lower.

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TIPS FOR BETTER PERFORMANCE

SPECIAL OPs

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STRONG MEMORIAL HOSPITAL

The discharge lounge at Strong Memorial Hospital in Rochester, New York.

Understanding Discharge Delays and Boarding

Inpatient discharge delays affect the ED, but these strategies can help

by SHARI WELCH, MD, FACEP

Many emergency departments got a brief reprieve from boarding during the COVID-19 pandemic, but most are seeing a return to prior volumes and, with it, the problem of boarding. While boarding is most acutely felt in emergency departments, the solutions to boarding are on the inpatient side, and they are not simple. They often require cultural and operational changes.

Improving the boarding burden is, in large part, predicated on bed capacity and efficient hospital-wide throughput. Decreasing the time patients spend in the emergency department after the decision has been made to admit does not just help us. Minimizing boarding is associated with a downstream positive effect on decreasing the entire inpatient length of stay (LOS).[1,2] This is important when trying to align the inpatient and ED efforts. Moreover, delays in getting patients to inpatient beds have been associated with a variety of adverse events.[3] Lengthy inpatient stays and discharge delays can lead to admission inefficiencies, resulting in a vicious cycle of delays in throughput.

Understanding Discharge Delays

Discharge delays occur when hospitalized patients remain in an inpatient bed beyond what is medically necessity. Discharge delays have negative consequences for both patients and hospitals. Just as the boarding of admitted patients in the emergency department is fraught with patient safety issues and suboptimal care delivery, discharge delays create comparable problems in the inpatient care continuum. With hospitals running at high occupancy, delayed discharges contribute to a host of negative conditions:[4]

- Delayed discharge is associated with increased mortality and infections and reductions in mobility and daily activities.[5]
- Delayed hospital discharges of older patients are common and associated with significant cost.[6]

However, addressing discharge delays is possible and can have benefits for the whole hospital system.

- New studies are helping to identify patients at risk for delayed discharges.[7,8]
- Discharge delays can often be remedied with care teams and managers.[9]
- Discharge timeliness has a positive impact on hospital crowding and ED flow.[10]

Boarding occurs due to demand-capacity mismatch, sometimes referred to as disequilibrium. Hospitals have staffed beds available for a finite number of patients, and they are occupied predominantly by ED patients, transfers into the facility, and procedural admissions. For most inpatient beds, there is already a new patient immediately ready for bed placement when the current occupant is discharged. Hospitals often operate under over-capacity conditions. When demand and capacity are so tightly matched, even the slightest delay in discharging a patient results in ED boarding.

Figure 1 shows a typical admission and discharge disequilibrium curve during a 24-hour cycle. The red demonstrates admissions, which occur even after midnight from the emergency department. There are generally no discharges after midnight. The 5 a.m. admission spike is artifactual and represents data being populated with procedural admissions. The gray curve represents discharges or open beds by hour. Note that in this typical example, capacity for patients admitted after midnight does not match demand until after 2 p.m. This correlates to high arrival times for the emergency department, which is already in its own state of disequilibrium stemming from both from a high volume of arrivals and admitted patients not moving upstairs. In addition, a surge in discharges means a surge in "dirty beds," which creates another mismatch: Environmental services/housekeeping typically scales back staffing at 3 p.m., just when emergency departments need them the most. This results in wasted inpatient bed capacity as rooms are empty but not clean.

Shifting the discharge curve to the left has a number of positive effects. It creates capacity earlier in the day and smooths the workflow of the team.

Every inpatient bed falls prey to inefficiency due to suboptimally designed operations and processes. Patients no longer needing medical beds continue to occupy them due to process and system failures. Some of the contributors to this squandered capacity are:[11–13]

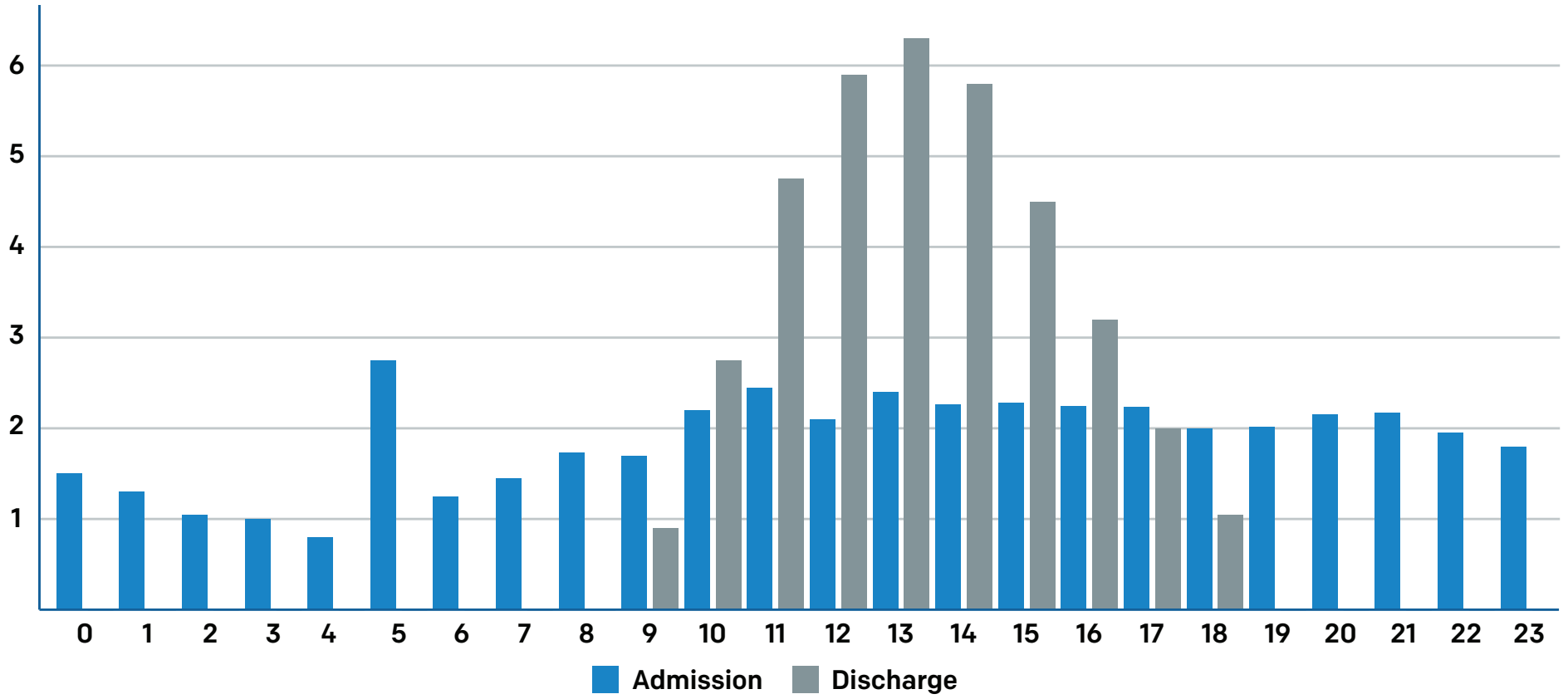
- Waiting for transportation
- Waiting for durable medical equipment
- Waiting for medications
- Waiting for a physician's order to be written
- Waiting for the nurse to provide education
- Waiting for a skilled nursing facility (SNF) or rehabilitation facility bed placement

Hospitalists have become the busiest admitting service in most hospitals across the country, and beds for hospitalist service patients are often the tightest. Facilities that adhere to strict hospital geography by service often find themselves with even more difficulty placing admitted patients because an available bed may not belong to the service or the team to whom the patient has been admitted, creating yet another fixable barrier contributing to boarding.

Improving Discharge Rates

Though many hospitals have employed early discharge orders (eg, by 10 a.m.) as a performance metric for inpatient clinicians (particularly hospitalists), this typically does not translate into an empty clean bed for several hours. Many institutions are tracking true empty beds using a discharge by noon (DBN) metric, which better reflects efficient operations and processes around discharge. Inpatient care is a team sport, and a patient discharge requires a cast of characters touching the patient, including bedside nurses, physical therapists, pharmacists, and case managers. Achieving great DBN numbers requires strong teamwork.

Figure 1: Typical Hospital Disequilibrium by Hour of the Day



Early discharges don't happen without a concerted effort. To avoid readmission, patients need education, medication, durable medical equipment, follow-up appointments, and safe transportation home. Often a physical therapy evaluation is necessary to identify whether the patient is a fall risk at home.

All this planning requires coordination. Care coordination (also called case management) is a newer discipline, and care coordinators or case managers have become indispensable members of inpatient health care teams. These team members can be nurses or social workers. More mature departments have separated utilization review from case management and have identified more experienced workers to manage patients with complicated dispositions. Typically, it is harder to get patients going to SNFs or rehabilitation centers discharged early, though it varies by institution. Though it is not the whole story, many hospitals are focusing on the patients being discharged to home for the best results in DBN. That's because the flow of these patients is the least dependent on factors that are out of the inpatient team's control.

An evolving body of research describes strategies that can help increase DBN by addressing the barriers listed above. Strategies to increase early discharges include:

- Implementing discharge-focused rounds
- Scheduling discharges
- Using discharge whiteboards at patient bedside
- Using electronic discharge notification/communication/tracking
- Establishing a discharge or transition lounge (for more on this, see below)
- Having pharmacy students fill discharge prescriptions
- When safe, using ridesharing to transport patients home
- Reserving nursing home beds and homeless shelter beds for inpatients

Top-performing institutions implement a discharge rounding process that identifies tomorrow's discharges today to allow the multidisciplinary team of nurses, case managers, physical therapists, pharmacists, and others to mobilize and organize their workflow to expedite the discharge and make sure it actually happens.[14,15] Some institutions are now even scheduling discharges to further load-level the day.[16] When an inpatient charge nurse knows who will be discharged, they can make nurse assignments accordingly so that one nurse does not have multiple patients to discharge. When physical and occupational therapists and case managers know who is being discharged, they can prioritize patients leaving that day. Even imaging and lab personnel can be organized to prioritize patients needing testing on the day of discharge. Highly efficient physicians will do much of the discharge paperwork the day before the anticipated discharge. These physicians also flip their personal workflow to discharge those patients first and then commence rounds on other inpatients. When everyone prioritizes getting patients home, capacity is created.

Good communication can facilitate early discharges. One surprisingly simple initiative that has shown improvement in early discharges is providing patient bedside whiteboards that notify and prepare them for discharge.[17,18] At NYU Langone Medical Center in New York City, an email is sent with a discharge list to all stakeholders (physical therapy, occupational therapy, case management, nursing, imaging, lab, pharmacy) so they can organize their daily workflow to expedite these patients' discharge process.[19] EPIC has a discharge module that helps track patient milestones and discharges. TeleTracking is a patient software module that has a special discharge system with milestones for communication among the team. Ultimately, each hospital can harness technology to improve discharge communication.[20]

The discharge lounge (see photo) is a newer concept that has been implemented around the country to help get patients out of their beds, particularly when transportation home is delayed. They have had varying levels of success.[21–23] A variation on this is the transition lounge. It remedies the problem of delays associated with interfacility transports, which often experience delays for patients waiting for basic or advanced life support–capable transport. In addition, receiving facilities have to communicate their willingness and ability to accept a patient. Valley Hospital Medical Center in Las Vegas created a transition lounge that could hold both patients going home who are awaiting their rides and patients going to SNFs or rehab centers. The hospital partnered with EMS to staff this lounge and parked a rig in the parking lot for the sole purpose of transporting these patients. Eventually, they evolved to a scheduled discharge and transport model.

Some regions have begun using ridesharing services like Uber and Lyft to transport patients who do not need a formal medical transport, with training to ensure that patients get into their homes safely.[24]

Another barrier to early discharge can be prescription provision, particularly during the COVID-19 pandemic. A promising approach includes using pharmacy interns or technicians to fill prescriptions for patients going home, with the process often beginning the night before.[25] Finally, innovative health care systems like UCLA in Los Angeles are leasing SNF beds for their discharged patients, while Providence St. Peter Hospital in Olympia, Washington, is leasing shelter beds in the community for homeless patients being discharged.[26]

As emergency physicians who have been struggling with a nationwide boarding burden for almost 20 years, we are right to demand that our respective hospital leaders seriously address the issue using effective inpatient strategies for creating capacity. It won't hurt if we understand the factors leading to discharge delays and bring a few ideas to the table.

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PROTECT YOURSELF FROM LEGAL RISK

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Physician in the Hot Seat

Knowing your case and practicing your testimony will help you put your best foot forward at trial

CHRIS WHISSEN & SHUTTERSTOCK.COM

by GITA PENSA, MD

Part 2 of 2

In Part 1 of this two-part series (published in April 2021), I discussed the sequence of events at trial and what a defendant might experience in the process. In this article, I'll discuss how to put your best foot forward at trial, testify well, and be your own best secret weapon.

After deposition, there will likely be a long period in which you do not have to actively participate in activities relating to your case. Your attorney will be working behind the scenes, and you may occasionally be updated or asked to review something. This period may last months or even years. It is normal and healthy to try to put litigation on the back burner during this time if you can. But once a trial date is set, start strategizing with your attorney about plans to adequately prepare to appear and testify at trial. Here are some general principles.

Know Your Chart, Know Your Deposition

Knowing the intricacies of your medical record was essential to performing well at your deposition; knowing both the medical record *and* your deposition testimony is key to being a strong trial witness.

Your chart will be used as an exhibit, presented to the jury, and projected on a large screen. Once on the stand, you will likely be taken through the chart word by word, line by line, in exhausting detail. The attorney will pore over minutiae that seem to have no real bearing on the medicine of the case. (It bears repeating here that the trial is largely *not* about medicine; it is about convincing a nonmedical jury to pick a side.) The plaintiff's attorney will take any and all opportunities to disparage your care, and they will use any charting errors or discrepancies to paint you as hurried, uncaring, unprofessional, or incompetent. It is important to know where the weaknesses in your chart are and how you will address these.

Likewise, you must carefully review your deposition testimony; your attorney will give you a printed copy of it. It is critical that you stay consistent with what you testified to under oath at deposition, even if it was years earlier. Discrepancy at trial will lead to the plaintiff's attorney reading your deposition words back to you, asking, "Were you lying then, or are you lying now?" If you have come across new evidence or have had your memory refreshed in some way since the time of deposition, you can testify to that—but it is important that you not outright contradict yourself. The jury is more likely to side with you if they view you as sincere, consistent, and trustworthy.

Optics Matter

Even when you are not on the stand, the jury will be sizing you up. Dress appropriately but not in an overly showy or expensive fashion. You may see the jurors outside of the courtroom at times, such as after you have all been dismissed for a recess or as everyone files in for the morning. You might pass them in the hallway or see them in the bathroom. Do not speak to them, but do keep your demeanor warm.

When in the vicinity of the courthouse, always maintain your composure. Jurors may be watching you as you walk into the courthouse before the day starts, and they may see you exit the grounds at the end of an exhausting day. Wait until you are far away before you fully relax your guard or speak in a normal volume to your attorney about the case.

The jurors will also watch you as you listen to other witnesses give uncomfortable testimony. They will naturally want to gauge your reaction to it. If you make faces, appear angry or exasperated, or even appear disinterested or bored, the jury will register it. Try to appear engaged but dispassionate, always exuding a sense of calm and mastery over your emotions. If you are permitted to take notes, do so—this makes you look engaged and also may help you keep your emotions at bay. Maintaining composure is easier said than done at times—you are human, after all—but a little effort at constraint will go a long way.

Practice, Then Perform

It is very important that you and your attorney spend time rehearsing what your role will be at trial. The experience of having a plaintiff's attorney badger you with relentless questioning in front of a judge and jury is daunting, but it is something you can prepare for and get better at with practice.

Trial testimony is markedly different from deposition. You want to convey the same competence and confidence—but while at deposition you wanted to limit your answers, at trial, your aim is also to educate the jury about your care and decision mak-

ing. Though the plaintiff's attorney will try to corral your answers into the narrow scope of their questions, when possible you can try to expand on your answers a bit to help the jury see your side. If you cannot do that without seeming argumentative or disrespectful, you will have an opportunity to explain further when your own attorney questions you. Your words, tone, and demeanor should exude both knowledge and caring. Know the central themes of your defense, and practice answering difficult questions in a way that supports those themes. Keep your language at a level the jury can understand, explaining any medical terms you use, and always emphasize how the care of the patient was at the heart of all your decisions.

Even your body language should support your testimony. Look toward the jury when appropriate. Speak directly to them, even turning your body slightly toward them in your chair, when you periodically try to engage and teach them. Spend some time with your attorney analyzing and working to correct any distracting habits of speech or nervous behaviors you may have. Envision this as a performance—practice makes perfect and also mitigates anxiety.

A plaintiff's attorney may try different tactics to rattle you. (Your attorney may personally know the plaintiff's attorney or their reputation, which may help you strategize in advance.) If their demeanor is threatening or angry, rise above it, knowing it is calculated to manipulate your emotions. They hope to make you angry or flustered; do not let them. Stay professional and composed. On the other hand, if the attorney appears to be kind and complimentary, do not get lulled into complacency. You will need to practice how to stay on your toes, thinking strategically, trying to anticipate where a line of questioning is going, and getting ahead of it when possible. Testifying can also be a test of endurance, as you may spend many hours on the witness stand. The more skilled you become at performing under these conditions, the longer your mind and body can stay focused on the task at hand.

The essence of testifying well at trial is that you want the jury to come away wishing that you were their doctor. Leave your ego, anger, and bitterness about this process at home. Bring to the stand only your best self while maintaining your vigilance under questioning. Be only the kind, compassionate, communicative, and knowledgeable physician who patients would want in their own time of need, no matter what gets thrown at you. Embodying that physician is not hard—it's what you bring to work every day. The real challenge is letting that part of you shine when under atypical stress, answering difficult questions in a hostile environment. Like anything in medicine, this is skill at which you can learn to excel—with practice.

Exciting opportunities at our growing organization

- Emergency Medicine Faculty Positions
- Pediatric Emergency Medicine Faculty Positions
- Vice Chair, Clinical Operations
- Vice Chair, Research

Penn State Health, Hershey PA, is expanding our health system. We offer multiple new positions for exceptional physicians eager to join our dynamic team of EM and PEM faculty treating patients at the only Level I Adult and Level I Pediatric Trauma Center in Central Pennsylvania.

What We're Offering:

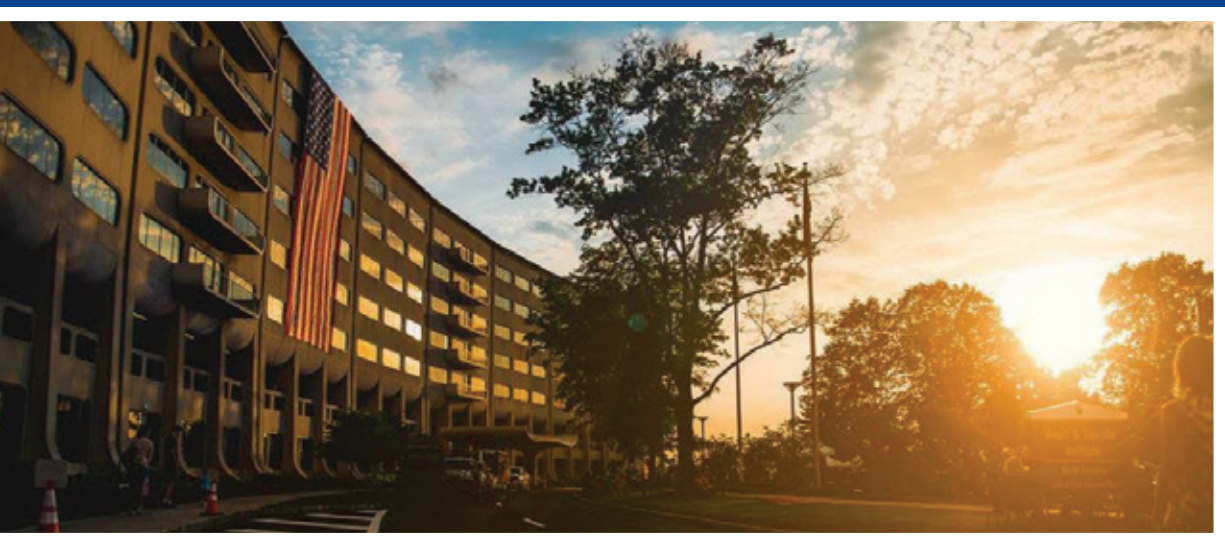
- Salaries commensurate with qualifications
- Sign-on Bonus
- Relocation Assistance
- Retirement options, Penn State University Tuition Discount, and so much more!

What We're Seeking:

- Emergency Medicine trained physicians with additional training in any of the following: Toxicology, Ultrasound, Geriatric Medicine, Pediatric Emergency Medicine, Research
- Completion of an accredited Residency Program.
- BE/BC by ABEM or ABOEM

What the Area Offers:

We welcome you to a community that emulates the values Milton Hershey instilled in a town that holds his name. Located in a safe family-friendly setting, Hershey, PA, our local neighborhoods boast a reasonable cost of living whether you prefer a more suburban setting or thriving city rich in theater, arts, and culture. Known as the home of the Hershey chocolate bar, Hershey's community is rich in history and offers an abundant range of outdoor activities, arts, and diverse experiences. We're conveniently located within a short distance to major cities such as Philadelphia, Pittsburgh, NYC, Baltimore, and Washington DC.



PennState Health

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