

EM Resident

Official Publication of the Emergency Medicine Residents' Association

August | September 2022

VOL 49 | ISSUE 4

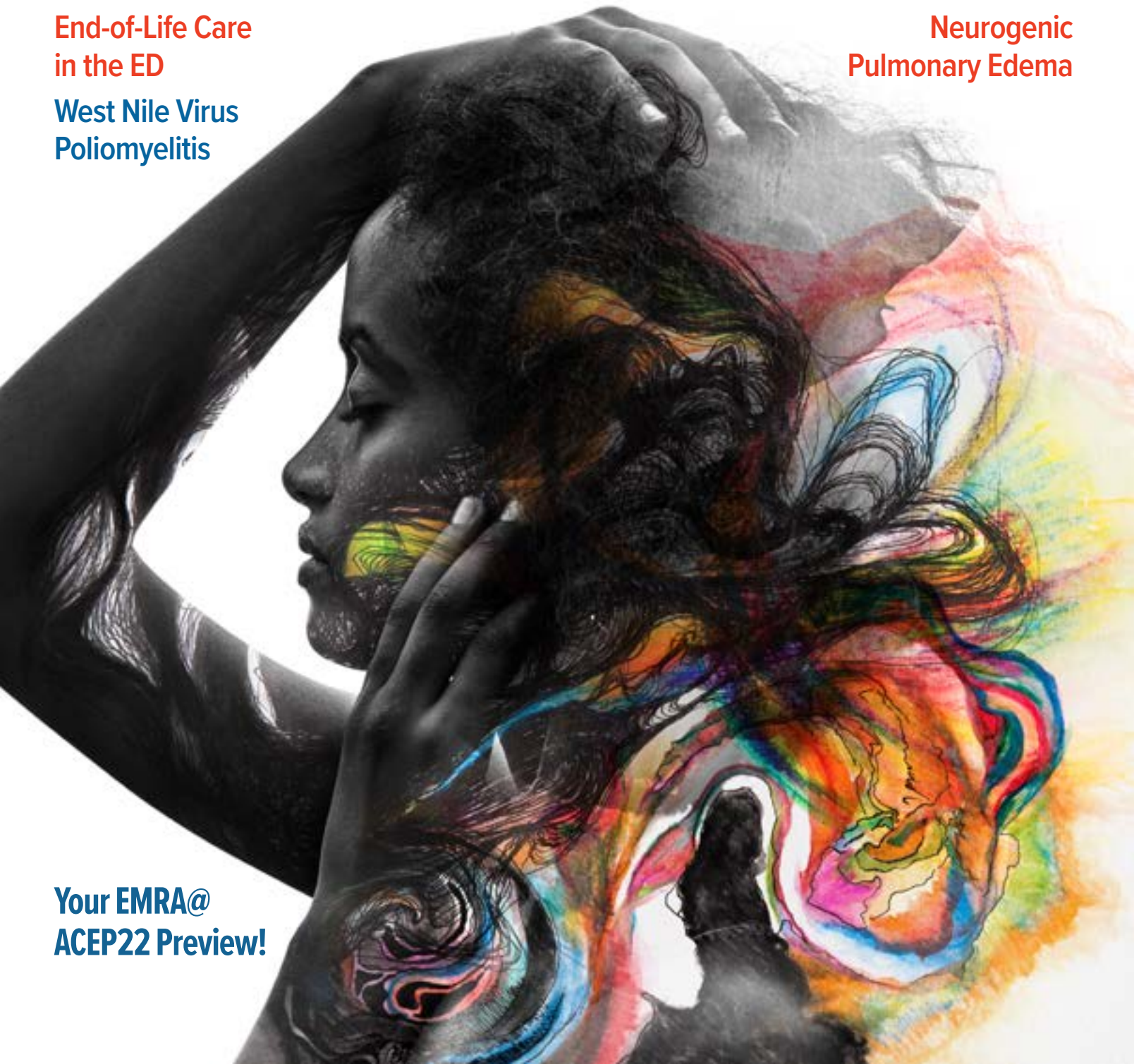
THE DR. LORNA BREEN ACT And How to Join the Fight for our Mental Health

End-of-Life Care
in the ED

West Nile Virus
Poliomyelitis

Neurogenic
Pulmonary Edema

Your EMRA@
ACEP22 Preview!





Join our team and advance your career.

Envision Physician Services is built for your career in **emergency medicine** — wherever it takes you. No matter where you are in your career journey, we can help you find an opportunity that fits your needs, schedule and goals.

EMERGENCY PHYSICIANS CHOOSE ENVISION BECAUSE...

They get to choose where their careers take them.

Diverse practice settings in attractive locations nationwide enable you to pursue your passion for emergency medicine in the area where you want to work and live.

Academic, Pediatric, Staff and Leadership Opportunities
with FT, PT, Per Diem, and ENVOY Ambassador Team flexibility.

Talk with our clinical leaders at these upcoming events to learn more



ACEP22 Scientific Assembly Booth #712

October 1-4
San Francisco, CA



EMRA Job & Fellowship Fair

October 1
San Francisco, CA



Contact our experienced recruiters today.

903.282.3225
EVPS.com/EMRA

 **Envision**
PHYSICIAN SERVICES



Before the Breaking Point

Jessica Adkins Murphy, MD | Editor-in-Chief, *EM Resident* | University of Kentucky

During my intern year, I met with a therapist for the first time in my life.

Unhealthy habits I had developed over years had started to catch up with me. When I was overwhelmed by the tragedies and violence I saw at work, I mostly bottled up my emotions. I told myself I was compartmentalizing, but, rather than discussing my painful experiences with my husband or my family at a later time or expressing myself in a constructive manner, instead I attempted to ignore my mounting stress. Of course, this wouldn't erase my memories of patients' suffering. It only delayed my response until the tension resurfaced as irritability, less efficiency at work, and a vague sense of disorganization in my life.

These were not debilitating habits in medical school when I was mostly just responsible for myself. But intern year was a wake-up call in which I found myself personally entrusted with patients' lives. This challenge clarified for me which of my behavior patterns were counterproductive and kept me from rising to the occasion. I was tired of feeling disorganized and falling short of my goals, and I was ready to prioritize my mental health.

That's when I found my therapist. We met several times over the course of about 6 months, and she helped me identify some ways that I could break my cycles of behavior that weren't serving me, while also giving myself grace as I grew through the difficult journey of early residency. If I hadn't made those changes more than a year ago, my life may not have felt manageable enough to take on opportunities like writing for *EM Resident*. It was a turning point, and I'm glad I made these changes before I would have reached a state of desperation.

Over the next few years, there will

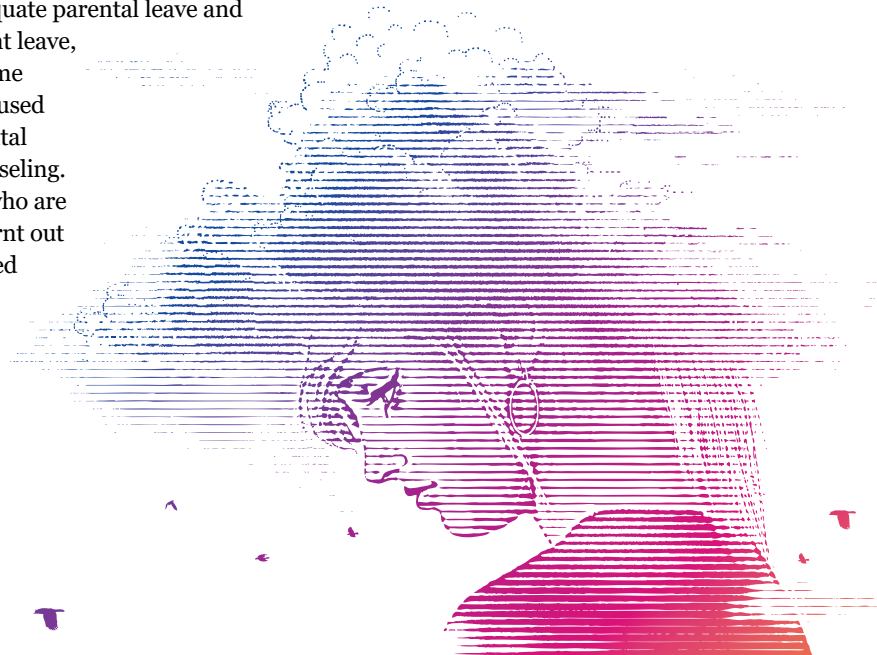
probably come a time when you feel you aren't your best self. Whether you are feeling early symptoms of burnout, are not quite meeting your goals, or suffer from clinical depression, anxiety, substance use, suicidal thoughts, or another severe mental health issue, it's essential that we have access to professional help and can seek help early.

To our chief residents and to programs' leadership, you now know the issues of burnout and wellness cannot be solved by an annual yoga outing or another lecture on burnout as an academic concept. Instead, we need real strategies that alleviate stressors at work and reassure us that we are safe to seek professional support. Consider removing your EM residents from an off-service rotation that has little educational value but excessive stress and scutwork. Staff your exceptionally busy shifts with sufficient nurses, techs, students, and scribes, which will allow us to serve patients as efficiently as possible. Secure adequate parental leave and bereavement leave, and offer time that can be used to seek mental health counseling. Residents who are severely burnt out may not need additional wellness activities to add to their list of

obligations. They may need a respite.

Now in my third year of residency, I haven't seen my counselor in several months. But I imagine there may be times in the future I will need support again. If it's during residency, I'll be glad my co-residents and program leadership are building a culture that listens and intervenes even before a breaking point is reached. And for my next workplace, I'll be glad to have federal policy, the Dr. Lorna Breen Health Care Provider Protection Act, incentivizing my hospital to prioritize wellness and security in seeking professional help. Check out the EMRA Wellness Committee's article in this issue for more on the Dr. Lorna Breen Act, which EMRA members helped bring to fruition.

Now, I want to hear from you. How are you supporting mental health for EM physicians at your program or through EMRA? Connect with me below by email or social media, and we may feature your work in the next issue of *EM Resident*! ★



Jess

Let's keep in touch!

Email emresidenteditor@emra.org | Instagram [@jessadkinsmurphy](https://www.instagram.com/jessadkinsmurphy) | Twitter [@DrAdkinsMurphy](https://twitter.com/DrAdkinsMurphy)

TABLE OF CONTENTS

- 1** Before the Breaking Point
EDITOR'S FORUM
- 4** Welcoming the Future
Leaders of EM's Evolution
PRESIDENT'S MESSAGE
- 5** 10 Tips to Make the Leap
From Resident to Attending
CAREER PLANNING
- 7** Clinical Informatics
Workshops: A Q&A With
Dr. Carrie Baker
INFORMATICS
- 9** Initiating End-of-Life Care
From the ED
GERIATRICS, PAIN MANAGEMENT,
PALLIATIVE CARE
- 12** Dr. Lorna Breen Act: One
Step in the Right Direction
for Physician Wellness
WELLNESS, OP-ED, LEGISLATION
- 14** West Nile Virus Poliomyelitis:
An Unnerving, Re-emerging
Disease on the Rise
INFECTIOUS DISEASE,
NEUROLOGY
- 18** "Feline" Feverish:
A Case Report
INFECTIOUS DISEASE,
PEDIATRIC EM
- 21** All That Blisters is Not
Necrotizing Fasciitis:
A Unique Presentation of
Acute Limb Ischemia
ORTHOPEDICS, TRAUMA
- 24** TCA Overdoses and Mimics
EKG, TOXICOLOGY
- 26** Pnuemocephalus: An Atypical
Presentation of Syncope
NEUROLOGY
- 28** Orthostatic Vital Signs: Have
They Fallen Out of Favor?
PATIENT INTERACTIONS
- 32** Drowning in a Flash:
A Review on Neurogenic
Pulmonary Edema
CRITICAL CARE, NEUROLOGY,
TRAUMA
- 36** One Health and Climate
Change: Why EM Physicians
Should Pay Attention
OP-ED
- 38** News & Notes in
Emergency Medicine
UPDATES TO EMRA'S CLINICAL
LINE, SEPTEMBER'S EM DAY
OF SERVICE, AND MORE
- 40** EMRA Events @ACEP22
ELECTIONS, REPCO, JOB &
FELLOWSHIP FAIR, CASE-CON,
SIMWARS, AWARD WINNERS, 20
IN 6, AIRWAY STORIES, MEDWAR,
RESIDENCY FAIR, MEDICAL
STUDENT FORUM
- 51** ECG Challenge
CARDIOLOGY
- 53** Board Review Questions
PEERPREP

EDITORIAL STAFF

EDITOR-IN-CHIEF

Jessica Adkins Murphy, MD
University of Kentucky

EDITORS

Matthew E. Apicella, DO
Mount Sinai Medical Center

Richie Cunningham, MD
Stanford Medical Center

Annemarie Daecher, MD
Einstein Healthcare Network

Zackary Funk, MD
University of Florida – Jacksonville

Lucas Goss, MD
Carolinas Medical Center

David A. Leon, MD
Johns Hopkins Medicine

Evan McMurray, MD
Mount Sinai Morningside West

Gabrielle Ransford, MD
East Virginia Medical School

MSC Editor

Jennifer Geller
*Rutgers Robert Wood Johnson
Medical School*

ECG Faculty Editor

Jeremy Berberian, MD
ChristianaCare

Toxicology Faculty Editor

David J. Vearrier, MD, MPH, FACMT,
FAACT, FAAEM
University of Mississippi

Orthopedics/Sports Medicine Faculty Editor

MAJ John Kiel, DO, MPH, FACEP, CAQ-SM
*University of Florida COM –
Jacksonville*

EM Resident (ISSN 2377-438X) is the bi-monthly magazine of the Emergency Medicine Residents' Association (EMRA). The opinions herein are those of the authors and not of EMRA or any institutions, organizations, or federal agencies. EMRA encourages readers to inform themselves fully about all issues presented. *EM Resident* reserves the right to edit all material and does not guarantee publication.

© Copyright 2022
Emergency Medicine Residents' Association

WHY I'M AN OWNER



“

At Vituity, I get to build the future of medicine. As a Fellowship director, I help physicians become true experts and leaders in their field.

- **Kimberly Sokol, MD, MS, MACM**

Co-Director of Clinical Teaching and Simulation Fellowship, Vituity

Vituity has always been **100% owned** by its physicians, and you **join as a Partner from day one**.

Learn more at vituity.com/EMcareers

Now hiring in: Arizona, California, Illinois, Indiana, Kansas, Missouri, Minnesota, Nevada, New Jersey, Oregon, South Carolina, Texas, Washington, and Wisconsin.

© 2022 CEP America, LLC. Vituity[®] is a registered trademark of CEP America, LLC. All rights reserved.

Welcoming the Future Leaders of EM's Evolution



Angela Cai, MD, MBA
EMRA President
[@angelagcai](#)

As our EM-bound future colleagues embark upon their sub-internships this month, it is prompting me to reflect on what I love about EM. Fifty years ago, emergency medicine consisted of

interns in a single room in a leaky basement (the “pit”), with no residents, no specialty status, and plenty of staffing struggles. How did we evolve into a specialty of innovation that cares for patients 24/7/365, from everyday problems to a global pandemic? How did we become the front-line specialists patients hope for when they ask, “Is there a doctor on board?”

Emergency medicine *is* about adrenaline-pumping, trauma-based critical care. In an exciting, almost poetic, sendoff of my last Critical Care Trauma residency shift, I took care of patients with GI bleeding, traumatic arrest after a fall, stroke symptoms, TB symptoms, diabetic

ketoacidosis, painless jaundice in shock, status asthmaticus, new-onset AFib with RVR, and penetrating trauma to the neck, extremity, and torso.

But emergency medicine is *also* about the “small” wins, like reassuring the first-time mom who thought her newborn was breathing funny at 3 a.m., the patient with abdominal pain who said, “Thank you for explaining that so well,” and the patient with end-stage cancer whom you steer toward a path of dignified death by establishing goals of care and home hospice.

Here is what unites all of these scenarios: Emergency medicine is about serving our patients, all of them. We are the only specialty legally and ethically bound to the principles of EMTALA — no choosing or dumping of patients. Our specialty emerged from patient demand as patients kept showing up to the “Accident Room” at all hours despite long wait times, nonexistent emergency care systems, and questionable care. We meet our patients where they are, in sophisticated hospital-based emergency departments, freestanding emergency centers, and increasingly in virtual and home-based settings.

This spirit of adaptability and

innovation is also reflected in our adoption of bedside ultrasound and FOAMed. Our unique window into our patients’ needs drives our leadership and advocacy for them, whether it’s pushing for a consult, admission, or follow-up within the hospital, or addressing policy issues and supporting social emergency medicine beyond hospital walls.

As EM-bound medical students, you will receive a lot of solicited and unsolicited opinions about your specialty choice. I’d like to pass on some advice I received as an MS-3: Don’t let anyone who is not in the specialty tell you what it’s about. This might be difficult if you don’t have mentors in EM, but now that you’ve read this article, you have at least one. EMRA can help you find you even more.

The only constant in our specialty’s future is change. To say otherwise would miss our entire history of evolving with the needs of our patients. Every specialty faces challenges and the unknown, but not every specialty shares our story, ethos, and potential to shape our future. I believe emergency medicine will continue to be an amazing specialty to work in because of the EM community you are about to join, and our willingness to be there for anyone, anything, anytime, 24/7/365. ★





10 Tips to Make the Leap from Resident to Attending

Kristyn J. Smith, DO

Clinical Assistant Professor
Emergency Medicine
Hospital of the University of Pennsylvania
Twitter: @Dr_kjsmith

Megan Gillespie, DO

Clinical Assistant Professor
Emergency Medicine
Hospital of the University of Pennsylvania
Twitter: @DrMegGillespie

Arthur J. Pope, MD, PhD

Clinical Assistant Professor
Emergency Medicine
Hospital of the University of Pennsylvania
Twitter: @Revdrpope

Congratulations! You made it through residency. On to the next stage of your emergency medicine career: attending-hood (and for some, fellowship)! Here are 10 tips we learned during our first year as attendings at an academic emergency medicine department.

1. Efficiency comes with time. Start off making sure you are methodical. Unlike residency, you will no longer have an attending physician re-ordering hemolyzed labs or ensuring a negative hcg prior to obtaining a CT scan. *You're the attending now.* No one will be there to catch your mistakes, so make sure you take time to be detailed. Create a disposition checklist, and do not hit the discharge or admit button without double checking that everything you wanted ordered has a result. Also, make sure to share all results and disposition

plans with your patient and the rest of the team.

2. Be the advocate your patients and colleagues need. We can all remember a time in residency where a patient disrespected a colleague, or vice versa, and the team just stood there. As the attending, you have control of patient-physician-team dynamics. If you have a consultant who is being disrespectful to a resident on your team, speak up and remind them that you are all working together toward a common goal of doing what is in the patient's best interest in a collaborative and safe setting.

3. Learn names. Trust us, this makes an enormous difference. Learning your colleagues' names and roles (i.e., nurses, technicians, unit secretaries, environmental service members) shows your interest in your new colleagues and

can help to form bonds and friendships. Furthermore, it reaffirms the valuable role each team member plays in the ED. Practically speaking, "name recall" helps you quickly know whom to ask when you are leading a resuscitation or need a piece of rarely used procedural equipment.

4. Trust your training. You trained for 3 to 4 years in an emergency medicine residency. You cared for victims of gunshot wounds and patients with hypotensive shock, you delivered babies, and you did everything in between. Although you will absolutely encounter presentations you never saw in residency (likely during your first attending shift), always go back to the basics — addressing a patient's ABCs will get you far.

5. If the dispo ain't dispo in' — do more tests or don't send the patient home! If

a patient's presentation is not matching the results of your workup, trust your gut! You may have overlooked something and need to do more lab tests or a CT scan to make the correct diagnosis. Your first year as an attending is not the year to worry about earning the title of the attending who "orders the least number of CT scans" or "admits the least." Whatever workup you need to go home and feel you provided the best possible care for a patient is the right workup.

The transition from resident to academic attending physician can be challenging, but you've got this! And we've got 10 tips to help smooth the journey.

6. Study for your boards. Although the pass rate for the ABEM boards was 92% for first-time test takers in 2021, we all know smart colleagues who did not pass. Don't wish you had studied; just study.

7. Remember, you always have something to teach your residents. As a new attending in an academic emergency department, you are responsible

for teaching EM and off-service residents and medical students. We all felt intimidated being a teacher as a new attending, especially if we were not the type of attending who could quote the latest research study. However, we all realized that how we gather a history, perform a physical exam, and execute workups, resuscitations, and procedures is teaching material. Varied practice patterns teach residents "the art of EM" as they finesse their own medical decision-making and practice patterns. Often, the best residency training is discussion of past patient cases.

8. Find a mentor and a sponsor. You are probably asking yourself, "Wait, what is the difference between a mentor and a sponsor?" A mentor provides guidance and knowledge to you about your career. A sponsor speaks about you in rooms where you are not present and actively presents opportunities to you to enhance your career. These people will be instrumental in helping you navigate the complexities of your academic career.

9. Remember, you are not alone. Imposter phenomenon is real! We were fortunate to be part of a crop of attendings fresh out of residency. Knowing that we can reach out to each other

(or to former co-residents at different institutions) without judgment for reassurance has been invaluable. Also, remember: Senior faculty from your residency or new hospital are a priceless resource and have been in your shoes. So, when you feel yourself freaking out, reach out to other newbie attendings, former co-residents, or senior faculty to put that imposter phenomenon in its place.

10. Take care of yourself. A little twist on the words of RuPaul, "If you can't take care of yourself, how are you going to take care of anyone else? Can I get an Amen?" Many of us have put off our own mental and physical health during medical school and residency to pursue our dream job. Figure out what it is that you need (e.g., exercise, time with family and friends, mental health counseling) to become the happiest version of yourself in and out of the ED. *

BONUS TIP: Believe that you've got this. You are going to be a great attending physician!



THIS FALL: Clinical Informatics Workshops for Residents, Fellows, and Students

Michael Dorritie, DO

Resident, PGY-2
Emergency Medicine
Cook County Health & Hospitals System

Nico Kahl, MD

Resident, PGY-4
Emergency Medicine
UC San Diego Medical Center



Beginning this fall, EMRA's Technology, Telehealth, and Informatics (TTI) Committee will host quarterly clinical informatics workshops with ACEP's Informatics Section leadership.

As a pre-launch introduction to these workshops, we spoke with Carrie Baker, DO, chair of ACEP's Informatics Section and Director of Medical Informatics Education and Innovation at Kettering Health. She shared how these workshops will help enhance knowledge and expertise in clinical informatics for residents, fellows, and medical students.

EMRA TTI Committee: How can a clinical informatics curriculum, and specifically the upcoming workshops, benefit residents and medical students?

Dr. Baker: Residents and medical students will learn tools that they can apply during their career to make improvements to patient care and their own efficiency. The workshops will provide instruction on fundamental concepts in clinical informatics, techniques to improve care delivery and patient outcomes (such as clinical decision support tools), data analytics, leadership skills, and project management basics. Attendees will develop a project that they may then implement at their training sites, if approved by their institutional leadership.

EMRA TTI: How will the curriculum for the workshops be structured?

Dr. Baker: The curriculum is

broken down into four parts based on the American Board of Preventive Medicine Clinical Informatics Content. Namely, the workshops will emphasize fundamental knowledge of clinical informatics, care delivery improvement and outcomes, enterprise information systems knowledge, data governance and analytics, and leadership development.

EMRA TTI: What types of projects can attendees plan to develop over the course of the workshops?

Dr. Baker: Projects will be developed based on the individual interest of each workshop attendee. Whether you have a specific project in mind or are seeking more guidance, the workshop leadership team will help support and craft your project of interest. Broadly, such project ideas include quality improvement, order sets, clinical decision support, process improvement, improved efficiency, and identification of data elements for quality and research.

EMRA TTI: What career opportunities are available to those with clinical informatics training?

Dr. Baker: Clinical informaticists work in a variety of areas, including academics, quality improvement, physician adviserships, utilization review, hospital leadership, and technology startups. Examples include those found online, such as on American Medical Informatics Association's jobs site. Those with an interest in hospital leadership,

for instance, may pursue roles as a chief medical information officer, chief medical officer, or medical director of informatics. Others may choose to work for vendors like Epic, Cerner, and Nuance. There are also an increasing number of opportunities available at technology startups, both in the private sector and through innovation centers at academic institutions across the country.

EMRA TTI: Will there be mentorship opportunities available to workshop attendees?

Dr. Baker: Yes! The workshop presenters are leaders of ACEP's Section for EM Informatics who want to support EMRA's members so that EM physicians-in-training can use informatics tools to make improvements in emergency medicine. We welcome and encourage those who are interested in informatics to continue working with us. You may also further your informatics knowledge through the 10x10 program at Oregon Health & Science University. If there is interest, we can set up a group forum in Basecamp. Additionally, I am happy to connect directly at Carrie.Baker@ketteringhealth.org. Just be sure to put "EM Informatics" in the subject line.

Be on the lookout for more information on Twitter and Basecamp regarding the workshops, and reach out to EMRA's TTI Committee with any questions at EMRAInformatics@emra.org. ★



Penn State Health Emergency Medicine

About Us:

Penn State Health is a multi-hospital health system serving patients and communities across 29 counties in central Pennsylvania. The system includes Penn State Health Milton S. Hershey Medical Center, Penn State Children’s Hospital, and Penn State Cancer Institute based in Hershey, PA; Penn State Health Holy Spirit Medical Center in Camp Hill, PA; Penn State Health St. Joseph Medical Center in Reading, PA; and more than 2,300 physicians and direct care providers at more than 125 medical office locations. Additionally, the system jointly operates various health care providers, including Penn State Health Rehabilitation Hospital, Hershey Outpatient Surgery Center, Hershey Endoscopy Center, Horizon Home Healthcare and Pennsylvania Psychiatric Institute.

In December 2017, Penn State Health partnered with Highmark Health to facilitate creation of a value-based, community care network in the region. Penn State Health shares an integrated strategic plan and operations with Penn State College of Medicine, the university’s medical school.



We foster a collaborative environment rich with diversity, share a passion for patient care, and have a space for those who share our spark of innovative research interests. Our health system is expanding and we have opportunities in both an academic hospital as well community hospital settings.

Benefit highlights include:

- Competitive salary with sign-on bonus
- Comprehensive benefits and retirement package
- Relocation assistance & CME allowance
- Attractive neighborhoods in scenic Central Pennsylvania



PennState Health

FOR MORE INFORMATION PLEASE CONTACT:
Heather Peffley, PHR CPRP - Penn State Health Physician Recruiter
hpeffley@pennstatehealth.psu.edu

Penn State Health is fundamentally committed to the diversity of our faculty and staff. We believe diversity is unapologetically expressing itself through every person’s perspectives and lived experiences. We are an equal opportunity and affirmative action employer. All qualified applicants will receive consideration for employment without regard to age, color, disability, gender identity or expression, marital status, national or ethnic origin, political affiliation, race, religion, sex (including pregnancy), sexual orientation, veteran status, and family medical or genetic information.



Initiating End-of-Life Care From the ED

Matthew Walton, MD

Resident, PGY-3
Emergency Medicine Residency
Geisinger Medical Center

Eric Melnychuk, DO

Core Faculty
Geisinger Emergency Medicine Residency
Critical Care Attending Physician
Geisinger Medical Center

CASE

A 55-year-old male with a history of pancreatic cancer presents with his wife to the ED with jaundice, dark urine, and malaise for one week. He has been following up with his oncologist and primary care doctor, and he explains there are no other viable options for treatment of his cancer and he will likely die within the next month. He would like to be at peace in his final days and wants to transition his care

to focus on his comfort. He has home health nursing 24/7 given his significant decline recently. Though sad given the circumstances, his wife is in agreement and is ready to support him with his decision.

END OF LIFE IN THE ED

We are often confronted with patients presenting to the ED in the final days of their lives, whether they are acutely decompensating or on a less acute trajectory toward death. There has recently been more recognition of the importance of having early goals of care discussions involving patients and their families and the initiation of palliative measures in the emergency care setting.¹ Some patients and families have made clear decisions regarding end-of-life care before they enter the ED, and others

may not consider these decisions until they arrive. In other instances, patients and their health care decision-makers have decided not to undergo aggressive measures such as intubation and CPR, but still may be agreeable with some degree of artificial hydration, blood products, or antibiotics.

For those who present to the ED looking for assistance with end-of-life care, the ED care team can help to coordinate first steps. Time should be taken to establish rapport with the patient and their family and to assess understanding of the situation. Medications will be an important part of assuring comfort. Documenting and completing a physician order for life sustaining treatment (POLST) form and an out-of-hospital do-not-resuscitate (DNR) form should be completed.

COMFORT MEASURES

When a patient or family members decide to focus on comfort as the primary goal of care, management changes significantly. Comfort measures include far more than simple pain control, which may be a challenge on its own. Patients may struggle with dyspnea, which can lead to anxiety. Other commonly recognized symptoms requiring aggressive management include difficulty clearing secretions, nausea and vomiting, fevers, delirium/agitation, and pressure ulcers. Traditionally, this care has been initiated in the hospital setting, ICU, or palliative medicine unit, although recently there has been more focus on initiating these measures in the ED.² Early initiation of palliative care has led to decreases in length of hospital stay, decreases in costs of care, and increases in patient and family satisfaction with care received.^{3,4} As long as a patient is hemodynamically stable, comfort measures may be initiated in the ED and continued on an outpatient basis.²

Whether intended as the totality

of care or as a bridge to hospice, comfort care discharge orders can avoid a costly hospital stay and allow patients and their family to have meaningful time together in the comfort of their home or in a nursing home. The orders placed for outpatient palliative care and comfort care should aim to control the same symptoms as inpatient orders. Rather than a one-size-fits-all approach, care should be taken to consider services a patient may already have access to, including nursing home care, skilled nursing, or home health services, and orders should be tailored accordingly. For example, many medications are given intravenously in the hospital, but this may not be an option for a patient in an outpatient setting. Similarly, a patient may be unable to tolerate swallowing medications. Familiarity with medication options and routes of administration will go a long way toward optimizing outpatient comfort. Anticipating the patient's needs



END-OF-LIFE CARE

and caregiver level of comfort should also be a priority. Family should be counseled on what signs/symptoms to expect and how they should respond as well as being given resources to reach out to if they become overwhelmed or have questions.

SYMPTOM CONTROL

The most common symptoms to address are pain, dyspnea, nausea, and agitation.⁵ While opioids may provoke controversy in other cases, they are invaluable in the management of pain and dyspnea in end-of-life care. There are many things to consider when attempting to control pain in end-of-life care, including history of opioid use, severity of pain, presence of end-organ dysfunction, and age.⁶ For example, kidney failure can impede clearance, or morphine metabolites and liver failure can impair metabolism of morphine itself. This may necessitate using an alternative such as fentanyl or hydromorphone. Knowing the basics about opioid metabolism can help with dosing. Opioid requirements may increase as the end-of-life approaches. Opioids also have the benefit of fighting air hunger.

Agitation and delirium may also be a significant source of distress to the patient and caregivers. Benzodiazepines are first-line, although antipsychotics can also be helpful. Benzodiazepines, however, may cause a paradoxical

Table 1. Medications used in the Symptomatic Care of Palliative Patients

Symptom	Medication (Conc/route/dose)	Time to Onset*	Half Life*
Anxiety	Lorazepam 0.5mg tab 1 tab PO q4h prn	15-30 min	12 hrs
	Lorazepam 2mg/ml PO susp 1mg PO (SL) q4h prn	15-30 min	12 hrs
	*Valproic acid 250mg/5ml PO susp (preferred for hyperactive delirium) 250mg TID	4 hrs	9-16 hrs
Nausea	Ondansetron 4-8 mg PO (SL) q8h	30 min	4.5 hrs
	Haloperidol 2mg/ml 1 mg PO (SL) q6h prn		10-37 hrs
Pain	Morphine sulfate 20mg/ml PO susp 5mg q4h prn	60-90 min	2 hrs
Shortness of Breath	Morphine sulfate 20mg/ml PO susp 5mg q4h prn	60-90 min	2 hrs
Secretions	Scopolamine TD 1 patch q72 hours	4-8 hrs	9.5 hrs
	Atropine 2 drops PO (SL) q4h prn	30 min	2.5 hrs
	Glycopyrrolate 4 mcg/kg PO (SL) q8-12h prn	30-40 min	3 hrs
Fever	Acetaminophen 650mg tab 1 tab PO q6h prn	1 hr	2-3 hrs
	Acetaminophen 650mg Supp 1 supp PR q6h prn		

*Time to onset and half-life information collected from DynaMed®



reaction leading to agitation in the setting of delirium. An ideal first choice for anxiety is lorazepam given its quick onset of action. Another choice for agitated delirium, which has shown utility in ICU settings, would be valproic acid.⁷

Nausea and vomiting may be controlled with antiemetics. Ondansetron is a common choice, and dosing is familiar for most physicians. Ondansetron also has the benefit of having a dissolvable form for patients who cannot tolerate swallowing. For refractory nausea and/or cancer-related nausea, an excellent choice is haloperidol.⁸

Secretions may also be an issue for patients as they can impair breathing and create an unsettling “death rattle.” Educating caregivers on gentle and frequent suctioning can go far to combat secretions. Anticholinergics are the drugs of choice to control secretions,

Compassion and a focus on comfort are keys when delivering end-of-life care in the ED.

and popular choices are scopolamine patches, glycopyrrolate, and atropine. Scopolamine patches should be applied once a day. Atropine and glycopyrrolate can be applied sublingually but require more frequent dosing. Anticholinergics

have been found to be associated with agitation in delirious patients.

CASE RESOLUTION

A long discussion was held with the patient and his wife about what to expect in the coming days to weeks. The patient and his wife went through the process of clearly identifying his wishes on a POLST form and an out-of-hospital DNR. He noted significant problems with anxiety, nausea, and pain, and options were explored to augment his existing home medications. Prescriptions and clear instructions were provided for the patient and his home health nurses. Messages were sent to his primary care doctor and oncologist informing them of the patient’s decision, and a referral to palliative care was placed. The patient returns home with his wife, and several weeks later you receive a message from his wife thanking you for helping make his last days comfortable at home. ★

Dr. Lorna Breen Act: One Step in the Right Direction for Physician Wellness

Leyanet Gonzalez, MD, MBA

EMRA Wellness Committee Chair-Elect
EM Resident, PGY-1
Northeast Georgia Medical Center
[@ley_gp](https://www.instagram.com/ley_gp)

Nicholas Melucci, MPH

EMRA Health Policy Committee Vice Chair
Medical Student, MS-4
Michigan State University College
of Osteopathic Medicine
[@n_melucci](https://www.instagram.com/n_melucci)

Leena Owen, DO, MPH

EMRA Wellness Committee Chair
EM Resident, PGY-3
The Johns Hopkins Hospital
[@leena_owen](https://www.instagram.com/leena_owen)

Lorna Breen always wanted to be an emergency room physician in New York City.¹ From childhood, she believed that medicine was the avenue she wanted to pursue to make an impact on people in her community. Dr. Breen's passion for patient care and the patient experience was fundamental to the way she practiced emergency medicine. She was an avid snowboarder, salsa dancer,

and the “cool aunt” to eight nieces and nephews.¹ Dr. Breen achieved her childhood dream, but then came the COVID-19 pandemic.

Dr. Breen was working at the peak of the first COVID-19 surge in New York City in April 2020. After working multiple 12+ hour shifts and not being able to sleep in over a week, Dr. Breen called her sister and stated she was

unable to move.^{1,2} Her family stated Dr. Breen grew worried about one aspect of her life in particular: her job. She was concerned that she would lose her medical license, or be ostracized by her colleagues, because she was suffering due to her work on the front lines. She was afraid to get help; she worried it would end the career that she had spent her entire life working for.¹

Dr. Breen's experience is not unlike many other physicians. Licensing boards throughout the country require disclosure by physicians of current or past mental health care (in some cases at any level), hospitals require disclosure for credentialing, and seeking mental health care is considered a sign of weakness among many medical professionals.¹

According to her family, Dr. Breen



“In order to take care of patients, you have to take care of yourself.”

— **Corey Feist, JD, MBA**

Dr. Lorna Breen's brother-in-law,
president and co-founder
of the Dr. Lorna Breen
Heroes' Foundation



had no known history of mental illness, and unfortunately her risk factor for suicide, being a physician, was overlooked until it was too late.²



DR. LORNA BREEN

References available online.

Every year, hundreds of physicians die by suicide. The rates of burnout and high levels of stress are not new experiences, and COVID-19 has exacerbated this issue.^{2,3}

On March 18, 2022, history was made when the Dr. Lorna Breen Health Care Provider Protection Act (H.R. 1667, S.610) was successfully enacted into law. This is the first law aimed at supporting the well-being of the healthcare workforce, an action that is long overdue. The primary goal of this law is to prevent and reduce burnout, suicide, and behavioral and mental conditions specifically among healthcare providers. Under this law, the Department of Health and Human Services (HHS) is set to award grants to hospitals and other healthcare entities to assist in establishing programs for evidence-based strategies for mental health promotion and resiliency. This includes relevant training for students, residents, and healthcare professionals to improve well-being and job satisfaction. The law also requires HHS to create

a campaign to encourage healthcare professionals to seek support and treatment and to adopt best practices for suicide prevention. It further adds that HHS is required to study and provide policy recommendations for improving the well-being of the workforce, removing barriers to access care, and developing strategies for the promotion of resiliency.⁴

Although burnout and mental health challenges can be driven by a multitude of diverse triggers, this crisis needs to be assessed on various organizational levels to develop necessary interventions and continue the fight toward the improvement of physician well-being and fulfillment.

The authors of this article would like to thank EMRA for all their work in making this law a reality. The historical enactment of the Dr. Lorna Breen Health Care Provider Protection Act (H.R. 1667, S.610) represents the victory of one battle in this critical war. ★

West Nile Virus Poliomyelitis: An Unnerving, Re-emerging Disease on the Rise

Patrick H. Mullett, MD

Resident, PGY-2
Department of Emergency Medicine
University of Arizona, Tucson College of Medicine - South Campus

Ryan Brzycki, DO

Ultrasound Fellow/Attending Physician
Department of Emergency Medicine
University of Arizona, Tucson College of Medicine

INTRODUCTION

Acute flaccid paralysis is among the most feared presentations of multiple disease processes such as Guillain-Barré syndrome (GBS) and, before vaccines, polio. A progressively more concerning cause of acute flaccid paralysis is West Nile Virus (WNV) poliomyelitis. The incidence of this disease is increasing at an alarming rate in the United States. This devastating infectious disease affects anterior horn cells of the spinal cord, exhibiting pathophysiology that is similar to polio. Unlike polio, there is no licensed vaccine available to humans.¹

First introduced into the U.S. in 1999 during an outbreak in New York, WNV is spread via mosquitoes that transmit the virus from birds, which act as amplifying hosts. Humans and other mammals serve as “dead-end” hosts, meaning they become infected, but viruses are not transmitted between mammals. The only known disease prevention measure is to stop avian-to-human virus transmission via preventing mosquito bites and mosquito population control.

Clinically, WNV illness classically presents with fever and typical viral syndrome symptoms, such as headache, myalgia, arthralgia, nausea, vomiting, diarrhea, and/or rash. WNV also causes an alarmingly high rate of meningitis, encephalitis, and neuromuscular disease, including a polio-like paralysis, collectively termed “neuroinvasive disease.”

From 1999-2020, there were 52,000

documented cases of human WNV, and 25,000 cases of WNV neuroinvasive disease in the U.S. alone.² Endemic to the state of Arizona, human WNV infections are rising at an unprecedented level. In 2020, there were 11 combined probable/confirmed cases of WNV reported in Arizona, and only 1 case of acute flaccid paralysis.^{3,4} The following year, in 2021, there were a shocking 1,644 combined probable/confirmed cases of WNV reported in Arizona. Of these cases, 1,114 were neuroinvasive, and 77 cases occurred with acute flaccid paralysis.^{5,6}

Although Arizona is being affected at the highest rate, WNV has been detected in both humans and animals in every U.S. state. The year 2021 marked a time when human WNV neuroinvasive disease incidence peaked across the country, increasing 20%. From 1999-2019, cumulative U.S. data indicated that, on average, 49% of WNV cases were neuroinvasive. In 2021, the percentage of WNV that caused neuroinvasive disease in the U.S. increased to 69% — in just one year.^{2,6}

This disease affects the immunocompromised and elderly the most, but all U.S. inhabitants are at

risk. It is unknown which region(s) of the country will be affected next by an onslaught of neuroinvasive WNV disease. The ability to recognize, diagnose, and manage this disease is essential to the delivery of high-quality care to ED patients, many of whom (along with health care providers) are at risk for this devastating illness.

CASE

In October 2021, a 50-year-old Spanish-speaking male with known uncontrolled type II diabetes, diabetic



neuropathy, congestive heart failure (CHF), schizophrenia, prior transient ischemic attacks (TIA), and a history of remote traumatic brain injury (TBI) presented to an academic ED in Arizona for worsening dyspnea. He was well-known to the ED and well-documented as a difficult historian and non-compliant with medications. History-taking (with a qualified interpreter) was limited, as the patient was tangential in his responses. Although he was focused on his dyspnea, he incidentally reported a new onset of inability to ambulate, which began as lower extremity weakness approximately two months ago. When this first began, he reported to the ED with lower extremity weakness during a febrile illness, was admitted for CHF exacerbation, and was prematurely diagnosed with a viral syndrome, with diabetic polyneuropathy cited as the cause of this weakness. Despite worsening motor function, he had not pursued further work-up for this issue since that visit. There was no prior analysis of cerebrospinal fluid (CSF) or spinal cord imaging. He denied recent trauma, urinary retention, saddle anesthesia, and loss of bowel or bladder function. He denied a history of alcohol/intravenous drug abuse, recent travel, or exposure to known infectious diseases.

PHYSICAL EXAM

Vital signs showed a temperature of 36.9 degrees Celsius, heart rate of 96, blood pressure 176/107, respirations 25, and oxygen saturation of 95% on room air. Physical exam demonstrated an ill-appearing man with profound lower extremity weakness and inability to ambulate. He had 2/5 strength in the bilateral lower extremities. Deep tendon reflexes (DTRs) were absent in both lower extremities. Bilateral upper extremities exhibited 2+ DTRs with 5/5 strength. His sensation was intact and symmetric throughout. Cranial Nerves II – XII were intact. No meningeal signs, facial asymmetry, dysarthria, or signs of trauma were appreciated. His neck and back were non-tender to palpation without step-offs or deformities. He was alert and oriented to person, place, time, and event. However, he was seemingly

confused about the timeline of his symptoms and displayed tangential thoughts during conversation. A small maculopapular rash was noted across the anterior chest.

STUDIES

Chest X-ray showed cardiomegaly and bilateral pleural effusions, with a pro-Brain Natriuretic Peptide level of 831pg/mL, consistent with CHF exacerbation. His electrocardiogram did not demonstrate acute abnormalities. His troponin and complete blood cell count were within expected limits. Blood glucose level was 213 mg/dL. Otherwise, comprehensive metabolic panel was normal. A negative inspiratory force test was performed, which was within normal limits, indicating the patient had adequate diaphragm muscle tone.

Spinal magnetic resonance imaging (MRI) showed T2 hyperenhancement along the entire spinal cord, but no acute fractures or cord compression. A computed tomography scan of the patient's brain showed encephalomalacia of the left frontal and anterior temporal pole — stable from prior studies. There was no evidence of acute infarct, hemorrhage, or enhancement to suggest meningitis.

A lumbar puncture was performed with CSF analysis. The CSF sample was clear and colorless without red blood cells. There were 27 white blood cells (WBCs)/mm³ (normal high: 5/mm³). Lymphocyte percentage was 50% and Monocytes/Macrophages percentage was 50% (reference range: 15-45%). Glucose: 61 mg/dL (within reference ranges). Protein: 93.7 mg/dL (normal high: 40 mg/dL). Oligoclonal bands were present.

Other serology tests were negative including human immunodeficiency virus screening, Lyme disease (*Borrelia* species), syphilis screening, tuberculosis screening, *Cryptococcus* antigen, and fungal serology.

DISPOSITION AND DIAGNOSIS

The patient was admitted to neurology for possible GBS or another cause of ascending paralysis and also treated for CHF exacerbation. He was discharged to a skilled nursing facility the following

day with a presumptive diagnosis of paraplegia secondary to diabetic polyneuropathy.

Several days later, CSF microbiology resulted with West Nile virus IgG positive, measured at 1.59 mg/dL (reference: greater than 1.49 means antibody detected). This indicated an indolent WNV infection of the central nervous system. The WNV IgM was less than 0.90 mg/dL (antibody not detected), indicating the patient was status-post acute infection.

Other pertinent negative CSF results included a CSF gram stain, which showed white blood cells (without organisms) and cultures that showed no growth at three days. A CSF virology PCR test was negative for human herpesvirus 6, cytomegalovirus, varicella zoster virus, enterovirus, Epstein-Barr virus, herpes simplex virus (HSV) 1, and HSV 2. There was no detection of IgG or IgM to western equine encephalitis Virus, eastern equine encephalitis virus, California encephalitis virus, or St. Louis encephalitis virus.

OUTCOME

At the time of publication of this manuscript, the chart review indicates that this patient has been in skilled nursing facilities and never regained the ability to walk, despite rehabilitation. He has suffered from morbid sequelae of paralysis (such as sacral ulcers and osteomyelitis).

DISCUSSION

Based on this extensive work-up, with an MRI showing T2 hyperenhancement and CSF with IgG to WNV, this patient's diagnosis is most consistent with a subacute form of WNV poliomyelitis. A retrospective chart review revealed that the patient had presented several months ago with a febrile illness and acute onset weakness during the summer. His presentation was most consistent with acute WNV poliomyelitis at that time. This occurred during the heavy 2021 monsoon season in southern Arizona when mosquito populations flourished and the incidence of WNV cases was unprecedented.⁵

What is unique about this case



is that the patient's weakness was previously incorrectly diagnosed due to multiple factors that confounded his clinical presentation. Unfortunately, his symptoms were not properly worked up with spinal imaging or an LP until this ED visit. This is presumed to be due to confounding factors of medical comorbidities, prior TBI, and psychiatric diagnoses. Furthermore, severe diabetes has the propensity to worsen any neurological dysfunction, and his uncontrolled diabetes with peripheral neuropathy provided a red herring, which distracted away from his neurologic dysfunction during prior visits. Prior consultation notes by neurology indicate there was likely an anchoring bias on this aspect of his presentation. This likely caused inaccurate diagnostic momentum, which presumably caused bias in his later admitting providers. This ultimately resulted in premature closure and an incorrect diagnosis.

Any patient with encephalopathy or unexplained neurological findings in an area with mosquitos should be considered as a potential WNV neuroinvasive infection case. Neuroinvasive disease from WNV can occur with (or without) an acute febrile

illness and classic symptoms of WNV. Care providers need to be aware of manifestations of WNV neuroinvasive disease, which include, but are not limited to, meningitis, encephalitis, and, with increasing incidence, neuromuscular disease, including acute flaccid paralysis.^{1,7}

Acute onset of myalgias and weakness during infection, fever, and leukocytosis hallmark the presentation of WNV poliomyelitis. Bowel and bladder dysfunction can be seen, and encephalopathy is often present. Infrequently, numbness, paresthesia, or sensory loss can occur. The distribution of weakness commonly occurs in an asymmetric pattern, ranging from monoplegia to quadriplegia and/or diaphragm paralysis. In the absence of fever or meningoencephalitis, acute flaccid paralysis can still occur. Like polio, WNV directly affects anterior horn cells of the spinal cord and motor axons. The CSF shows pleocytosis with elevated protein levels (seen in this patient's case). Oligoclonal bands are non-specific and, although infrequently seen, can be present in the CSF after WNV poliomyelitis (also seen in this case).^{1,7}

Importantly, GBS and WNV

poliomyelitis can be confused for one another. There are important subtle differences. In contrast, GBS demyelinate peripheral nerves and presents weeks after an acute infection, with symmetric weakness, loss of sensory function, absence of fever or leukocytosis, and with albuminocytologic dissociation in the CSF (elevated protein without pleocytosis).^{1,7}

A neuromuscular disease differential diagnosis list should include WNV neuroinvasive disease, GBS, other immune-mediated myopathies/neuropathies, and axonal polyneuropathies (i.e., diabetic polyneuropathy). Other important considerations: bacterial meningitis or encephalitis (including Lyme disease); tick paralysis; non-infectious brain/spinal cord pathology (e.g., spinal cord compression, stroke, tumors of the central nervous system); viral meningoencephalitis (La Crosse virus, Coxsackie virus, echovirus, enterovirus); and other arbovirus encephalitis (i.e., equine encephalitis viruses, tick-borne encephalitis viruses, St. Louis encephalitis virus, and Japanese encephalitis virus).¹

To diagnose WNV poliomyelitis, LP

and spinal imaging should be performed. In the setting of limb paralysis, MRI can sometimes show T2 hyperintensities, especially along the anterior horns of the spinal column (seen in this patient's case). Serum blood tests can help elucidate the diagnosis, but analysis of CSF is the gold standard. Detection of WNV ribonucleic acid via PCR in serum and spinal fluid is possible within 3-5 days following infection but has low sensitivity compared to IgG and IgM antibodies to WNV. The IgM antibody to WNV indicates a recent infection, whereas IgG indicates a more indolent infectious process.⁸ Samples of CSF will generally show a moderate pleocytosis (generally greater than 200 but less than 500 WBCs/mm³), increased protein, and normal glucose.^{1,7}

Disease prognosis varies. There is significant distress associated with early disability, but a substantial percentage of patients can recover to functional independence with intensive rehabilitation over several months. Currently, no proven effective treatment for WNV neuroinvasive disease exists. Management involves supportive care

and rehabilitation. Several case series and small studies have been performed with intravenous immunoglobulin, steroids, interferon, ribavirin, and a monoclonal antibody that is no longer on the market. All therapies reported varying degrees of success rates without any reliable consistency. Ongoing research for the development of rapid antigen testing, therapies, and vaccines continues. At the time of publication, there are multiple trials still underway. There is not yet a federally approved, licensed vaccine available for WNV in humans. However, there are licensed vaccines for WNV available for horses. As of 2015, there have been multiple vaccine prototypes developed, some of which were reported to be in Phase 1 and Phase 2 trials, according to the National Institute of Allergies and Infectious Diseases.^{9,10,11}

CONCLUSION

This case highlights key points of an uncommon disease that is re-emerging. The increasing incidence of WNV, higher rates of neuroinvasive disease, and increasing percentage of WNV

poliomyelitis cases are alarming. If these trends continue, EM physicians will inevitably encounter this disease entity. It is important to consider WNV as a major cause of neuroinvasive disease and acute flaccid paralysis. If a febrile, altered patient comes into the ED with focal neurological deficits, it is vital to include this unnerving disease on the list of differential diagnoses and perform an appropriate workup. Keep in mind that WNV neuroinvasive disease can present without focal neurological deficits, and WNV poliomyelitis can occur in the absence of fever or encephalopathy. It is essential to perform the LP and order the correct diagnostic testing, including CSF antibodies to WNV (speak to your ED's laboratory regarding various forms of WNV testing available). All providers must report positive cases of WNV to local health departments; physicians are mandated reporters. The incidence of WNV is highest in the summer months. Although we are now exiting the summer season, the incidence of WNV poliomyelitis could rise again in 2023. If this occurs, all EM physicians will also need to rise to this occasion. ★

TAKE-HOME POINTS

- West Nile Virus (WNV) incidence has increased dramatically across the U.S. in recent years. Rates of neuroinvasive diseases caused by WNV, specifically WNV poliomyelitis, are higher than ever before.
- Know the difference between Guillain-Barré syndrome (GBS) and WNV poliomyelitis.
 - WNV poliomyelitis:
 - occurs during the acute phase of illness, and often presents with fever and encephalitis (but can occur without fever or encephalitis)
 - usually involves asymmetric loss of motor function; the sensory function is generally preserved
 - CSF shows pleocytosis with elevated protein levels
 - GBS:
 - occurs in the weeks after an illness/instigating event
 - usually involves symmetric loss of motor and sensory function
 - CSF shows albuminocytologic dissociation (elevated protein level without pleocytosis)
- Pause to consider a broad differential of etiologies when a patient has a neuromuscular disease.
- Recognize diagnostic momentum and anchoring bias as reasons for premature closure and inaccurate diagnoses, especially in complex cases with multiple confounders and social biases.
- Be a champion of public health. Scout out this disease and report it to your local health department. Physicians are mandated reporters for infectious diseases. The more public health information health departments have, the more this will help with testing, therapy, and vaccine development. ★



“Feline” Feverish: A Case Report

Shanice Latham, MD, MPH

Resident
Pediatrics
Baylor College of Medicine/San Antonio

Hussain Shakir, MD

Attending Physician
Pediatric EM
Baylor College of Medicine/San Antonio
The Children’s Hospital of San Antonio

CASE

A 22-month-old male presented to the pediatric ED for approximately 6 weeks of daily fever with temperatures greater than 101°F both oral and axillary, decreased appetite, increasing fatigue, and new-onset right upper quadrant pain. When the fever first began, he was seen at an urgent care center and diagnosed with acute otitis media. One week later, he was again seen at urgent care for continued fevers and a swollen toe. He was diagnosed with cellulitis and prescribed cefdinir, but the fever

persisted despite antibiotics. He was then taken twice to the emergency department, where he was diagnosed with enterovirus/rhinovirus and treated with ceftriaxone for leukocytosis noted on CBC.

On this presentation, the family was concerned for his persistent fever and nasal congestion but no other active symptoms. Social history was negative for travel. He had contact with dogs and cats at home. Review of systems was positive for fevers, right upper quadrant abdominal pain, decreased

appetite, headache, and fatigue. There was no report of cough, hemoptysis, weight loss, night sweats, joint pain or swelling, rashes, swollen glands, or mucosal changes. On exam, vitals were normal and he was afebrile at 99°F. He had cervical lymphadenopathy with a right-sided 2 cm soft mobile lymph node. Labs were significant for leukocytosis, elevated ESR and CRP, normal uric acid, and mildly elevated LDH. Titers for zoonoses and viral infections like EBV, typhus, Bartonella, and CMV were sent and remained pending while the patient

was in the ED. Given lab results and clinical history, infectious disease (ID) was consulted due to this presumed fever of unknown origin. ID recommended inpatient admission and an abdominal ultrasound, given cat exposure and risk for disseminated cat-scratch disease. After admission, the ultrasound showed multiple cystic lesions in the liver and spleen, consistent with disseminated cat-scratch disease. (Figures 1 and 2)

without antibiotic treatment. However, approximately 5-25% of CSD cases are atypical or disseminated, where the infection has spread from the site of inoculation to other visceral organs such as the heart, eyes, liver, spleen, or brain.

A CSD diagnosis is typically done through a combination of clinical findings, historical exposure to cats, and serologic testing. Atypical CSD can present with nonspecific symptoms

Because of the high prevalence of CSD in association with FUO, abdominal imaging is essential in these patients. Almost 70% of patients with hepatosplenic CSD will have microabscesses in their spleen and liver.⁴ These abscesses typically present as hypoechoic lesions on ultrasound and histologically represent necrotizing granulomas. Most recently it has been suggested that hepatosplenic involvement may be associated with even typical CSD, since one study has found a hepatosplenic compromise in both cases of typical and atypical CSD.² Nevertheless, management of patients with hepatosplenic CSD and other forms of atypical CSD has been somewhat controversial, since antibiotic therapy for even typical CSD is not generally indicated but is used for shortening the duration of symptoms. Most children who are hospitalized due to disseminated CSD receive antibiotic therapy for symptomatic management as well as to prevent further complications, especially in cases of aseptic meningitis, neuroretinitis, and culture-negative endocarditis.

Behind typhoid fever and urinary tract infections, cat-scratch disease is the third most common cause of fever of unknown origin in pediatric patients. CSD should be high on the diagnostic radar whenever a child with prolonged fever presents to the emergency department.

DISCUSSION

Bartonellosis is a zoonotic infectious disease caused by *Bartonella henselae*, a gram-negative bacterium. It typically presents as benign cat-scratch disease (CSD) in about 90% of cases, predominantly in the summer-autumn months.¹ The pediatric incidence of CSD is approximately 4 per 100,000, with the highest incidence at ages 5-9 years.²

Cat-scratch disease typically presents after a bite or scratch from a cat, more commonly a kitten, resulting in localized painful lymphadenopathy, fever, malaise, rash, and headache. Although kittens are the natural carriers of *Bartonella*, it should be said that it is also associated with dogs since the transition occurs primarily through flea bites.¹

Often, cat-scratch disease goes undiagnosed and resolves spontaneously

such as abdominal pain, weight loss, headache, and fever of unknown origin. It is the investigation of fever of unknown origin that characteristically leads to the diagnosis of atypical CSD, as in our patient.

Behind typhoid fever and urinary tract infections, CSD is the third most common cause of FUIO and should be high on the differential whenever a child with prolonged fever presents to the emergency department.³ Hepatosplenic CSD, a subset of atypical CSD, more commonly occurs with FUIO and other systemic symptoms such as abdominal pain and weight loss. The evaluation of patients suspected of having disseminated CSD should include CBC, inflammatory markers such as ESR and CRP, and serologic titers (both IgG and IgM).

Typical CSD	Atypical CSD
Regional tender lymphadenopathy	Hepatosplenic abscesses
Vesicular lesion at site of inoculation	Neuroretinitis/ Parinaud oculoglandular syndrome
	Endocarditis
	Encephalitis



FIGURE 1

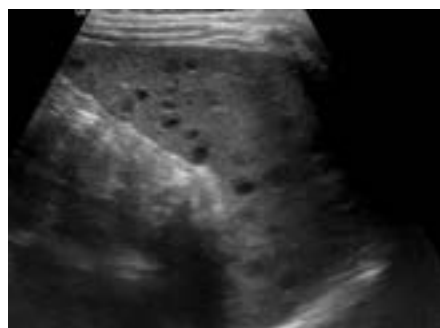


FIGURE 2

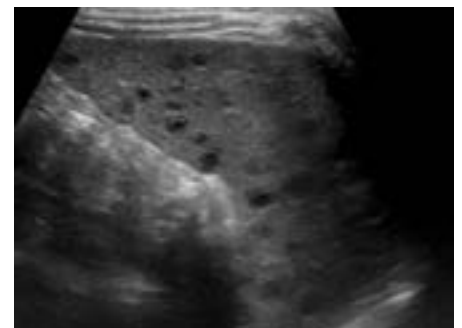


FIGURE 3



persistent CSD. Other antibiotic regimens include azithromycin alone for 14 days, gentamicin in combination with TMP-SMX, and rifampin for up to 4 weeks. The precise treatment of hepatosplenic CSD remains unclear; however, the literature recommends treatment for all patients diagnosed. The clinical response in a patient who is immunocompromised compared to immunocompetent is dramatically improved when treated with antibiotics.

CASE CONCLUSION

The patient was started on azithromycin and rifampin for 14 days based on ID recommendations. He was discharged home with ID outpatient follow-up. Several days after discharge, the patient's serologic titers came back positive with IgG 1:1024 and IgM 1:128. The family, when called, said his fevers had resolved and he was back to his normal activity level.

Atypical cat-scratch disease can manifest in a variety of ways, most notably hepatosplenic disease in both the immunocompetent and immunocompromised child. Recognizing that this disease is one of the top causes of fever of unknown origin is important in the diagnosis and management of pediatric patients. Undiagnosed cat-scratch disease can have high morbidity. Therefore, it is important for emergency physicians to correlate a good history and physical examination, as well as appropriate laboratory and imaging studies, in order to appropriately treat these patients. ★

MANAGEMENT OF ATYPICAL CSD

There is no consensus on the treatment of atypical CSD. Various case series and reports have proposed a mixture of regimens that include azithromycin, rifampin, gentamicin, trimethoprim/sulfamethoxazole, and broad-spectrum cephalosporins. However, no in vivo randomized controlled trials (RCTs) have been conducted to compare efficacy. Only one in vivo RCT has been done to evaluate the effect of azithromycin on CSD. That trial found a significant reduction in lymph node size in the treatment group.⁵ Therefore, treatment

is individualized and based largely on symptom severity. Perhaps the most utilized regimen for hepatosplenic CSD is a 14-day course of rifampin, either alone or in combination therapy.⁴ This regimen has been shown to be highly effective in most immunocompetent patients with almost none of those patients developing





All That Blisters is Not Necrotizing Fasciitis: A Unique Presentation of Acute Limb Ischemia

LT Matthew J. Christensen, MD

Resident, PGY-2
Emergency Medicine Residency Program
Naval Medical Center San Diego

LCDR William Bianchi, DO, MSc

Attending Physician
Department of Emergency Medicine
Naval Medical Center San Diego

Intern year is full of teachable moments, as new physicians build repetitions seeing typical presentations of common chief complaints. But the true beauty of an intern year in emergency medicine comes when that intern finally develops a basic foundation of expediently recognizing and managing emergent life-threatening conditions, only to be swiftly confronted by a zebra. This was one of those cases.

CASE INTRODUCTION

A 66-year-old man is brought in by ambulance with pain, discoloration, decreased sensation, and serous-draining bullae to his right lower extremity. He states that his right leg has been mildly painful for about a week, the overlying skin began to change color three days

ago, and he woke up this morning with multiple tense hemorrhagic bullae and blisters to the area, which prompted his call to EMS. His medical and surgical history is notable for hypertension, heparin-induced thrombocytopenia (HIT), right forefoot amputation, and “multiple vascular surgeries,” regarding

which he could recall no details. He denies any daily medications or drug allergies, recent trauma, and new exposures of any kind. His social history is notable for chronic alcohol and tobacco use, and he is living in a homeless shelter at this time. This patient is ambulatory at baseline without assistance, wearing

References available online.

footwear in poor condition with multiple holes that could allow for direct skin exposure to the environment.

On initial evaluation, he is tachycardic to 124 beats per minute, febrile to 38.8°C, and borderline hypoxic at 92% on room air. He is fully alert and oriented, breathing at 18 breaths per minute, and initial blood pressure is measured at 158/85 mmHg. The skin of his right lower leg below the knee is purple and mottled, with multiple overlying blisters and bullae. There is no fluctuance or crepitus, and the bullae are Nikolsky negative. Blanchable erythema is noted to the right mid-thigh without scrotal swelling, discoloration, or tenderness. There are no oral lesions or mucosal abnormalities. Assessment for dorsalis pedis or posterior tibial pulses in his right foot is not possible

due to the extensive bullae formation. The remainder of his skin exam is unremarkable, as is the pulmonary and gastrointestinal exam. There is decreased sensation to light touch over the discolored area of the right lower extremity, but no other focal neurologic deficits.

DIFFERENTIAL DIAGNOSIS

Febrile and tachycardic with acute-onset lower extremity skin changes after walking in poorly sanitized conditions with tattered footwear? A septic picture due to underlying necrotizing fasciitis is right at the top of the differential diagnosis. Additional differentials considered include infectious etiologies such as osteomyelitis, Fournier’s gangrene, erysipelas, and cellulitis. Potential vascular etiologies include deep

vein thrombosis leading to phlegmasia cerulea dolens, or arterial occlusion causing acute limb ischemia. Other possibilities considered are orthopedic causes such as compartment syndrome, or pathology on the dermatologic spectrum of Steven-Johnson syndrome to toxic epidermal necrolysis (SJS/TEN). Autoimmune conditions may present like this as well, with bullous pemphigoid or atypical systemic lupus erythematosus coming to mind.

DIAGNOSIS AND INITIAL MANAGEMENT

An initial workup for sepsis due to necrotizing fasciitis is pursued, with aggressive fluid resuscitation and antipyretics given, labs and blood cultures drawn, empiric antibiotics administered, and plain film radiographs of the right lower extremity obtained. Lab results show mildly elevated lactate of 1.89 and leukocytosis to 14.6, but are otherwise unremarkable; he has normal electrolytes, renal function, and coagulation studies, without anemia or thrombocytopenia. His ECG demonstrates a sinus tachycardia without ischemic voltage changes, and his chest X-ray is normal. He is incidentally found to be COVID-19 positive despite no respiratory complaints or pulmonary findings on physical exam. Plain films of his right lower extremity show no gas or periosteal reaction, with soft tissue swelling from the calf to forefoot and multiple vascular surgical clips in place.

This is when it became clear that perhaps a leading diagnosis of necrotizing fasciitis wasn’t quite as certain as anticipated, and vascular catastrophe quickly became the most likely culprit.

General surgery and vascular surgery were consulted; they evaluated the patient and requested CT angiogram of the abdomen and pelvis with lower extremity runoff. This imaging confirmed a complete occlusion of the right common femoral artery with extension into the aorta, caused by extensive thrombosis of an aortobifemoral graft (Figure 1).

With a diagnosis of acute limb ischemia clinched, an argatroban drip



FIGURE 1. COMPLETE OCCLUSION OF THE RIGHT COMMON FEMORAL ARTERY WITH EXTENSION INTO THE AORTA, CAUSED BY EXTENSIVE THROMBOSIS OF AN AORTOBIFEMORAL GRAFT.

was started. The patient crossmatched for two units of blood and was expediently transported to the operating room under the care of vascular surgery, where a right through-knee amputation was performed along with multiple embolectomies and stents.

HOSPITAL COURSE

The patient was admitted to the ICU for cardiac monitoring and q30 minute right femoral pulse checks. He was successfully extubated on post-operative day 1.

On post-op day 2, however, the patient developed hypotension with an elevated jugular venous pulse to his earlobe, dependent edema, and new bibasilar crackles on lung auscultation. ECG showed new T-wave changes without ST-segment elevation, but repeat labs revealed a new serum troponin elevation — this patient was experiencing an NSTEMI. A newly elevated BNP of 6,027 prompted a transthoracic echocardiogram, which showed a left ventricular ejection fraction of 40 percent. A repeat troponin was downtrending and the patient denied chest pain or any other symptoms, so the ICU team diuresed with Lasix, made the patient NPO, and prepared him for cardiac catheterization the following morning.

Cardiac catheterization revealed chronic total occlusion of the right circumflex artery (RCA). The myocardium typically perfused by the RCA had developed retrograde collateral flow from the left anterior descending artery (LAD) to compensate; however, this patient's new hypercoagulable state and the physiologic stress of his ischemic limb had led to severe LAD stenosis, causing his NSTEMI. Two LAD stents were placed, the patient stabilized, and he was returned to the ICU. An above-knee amputation was completed two days later (delayed due to the NSTEMI) with right groin washout and wound vac placement. The patient then underwent careful medical optimization with daily physical and occupational therapy (PT/OT). Ultimately, this patient was discharged to a nursing and rehabilitation center for continued PT/

OT and daily Eliquis and Brilinta after a 19-day hospital admission.

DISCUSSION

Acute limb ischemia (ALI) is a thromboembolic condition that occurs when arterial supply to the affected extremity becomes critically compromised, limiting perfusion and threatening limb viability if not emergently identified and managed. Thrombotic events are four times more likely to be the cause of an acutely ischemic limb than an embolic event.¹ The lower extremity is 10 times more likely than the upper extremity to experience a thrombotic event, and three times more likely to experience an embolic event.⁴ Collateral vessels may develop in patients with underlying thrombosis; however, these collateral vessels cannot effectively perfuse the limb once complete primary vessel occlusion occurs.² Prompt diagnosis and intervention is critical, with as many as 10 to 15 percent of patients hospitalized for acute limb ischemia ultimately requiring amputation of the affected extremity.³

ALI primarily occurs in patients with a history of peripheral vascular disease, atrial fibrillation, large vessel aneurysm, or hypercoagulable states (e.g., COVID-19).¹ The classic presentation of an acutely ischemic limb includes the “6 P’s”: unilateral pain, pallor, paresthesia, paralysis, pulselessness, and poikilothermia. However, these are late findings in extremities at high risk of amputation.⁶ Severity is graded according to the Rutherford classification scale,¹ which is used by vascular surgery to drive their management plan with regards to how salvageable the limb may or may not be. Time is tissue, as the saying goes, and prompt diagnosis is critical — irreversible necrosis begins as early as six hours after the primary vessel becomes occluded.⁵ While an ankle-brachial index may be performed, CT angiography is the diagnostic study of choice due to its high sensitivity and specificity.¹

CLINICAL PEARLS

Acute limb ischemia is a devastating



condition if not expediently recognized and managed. While acute bullae formation are typically associated with primary infectious, dermatologic, or autoimmune etiologies (e.g., necrotizing fasciitis, SJS/TEN, bullous pemphigoid), acute limb ischemia may also present with these findings and must be considered.

Once diagnosed, anticoagulation should be initiated and vascular surgery promptly consulted for either medical or surgical management. Direct thrombin inhibitors such as argatroban should be substituted when heparin is contraindicated, such as in this patient with HIT.

It is critical to understand that a patient who is hypercoagulable enough to cause acute limb ischemia is at high risk for thromboembolic disease elsewhere, and clinicians must have a high index of suspicion for impending complications such as coronary artery disease, stroke/TIA, pulmonary embolism, etc., in these patients, as evidenced by this patient's NSTEMI during his ICU stay. ★

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



TCA Overdoses and Mimics

Don't Delay, Don't Be Led Astray

Lance Shaul, MD

Resident, PGY-2
Emergency Medicine
Boston Medical Center

Andrew Mittelman, MD

Assistant Professor
Emergency Medicine
Boston Medical Center

EMS ARRIVED WITH A 38-YEAR-OLD MALE WHO WAS FOUND DOWN BY HIS ROOMMATE. The patient was unresponsive with an irregular respiratory rate of 20. He was in a wide-complex tachycardia with a Glasgow Coma Score of 3. Narcan was given with no effect. The patient was intubated on scene with rocuronium and etomidate before ketamine was administered for sedation. There was a rhythm change en route to the hospital. EMS 12-lead showed a regular, wide-complex tachycardia with RsR' ST-elevation in aVR and diffuse t wave changes. There was rightward axis deviation, and the QRS was widened to 138 milliseconds.

EKG FINDINGS: A RED HERRING?

QRS prolongation and a terminal R' should raise concern for tricyclic antidepressant (TCA) overdose, a diagnosis that often must be made on EKG alone due to a quick clinical progression and a high mortality rate. In cases of TCA overdose, a QRS greater than 100 ms is predictive of seizures, and as many as 50% of patients with a QRS over 160 ms will experience ventricular arrhythmias.¹

TCAs exert their therapeutic effect by inhibiting the reuptake of norepinephrine and serotonin. At toxic doses TCAs also block sodium channels, impeding sodium entry into myocardial cells. Slower sodium entry delays membrane depolarization, therefore

decelerating ventricular contraction and prolonging the QRS. The right-sided intraventricular conduction system is more susceptible to sodium channel blockade than the left bundle. The R' seen in TCA overdose is most prominent in aVR because the right ventricle contracts more slowly than the left ventricle.^{1,2}

In addition to arrhythmias caused by sodium channel blockade, TCA overdose causes hypotension through several other mechanisms. Anticholinergic effects from TCA muscarinic blockade cause tachycardia and decreased filling time. Alpha adrenergic receptors are blocked by TCAs in the periphery, causing vasodilation. Heart block (most notably right-sided conduction delays including

bundle branch block) and bradycardia are late-stage manifestations, further exacerbating hypotension.³

DECOMPENSATION AND CRITICAL INTERVENTIONS

Within 5 minutes of arrival, the patient lost pulses. CPR and standard resuscitation therapy were initiated. Bicarbonate, magnesium, and calcium administration were also prioritized, and return of spontaneous circulation (ROSC) was achieved after four rounds of compressions. Intermittent QRS prolongation responded well to boluses of bicarbonate throughout the next few hours.

First-line treatment of TCA overdose consists of bicarbonate and

hyperventilation, both of which alkalinize the blood. TCAs are weakly basic, and their ionized effective state is most stable in acidic environments. At a higher pH, TCAs become protein bound, thereby decreasing their chemical activity. In overdose, alkalization therefore minimizes TCA toxicity. Monitoring with serial EKGs is necessary as QRS prolongation can recur up to 72 hours after an initially adequate response to treatment.⁴

The EKG findings of TCA toxicity should prompt the clinician to also consider other etiologies. Right bundle branch block, T wave inversions in V2–V3, and ST elevation in aVR are all associated with increased risk of circulatory shock and death in cases of pulmonary embolism (PE).⁵

UNEXPECTED CAUSES FOUND

Prior to ICU transfer, the patient was sent for CT pulmonary angiography, which showed bilateral pulmonary emboli involving the right distal main pulmonary artery. Reflux of contrast into the IVC raised concern for right heart strain. Serial serum TCA testing was negative. Urine toxicology resulted positive for cocaine and benzodiazepines.

Apart from PE and TCA overdose, additional hypotheses for the case's EKG findings are reviewed below.

Cocaine toxicity can cause sodium channel blockade, an 'S' wave in leads I and aVL, and an 'R' wave in lead aVR. It can be accompanied by an incomplete RBBB pattern in the precordial leads.⁶ Trazodone, which the patient was prescribed, is also known to cause wide-complex tachycardia and heart block in the setting of overdose.⁷ Lastly, hyperkalemia should be considered in any case involving prolonged QRS or unexplained EKG findings.⁸

Repeat potassium on arrival to the MICU (around 4 hours after ROSC) showed an increase to >7. An EKG was taken around this time, prior to treatment. Serial EKGs showed no changes corresponding with this subsequently elevated potassium. (Figures 1 and 2)

TAKE-HOME POINTS

- At toxic doses, TCAs cause blockade of sodium channels, muscarinic receptors, and alpha-adrenergic receptors. Slowed ventricular contraction, tachycardia, and vasodilation cause hypotension in these cases.
- QRS prolongation and a terminal R' should raise concern for TCA overdose,

but may be found in other acute pathologies as well. Consider empiric administration of bicarbonate based on EKG alone.

- Both TCA overdose and PE specifically strain the right ventricle. ST elevation in aVR, often seen in PE, can mimic the classic R' seen in aVR during TCA overdose. ★

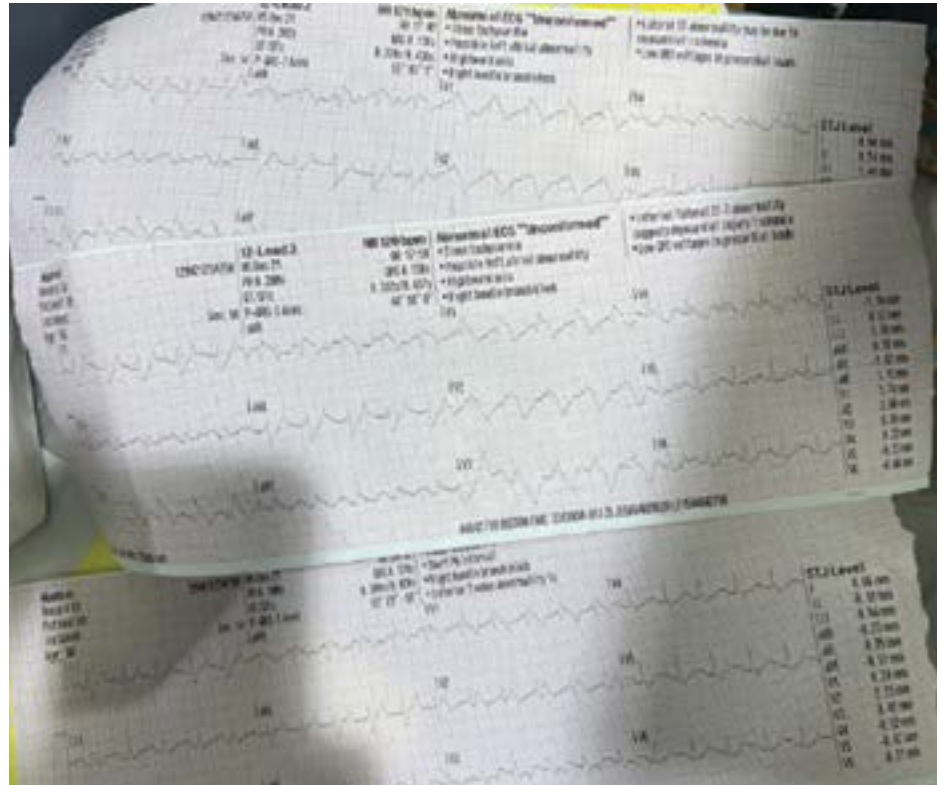


FIGURE 1



FIGURE 2



Pneumocephalus: An Atypical Presentation of Syncope

Avinash Ram, DO

Resident, PGY-3
Emergency Medicine
The Brooklyn Hospital Center

Mollie Williams, MD, MPH

Program Director
Emergency Medicine
The Brooklyn Hospital Center

Omar Hashmi, DO

Attending Physician
Emergency Medicine
The Brooklyn Hospital Center

INTRODUCTION

Pneumocephalus is a condition that is defined as air present in the intracranial space. The disease process is usually seen in patients with disruption of the skull and is a rare finding in the absence of prior neurosurgical intervention or trauma.¹

Cases of pneumocephalus in the absence of trauma have been published in case reports and include underlying

etiologies such as pneumococcal meningitis, CSF leaks, and epidural blocks.² Patients with pneumocephalus can have a wide-ranging presentation from benign to respiratory arrest,³ with the most common presenting complaint being headache.

This case highlights a unique finding of syncope being a presenting symptom of pneumocephalus, which has not been reported before.

CASE REPORT

A 62-year-old female with a history of hypertension (HTN) and chronic shoulder pain presented to the emergency department for evaluation of syncope. The patient was getting an epidural steroid injection and immediately after the injection had a syncopal episode witnessed by office staff. The patient was caught by staff before falling to the ground. Upon EMS

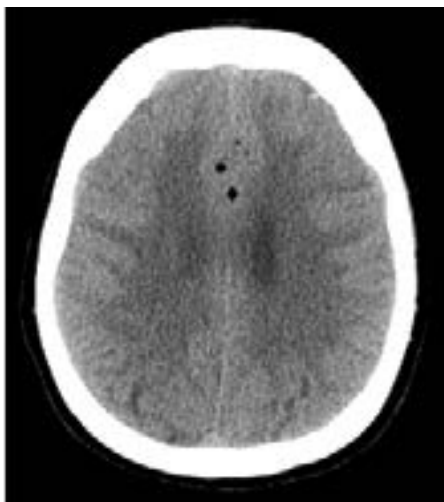


FIGURE 1



FIGURE 2

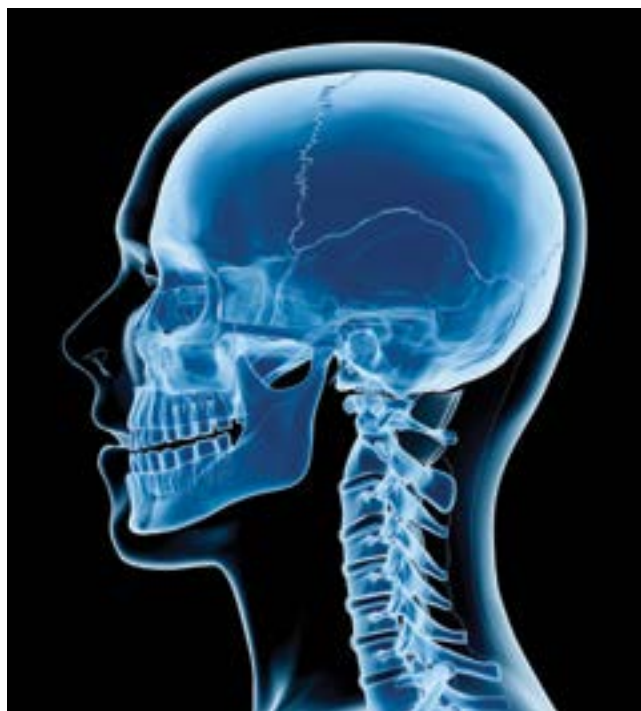


FIGURE 3

arrival, the patient was alert and oriented. Vitals at that time were notable for blood pressure (BP) 70/40 and heart rate (HR) of 40.

Upon presenting to the ED, the patient was mentating well, with vitals showing BP of 106/62 and HR of 68. The patient had no complaints aside from a headache that was currently resolving. A STAT EKG was obtained. It showed normal sinus rhythm at 69 beats per minute, with no acute ischemic changes. Labs were unremarkable. A chest X-ray (CXR) showed no radiographic evidence of acute pulmonary disease. Computed tomography (CT) of the head was performed (Figures 1, 2, and 3), which showed: "Numerous foci of pneumocephalus throughout the brain. Given the history or recent epidural steroid injection, findings may be related to injection of air into the thecal sac during the procedure. There is no significant mass effect of midline shift."

The patient's repeat neurological exam continued to be unchanged from initial baseline presentation. No focal deficits were noted, and the patient only complained of a headache. She remained vitally stable and was transferred to another institution for neurosurgical evaluation and management.



DISCUSSION

Current reported cases of pneumocephalus due to epidural injections have shown that the most common presenting symptom in these patients is an intense headache.⁴ There is one case report that identifies respiratory arrest as the presenting symptom, showing that pneumocephalus can present with a wide range of neurological symptoms, from benign to life threatening.

Pneumocephalus is a known

complication of accessing the epidural space. This can occur from air accidentally being injected into the intrathecal space when the "loss of resistance" technique is used to identify the epidural space.⁵ The presence of air in the epidural space can cause seizures by irritating the cerebral cortex.⁶

In our patient, it is plausible to assume that irritation of the cerebral cortex could have caused her to have a syncopal vs. seizure episode.

CT is the gold standard for diagnosing pneumocephalus and can detect as little as 0.55mL of intracranial gas. Treatment of pneumocephalus is usually conservative, with 85% of reported cases reabsorbing spontaneously without intervention during the

first week of developing the condition.⁷

It is important to keep a high clinical suspicion in the appropriate clinical environment, as this is a rare complication in the non-traumatic setting, with the main barrier to diagnosis being a low index of suspicion. This case highlights the importance of early consideration, recognition, and treatment of pneumocephalus, especially given the low incidence rate and likelihood for atypical presentations outside of the traumatic setting. ★

Orthostatic Vital Signs: Have They Fallen Out of Favor?

E. Daxton Spencer, DO, MS

Resident, PGY-2
Emergency Medicine Residency
Central Michigan University

Lindsay Davis, DO, MPH

Resident, PGY-2
Emergency Medicine Residency
Central Michigan University

CASE

A 46-year-old female presents to the emergency department with a chief complaint of syncope, continuing to feel unwell and lightheaded. Her vitals upon arrival are: blood pressure 138/84; pulse 74; respirations 16; and oxygen saturation 98% on room air. The patient was working outside, serving hotdogs at a concession stand during the summer. She has a history of depression and hypercholesterolemia, but otherwise an unremarkable history. Are orthostatic vital signs useful in the workup of this patient?

BACKGROUND

When performed correctly, orthostatic vital signs measure blood pressure and pulse changes in a sequence of lying, sitting, and standing, waiting 3 minutes between each position.¹ A person is said to be “orthostatic positive” if one of the following is true: a reduction of systolic blood pressure of at least 20mmHg; a reduction in diastolic blood pressure of at least 10mmHg; or an increase in heart rate by >30 beats per minute, when comparing lying to standing.^{1,2}

The pathophysiology of orthostatic hypotension is that people, when standing, have pooling of blood in both the lower extremities and splanchnic circulation. This initiates a cascade to quickly increase cardiac output to limit blood pressure decrease through increased sympathetic outflow. Orthostatic hypotension results when these compensatory mechanisms are inadequate, leading to decreased cerebral perfusion pressure and, subsequently, symptoms such as syncope and lightheadedness.³

When patients are found to be orthostatic positive, fluid loss or

decreased volume status is assumed most likely, and the go-to treatment is usually fluids, most commonly intravenous.^{1,3}

Although this is the typical use of orthostatic vital signs, their utility has been debated in literature and in the emergency department for decades. Supporters point to orthostatic vital signs as being useful in determining fluid loss as a cause of syncope, lightheadedness, and vertigo.³ They further tout their use as a risk-stratification tool in the elderly population.^{4,5} Those opposed say a patient can still be hypovolemic and in need of treatment for such, but not have orthostasis.¹ Another risk is the potential pitfall of orthostatic vital signs causing anchoring bias of hypovolemia as the cause of syncope.⁶

SUPPORT

With syncope, patients commonly present with multiple symptoms, such as lightheadedness, palpitations, tremulousness, generalized weakness, blurred vision, and fatigue.³ These can be vague symptoms, and orthostatics give objective data to accompany these subjective symptoms and to support the diagnosis. Proponents argue that obtaining orthostatic vitals can help differentiate orthostatic causes of syncope versus non-orthostatic causes, such as autonomic peripheral neuropathies, pure autonomic failure, multiple system atrophy, and more.^{2,7,8} Positive orthostatic vital signs can help tailor treatments, such as more frequent hydration or medication review, particularly in those with polypharmacy or in the elderly, who may be more sensitive to medication changes.^{2,3,6}

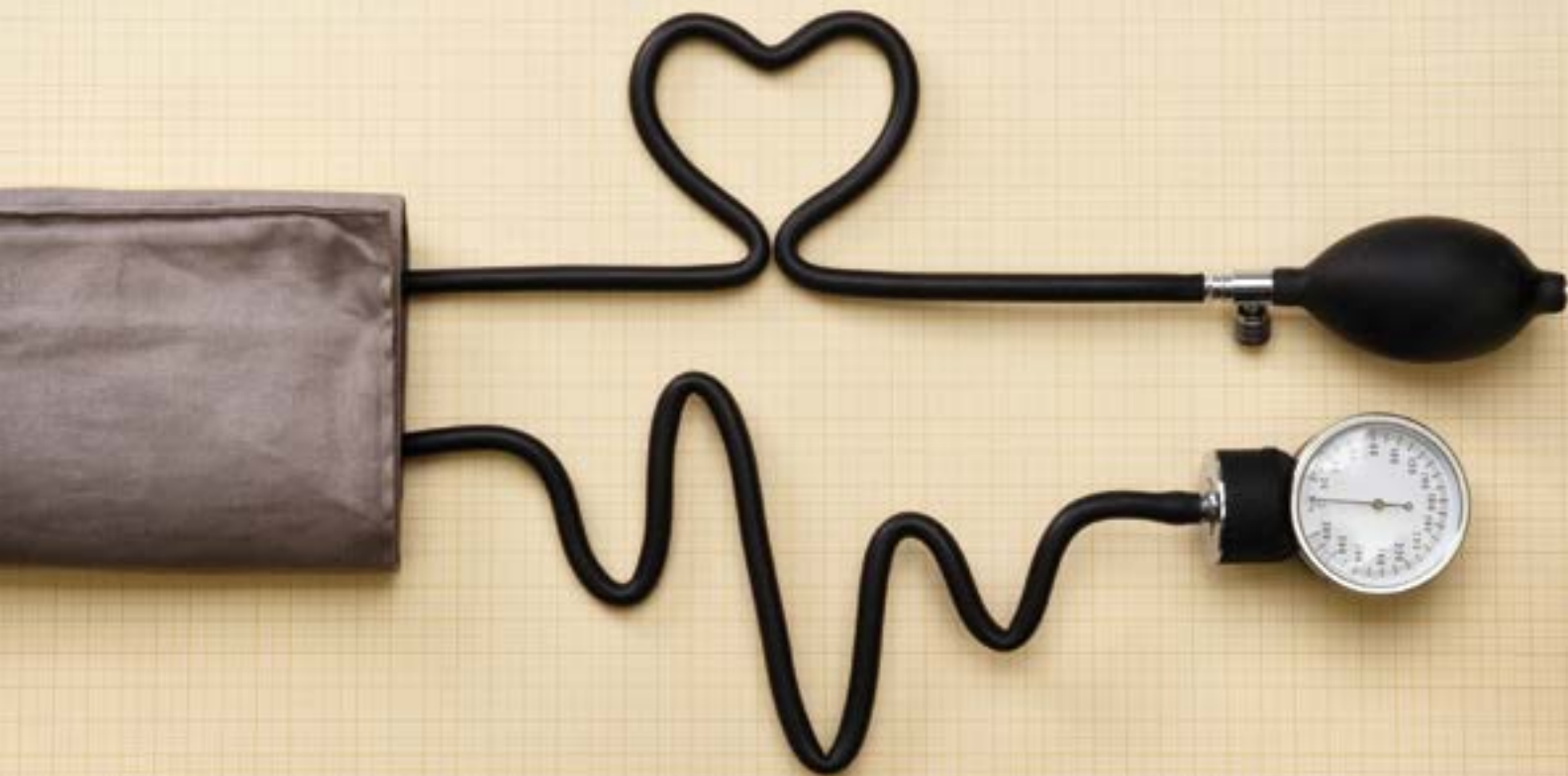
The 2017 American Heart Association (AHA) Guidelines on syncope stress the importance and need for obtaining

orthostatic vital signs along with a thorough medical history and physical exam, particularly in older patients.² One prospective single-center study of more than 800 ED patients by Cohen and colleagues showed that orthostatic vital signs can be helpful to risk-stratify the elderly population, as patients 75 years and older who were orthostatic positive in the ED had an increased risk of hospitalization, all-cause mortality, and falls.⁴ More support for orthostatic vitals, a cost-effective and non-invasive test, comes from a retrospective study in which 2100 patients 65 years and older underwent analysis of workups for syncope. Only 38% of the 2100 patients had orthostatic vital signs performed; however, when performed, the tests helped inform diagnosis, management and etiology of syncope in over 30% of cases.⁹ Compare those findings with CTs, which were obtained in 63% of cases, and electrocardiograms and cardiac enzymes, which were obtained in greater than 95% of cases; these diagnostics changed management and diagnosis in less than 5% of cases.⁹

AGAINST

While support for orthostatic vital signs shows them to be cheap, non-invasive, and particularly useful to help differentiate causes of syncope, their use has been called into question.

Skeptics believe that orthostatic vital signs are not useful and can even be dangerous, as they may cause anchoring bias and lead the physician to stop looking for further causes of the patient’s symptoms. White and colleagues concluded that orthostatic vital signs have no role in diagnosing life-threatening causes of syncope, such as stroke or cardiac arrhythmia, and can



Just as there are two sides to every coin, some medical literature supports taking patients' orthostatic vitals when they present to the ED with syncope, lightheadedness, and vertigo; other literature takes a different view. Although patients found to be orthostatic positive are usually treated with fluids, consider that many emergent diagnoses are associated with orthostasis. It is best to keep your differential large and consider the whole scope of causes.

lead to misattribution to orthostasis.¹⁰ White's study, a prospective multi-center observational analysis of 11 emergency departments, evaluated adults 60 years and older presenting with syncope and near syncope and showed orthostatic vitals had no independent correlation with 30-day adverse outcomes such as mortality; this was in direct contrast to the study by Cohen, which showed increased mortality.^{4,10} In the same study in which Cohen showed that orthostatic

vital signs can be used to risk-stratify the elderly, it was also found that 25% of people presenting to the ED were orthostatic positive, regardless of their complaint.⁴

As discussed initially, orthostatic vital signs are used to determine fluid status; however, in one small single-center study, orthostatic vital signs were performed on 23 pregnant patients with hyperemesis gravidarum, and the tests were found to have inadequate sensitivity

for determining volume status.¹¹

In another study, Aronow evaluated 476 patients older than 60 years of age, living in long-term care facilities. This population was found to have significant variations in orthostasis throughout the day, with higher likelihood of orthostatic hypotension presence in the morning before breakfast.¹² Aronow concluded that in the non-acutely ill, there are significant cofounders, many coexisting (such as antihypertensives and beta-

blockers), that make orthostatic vital signs less useful in this population.¹²

In a prevalence study of more than 900 long-term nursing home residents, Ooi and colleagues also showed that older patients are more likely to have baseline abnormal orthostatic vital signs due to medications and autonomic dysfunction, and the finding of orthostasis in the ED may be unrelated to the cause of syncope.¹³

In adolescents, a descriptive population study of 307 high school students proved orthostatic heart rate changes are common in euvoletic teens, and their blood pressures tend to fall within normal adult limits even if they are hypovolemic.⁵ Another study looked at 23 healthy adolescents and found that transient orthostatic hypotension is common as well in this population, attributed to underdeveloped vasoconstriction, with standing.¹⁴

If a patient is found to have positive orthostatic vital signs, there is a

significant concern of anchoring bias.^{6,10} A physician could potentially treat the patient with intravenous fluids and not do further testing, potentially leading to a missed diagnosis.

DISCUSSION

The argument, given the data, is whether orthostatic vitals add anything to the evaluation and treatment of patients in the emergency department. Do we need to perform them? The AHA supports the use of orthostatic vitals in its 2017 update, and Cohen and colleagues demonstrated risk stratification for the elderly population.^{2,4}

Despite this, as noted in a study by Mendu and colleagues, orthostatic vitals are not performed in even half of syncope cases.⁹ In contrast, Ooi, Skinner, and Stewart found that orthostatic hypotension was common in non-acutely ill patients belonging to various age groups. Cohen even noted that, despite increased risk in the elderly, 25% of

all ED patients were orthostatic upon evaluation.^{5,13,14}

Despite these conflicting studies, we should likely err on the side of caution and perform orthostatic vital signs more often, in conjunction with other diagnostics, to form a more complete picture of a patient's volume status and risk factors.

As research continues, better alternatives to the use of orthostatic vitals may be discovered or refined, such as the Shock Index.¹⁵ The Shock Index is defined as the heart rate (HR) divided by systolic blood pressure (SBP). A result of >1 has been widely found to predict increased risk for mortality; the normal range is 0.5 - 0.7. Koch discusses that while heart rate and blood pressure have been traditionally used to characterize shock, they can appear normal due to confounding factors such as medications, leading to a delay in treatment.¹⁵ Research is promising for the Shock Index; however, a limited number of



studies performed leads to inadequate data to support its use as a replacement for orthostatic vitals, at this time.

Routine evaluation of patients' hydration status through intake and output, as well as evaluation of medications for effect on blood pressure, could give more accurate and reliable data, but these methods are likely not possible for patients being discharged.³ Future research into orthostatic vitals and alternatives need to include further prospective studies. These could include using orthostatic vital signs and following outcomes when used versus not used in a patient's evaluation and management.

CONCLUSION

Just as there are two sides to every coin, some medical literature supports taking patients' orthostatic vitals when they present to the emergency department with syncope, lightheadedness, or similar complaints; other literature takes a different view. However, as a low-cost and low-resource test, orthostatic vital signs should be used more often to help inform diagnosis and management of syncope.

If better alternatives, such as



the Shock Index, are proven to help elucidate syncope etiology and inform management, then maybe use could fade. Caution must be taken not to anchor on hypovolemia as a cause of orthostasis;

many emergent diagnoses can have orthostasis associated with them. Always keep your differential large and work up other causes before only giving fluid and discharging the patient. *

CASE RESOLUTION

You decide to obtain orthostatic vitals, ECG, BMP, and CBC. Orthostatic vitals before treatment are:

Lying - blood pressure 134/92, pulse 71

Sitting - blood pressure 130/84, pulse 83

Standing - blood pressure 108/76, pulse 97

EKG - NSR at 74 bpm

You decide to give the patient 1 L of normal saline IV. Her BMP and CBC were unremarkable. You recheck orthostatic vitals after treatment, and they are as follows:

Lying - blood pressure 130/90, pulse 74

Sitting - blood pressure 126/84, pulse 75

Standing - blood pressure 122/78, pulse 80

You re-evaluate the patient as she is no longer orthostatic. On re-evaluation, she is still complaining of lightheadedness and dizziness. You obtain a CT scan of the brain, which is normal. You give the patient meclizine, which improves her symptoms. You diagnose the patient with orthostatic hypotension secondary to dehydration, and she is discharged with strict return precautions and told to follow up with her family physician. This turned out to be a straightforward case of orthostatic hypotension related to dehydration, but it is important not to anchor and to consider electrolytes imbalance, cardiac arrhythmia, and more.

Drowning in a Flash: A Review on Neurogenic Pulmonary Edema

Bryce Taylor, DO, MS

Resident

Emergency Medicine

Carilion Clinic, Virginia Tech Carilion

You're at the head of the bed when a trauma is brought in. A man in his early 30s is moved over to your stretcher. His head is wrapped in gauze that is saturated in blood. You're told that he has two apparent ballistic wounds, one to his right temple and one to his left. He has been intubated in the field and is oxygenating well with no overt secretions in his tube. Aside from his dismal neurologic exam, he is hemodynamically stable. You run through the trauma resuscitation, and the patient is ultimately moved to the CT scanner and then to an ED room while awaiting a surgical ICU bed.



Once in the room, the patient suddenly becomes hypertensive and tachycardic. You push a dose of Labetalol and call for a Nicardipine drip. A few seconds later, the patient's oxygen saturation drops to the 60s, and the ventilator alarms due to high pressures. He is deep-suctioned with return of copious bloody, frothy secretions. His pulse ox continues to drop. You move him to the bag valve to no avail. He becomes increasingly difficult to bag. The next few minutes consist of frantically suctioning whatever you can and trying to bag. His blood pressure then drops, his pulse is lost, and CPR is begun. Despite the heroic efforts the team puts in coding him, he ultimately succumbs to his injuries, and the time of death is called. You look at the suction canister and see 800 cc's of bloody, frothy sputum.

While this patient would have likely succumbed to his devastating neurologic insult, the immediate cause of his death was severe and sudden pulmonary edema. This patient experienced a phenomenon known as neurogenic pulmonary edema (NPE), which was first described by Shanahan in 1908 and can occur with any neurologic insult.¹ Presentation of NPE can be rapid or delayed and presents similarly to an acute respiratory distress syndrome (ARDS) picture.¹

EPIDEMIOLOGY

NPE can occur in any patient who has sustained a central nervous system (CNS) insult.¹ This phenomenon is likely underreported and is most often described in case and autopsy reports.² NPE is also associated with a high mortality, with one large population study evaluating deaths among traumatic brain injury patients showing that 32% of this population had developed NPE, increasing to 50% if death occurred within 96 hours of the initial insult.³ Also notable, studies have found that more than 80% of those who die from status epilepticus are found to have developed NPE as well.⁴

While NPE can occur from any central nervous system insult, it can be most expected among those who have sustained a sudden CNS insult that led to an abrupt increase in intracranial

pressure (ICP).^{1,3} NPE is most seen in subarachnoid hemorrhages and head trauma.¹ Risk factors associated with the development of NPE include increasing extent of injury, increasing age, delay to treatment, vertebral artery origin or involvement, and status epilepticus.¹ NPE can also occur post-operatively in neurosurgical patients, particularly

Neurogenic pulmonary edema, associated with high mortality, can occur after any neurologic insult and is best addressed by controlling intracranial pressure.

in those who have undergone a tumor resection or are experiencing a shunt malfunction.⁵ Any process that leads to an extreme elevation in ICP can precipitate NPE.^{1,3}

PATHOPHYSIOLOGY

There are four main theories for the etiology and pathophysiology of NPE; however, its development is likely a combination of multiple inciting processes.¹ It is widely accepted that there is an interplay of the neurologic, cardiac, and pulmonary systems; however, there is potentially an inflammatory component as well. It is likely that there are still many aspects of this disease process that remain undescribed.

The first major mechanism proposed is based on the neurocardiac axis. This mechanism is proposed to occur after a CNS insult leads to a massive catecholamine release causing reversible damage to the myocardium, leading to decreased cardiac output, backup of blood into the pulmonary vasculature, and leakage of fluid into the alveoli, known as pulmonary edema.^{1,3} This mechanism is likened to Takotsubo cardiomyopathy.³ Research has supported this mechanism by showing an increased occurrence of echocardiographic segmental wall abnormalities, including decreased left ventricular ejection fraction, elevated myocardial enzymes in the serum, electrocardiographic changes, and histologic signs of contraction band

necrosis post-mortem in NPE patients.^{6,7}

The second theory proposed is the neurohemodynamic mechanism. A sympathetic surge is also central to this theory. Following a CNS insult, there is a massive catecholamine release that affects both the heart and the vasculature. The increased sympathetic tone leads to an initial increase in left

ventricle function; however, it quickly crosses its optimum functional threshold and begins to fail. In addition, systemic vascular resistance is increased, worsening the demand of the left ventricle. This causes a shift in blood into the pulmonary circuit, where there is a buildup of hydrostatic pressure, forcing fluid from the pulmonary vasculature into the alveoli.^{1,3} This alone, however, would not explain the protein and red blood cells found in the translocated fluid.

The blast theory is the third major mechanism. This proposes that some combination of the mechanisms described above leads to increased capillary pressure which, in addition to barotrauma, leads to irreversible damage to the endothelial lining of pulmonary capillaries and the alveolar membrane.^{1,3,8} This leads to leakage of fluid as well as protein and red blood cells. This mechanism is supported by case studies showing persistence of pulmonary vasculature damage even after reversal of all other components of NPE.⁹

The fourth mechanism proposed is the pulmonary venule adrenergic hypersensitivity theory.^{1,9} This theory posits that the massive catecholamine and chemical signal release following a CNS insult causes direct damage to the pulmonary vasculature. With this mechanism, pulmonary edema would develop regardless of systemic vasculature changes.^{1,9} This is supported by the fact that pulmonary vascular beds have adrenergic receptors. Following



a catecholamine surge, the pulmonary vascular beds can be stimulated to vasoconstrict, which can ultimately lead to endothelial damage.¹⁰ Other molecules of note are endothelin-1, which is released as an acute phase reactant following CNS insult, and neuropeptide-Y, which is released into the CSF and blood following CNS injury.^{11,12} Both are also potent vasoconstrictors.^{11,12} Vasoconstriction and endothelial damage can then precipitate NPE.¹

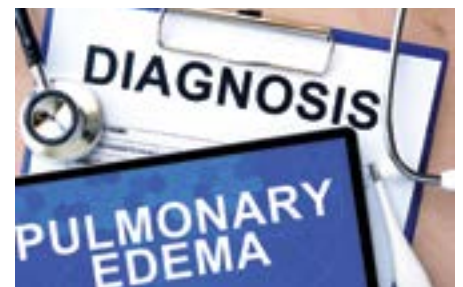
There is also evidence of an inflammatory component to NPE. Following injury to the brain, there is a substantial release of cytokines.¹³ These cytokines can cross the blood brain barrier due to its loss of integrity following the injury. They can then travel to the lungs where they both act directly on pulmonary vasculature as well as stimulate release of other cytokines. This response will lead to an inflammatory

lung injury, including endothelial leakage allowing for translocation of fluid from capillaries into the alveoli. Hyperventilation may also contribute to this mechanism.¹³

There is no single mechanism proposed that is widely accepted as being the sole cause of NPE. Rather, it is likely that NPE occurs as a result of a combination of the mechanisms described above and additional processes yet to be described.

CLINICAL CHARACTERISTICS

NPE is characterized as occurring in either early or late phases.³ NPE in the early phase occurs most often, with an onset of 30 to 60 minutes following neurologic insult. NPE is a clinical diagnosis in both stages. It is important to rule out ventilator-associated pneumonia, aspiration pneumonitis, sepsis-related ARDS, transfusion-related acute lung injury, heart failure, and post



airway obstruction pulmonary edema.³

Clinically, the patient will have a sudden onset respiratory distress and hypoxia, and start producing pink and frothy sputum.³ Patients who are intubated may require frequent suctioning. A chest X-ray will show bilateral opacifications. An important aspect of NPE is that if the patient survives initial decompensation, there should be a rapid resolution after 48-72 hours.³ If the pulmonary edema does not begin to improve, evaluation for other causes should be investigated.³



MANAGEMENT

NPE is ultimately treated by addressing the neurologic process causing the increased ICP.¹⁴ Patients are typically mechanically ventilated if they have suffered an insult significant enough to cause NPE, and if they are not, then intubation should be considered to optimize pulmonary supportive care, providing adequate PEEP and frequent suctioning.¹⁴

Controlling the ICP is essential in managing patients with NPE. Patients should be maintained with normotension, normocapnia, and normothermia. Hypertonic infusions may be necessary to promote osmotic diuresis. Seizure prophylaxis is also often warranted following a severe neurologic insult, as seizures can increase ICP.¹⁴ If possible, placement of an external ventricular drain or a cranial decompression may be necessary. If the ICP is continually unmanageable

despite aggressive resuscitative measures, the patient may require a barbiturate coma which decreases ICP by decreasing cerebral metabolism and oxygen demand, leading to a lower blood volume.^{14,15}

It should be noted that there are several methods that are contraindicated in traumatic brain injuries.¹⁴ Hypercapnia should only be used as a temporizing measure, if used at all, and continued hyperventilation has been associated with increased ICP.¹⁴ Fluid restriction is not effective in treating NPE and may lead to hypotension which can increase ICP.¹⁴ Proning of a patient may also lead to an increase in ICP.¹⁴

PROGNOSIS

The prognosis associated with patients who develop NPE is generally poor. Mortality rates in patients developing NPE have been recorded to be between 60 and 100%.³ Early recognition and

initiation of supportive measures in patients developing NPE is crucial. ★

TAKE-HOME POINTS

- NPE is a clinical diagnosis. It is essential that other pathologies that may require different treatments be excluded.
- Support your patient through NPE while addressing the neurological insult.
- Understand and become comfortable with non-surgical methods in decreasing ICP, such as the use of hypertonics, hemodynamic optimization, and pharmacologic interventions.
- NPE is associated with a very high mortality. ★



One Health and Climate Change: Why EM Physicians Should Pay Attention

Alison Neely

Medical Student, MS-4
Albert Einstein College of Medicine

Kyle Denison Martin, DO, MPH

Assistant Professor
Emergency Medicine
Alpert Medical School of Brown University

“Is the scene safe?” That’s something we’re taught to ask during our earliest days of medical school, as we learn how to perform basic life support.

While the context of that question is mostly about our physical safety as providers, it can also be asked in a larger sense: What else is going on in the environment around my patient? No patient or case exists in a vacuum, and understanding how the environment can affect the care we provide can increase our chances of success. This goes for the hospital environment as well as the greater environment around us. And this is where the One Health approach comes in.

The One Health approach focuses on the interconnections of human health, environmental health, and animal health, and recognizes that all of these factors need to be addressed to optimize the overall health of the world in which we live and work.¹ This concept is not new, but it is increasingly important that we as practitioners of medicine recognize the importance of the close connections our communities and patients have to the animals around

us and our shared environment. For emergency medicine physicians, the impacts of this approach are far reaching, from increased antibiotic resistance and water contamination to improvements in mental health that come from human-animal bonding.

As we examine how these elements affect the communities that we care for, there is one complicating factor that cannot be ignored: climate change. The impacts of a changing climate on

human health are numerous and will significantly influence physical health, mental health, and social stability.² The effects of these shifts on vector-borne and waterborne infectious diseases offer an excellent example to illustrate the important interconnections at play in our ecosystems — and why we, as emergency physicians, should pay attention.

Dengue, a vector-borne disease with four distinct serotypes and severity that can range from asymptomatic to severe or fatal, has had a 30-fold increase in incidence in the past 50 years. In addition to changes in the climate, this increased caseload has been connected to population growth, urbanization, increased travel, inadequate public health infrastructure, and ineffective vector control.³ Transmission of dengue occurs primarily by the mosquito species *Aedes aegypti*, and the transmission cycle of the virus has been shown to be strongly determined by climatic factors.³ Temperature, humidity, and rainfall have direct effects on not only range, density, and vector efficiency of *A. aegypti*, but also on the rate of virus multiplication and transmission. Increased temperature extends the global range of *A. aegypti*, increases the population density of

the mosquito within that range, and, along with changes in relative humidity, decreases the length of the *A. aegypti* life cycle.³ This shortened life cycle produces smaller adult mosquitos with less energy reserve, requiring them to feed more often and thus increasing the biting rate of the mosquitos — and the more the mosquitos bite, the higher the chances of dengue transmission.³ While the specific mechanisms of this shift hold importance, the greater lesson to take away is how a small change in temperature or humidity can have profound effects on the number of dengue infections we see in the people that we care for, both in the United States and abroad.

The transmission of cholera, which continues to be one of the biggest threats to global health in low-income and developing countries, will also be affected by temperature and rainfall shifts that come along with climate change.⁴ As a food and waterborne disease, cholera is heavily tied to inadequate sanitation access, poor hygiene, and food contamination, but the climate change connection must be part of the public health approach to disease prevention and treatment.⁵ The connections between

climatic factors and cholera transmission are less consistent than with dengue, but there are clear associations. In some global regions, increased temperature is positively correlated with disease transmission, whereas elsewhere a higher air temperature means lower transmission.⁴ The same is true for rainfall; monsoons can lead to increased cholera transmission, as can droughts.⁴ This echoes the unpredictability of climate change itself and emphasizes how important it is for physicians who will work in these regions to understand how the environment plays a role in the practice of medicine.

Talk about climate change and its effects can feel incredibly daunting and can seem out of our control. We do need to make systemic changes and work to mitigate the climatic shifts themselves, but we as providers can also do our part simply by implementing the One Health approach and increasing our awareness of the environment around us.

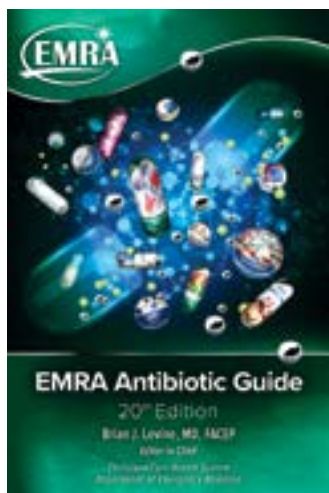
Successful interventions must incorporate human, environmental, and animal cooperation; the health of one cannot come without the health of others. Our patients, and our world, could be much better for it. ★



News & Notes in Emergency Medicine

UPDATES IN EMRA'S CLINICAL LINE

In keeping with EMRA's goal to help you be the best doctor you can be, three new editions of EMRA clinical resources have been released this summer.



EMRA ANTIBIOTIC GUIDE, 20TH ED.

The bedside resource you need for every shift hit a milestone this year, marking the 20th edition of the **EMRA Antibiotic Guide**. (Look closely at the cover to see the history of this vaunted guide.)

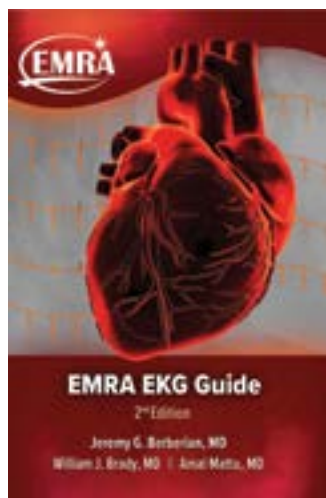
Brian Levine, MD, led the faculty and residents of **ChristianaCare** in developing the content, along with infectious disease and pharmacy experts **Nicole Harrington**, PharmD, BCPS AQ-ID, **Bryan Hayes**, PharmD, DABAT, FAACT, FASHP, and **Jamie Rosini**, PharmD, MS, BCCCP, BCPS, DABAT. The team leveled up for the new edition, updating guidance in every chapter, and adding a chapter on tuberculosis.

"We are so proud to continue our work on this guide. It's a true collaboration with our colleagues in infectious disease pharmacy who are extremely knowledgeable," Dr. Levine said. "We are careful to develop the best recommendations sourced from the literature and specialty society guidelines to guide your care in the emergency

department. And because we're always looking to improve, we continuously update the app between print editions. We think this is the best edition yet!"

The EMRA Antibiotic Guide is a free benefit in every EMRA Member kit. Additional copies are available at www.emra.org/amazon, and EMRA members get a discount when purchasing through the ACEP Bookstore (must log in to see the discount).

EMRA appreciates US Acute Care Solutions for their support of this resource.



EMRA EKG GUIDE, 2ND ED.

The **EMRA EKG Guide** provides context and clinical guidance for your next steps after interpreting an EKG on-shift. The new edition builds on the success of the inaugural book, offering clarity while highlighting the nuances of EKG interpretation.

Editor-in-chief **Jeremy Berberian, MD**, and editors **William Brady, MD, FACEP**, and **Amal Mattu, MD, FACEP**, focused on updated evidence to guide management on-shift. The print version is supplemented by an app within MobileEM.

Why do you need this? Dr. Berberian offers this:

Beans, beans – they're good for your heart.

So is the EKG Guide, so put one in

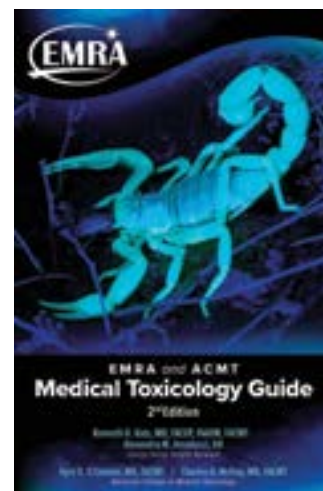
your cart.

The more you read it, the smarter you'll feel.

You should use this for every heart you heal.

The EMRA EKG Guide is a free benefit in EMRA Resident Member kits. Additional copies are available at www.emra.org/amazon, and EMRA members get a discount when purchasing through the ACEP Bookstore (must log in to see the discount).

EMRA appreciates DFES for their support of this resource.



EMRA AND ACMT MEDICAL TOXICOLOGY GUIDE, 2ND ED.

When consulting a regional poison control center, build your presentation and your plan based on guidance from the **EMRA and ACMT Medical Toxicology Guide**. The new edition introduces chapters on vaping, vitamin usage, medication-assisted therapy, household exposures, and all the core content contained in the inaugural book.

Led by editor-in-chief **Ken Katz, MD, FACEP, FACMT**, and associate editor **Alexandra Amaducci, DO**, the chapters were developed and researched by the residents and faculty of **Lehigh Valley Health Network**. ACMT board members **Ayrn O'Connor, MD, FACMT**, and **Charles McKay, MD, FACMT**, spearheaded the team of senior

toxicology experts who reviewed and enhanced the material.

This collaboration with ACMT has set the benchmark for bedside guidance of toxicology-related conditions, Dr. Katz said. The app version within MobileEM will be continually updated in between print editions.

The EMRA and ACMT Medical Toxicology Guide is a free benefit in EMRA Resident and Alumni Member kits. Additional copies are available at www.emra.org/amazon, and EMRA members get a discount when purchasing through the ACEP Bookstore (must log in to see the discount).

EMRA appreciates BTG for their support of this resource.

SEPTEMBER'S EM DAY OF SERVICE: HOW WILL YOU PARTICIPATE?

Emergency physicians, residents, nurses, physician assistants, and medical students are servant leaders in our communities. We care and advocate for our patients while working clinically. We

also respond to the call to give back to the communities we serve.

The EM Day of Service

– celebrated throughout the month of September each year – was created with this essential concept in mind. The EM Day of Service is a specialty-driven initiative during which emergency care providers identify community needs and volunteer to address those needs. From shelter and food to clean and healthy outdoor environments, all efforts highlight the role EM can play in public health.

Please share your efforts on social, and tag @emresidents and #EMDayofService.

ABEM CERTIFICATION: UPCOMING DATES

The American Board of Emergency Medicine sets the gold standard for ensuring EM physicians meet educational and professional



requirements. The externally developed certification exams occur on a regular cycle. Take note of the next dates:

- ABEM Qualifying Exam: Oct. 31-Nov. 5
- ABEM Oral Board Exam: Sept. 21-24

MEDICAL STUDENT FORUM

The EMRA Medical Student Forum takes place virtually Aug. 13, beginning at 9:30 am Central. This event brings together program directors and faculty to answer questions specific to your phase in training. It's free for EMRA members. Register and get more info online at emra.org

VIRTUAL RESIDENCY PROGRAM FAIR

The EMRA Residency Program Fair will be hosted virtually Aug. 13-18. This event is free for EMRA medical student members and allows medical students to connect directly with residency programs. Register online at emra.org; MS-4s have first access. ★

Celebrating 50 years funding research to support our future EM physicians

EMF Emergency Medicine Foundation®

Join the Resident VIP Club

EMRA Events @ Scientific Assembly **22** SAN FRANCISCO

SEPT. 26 - OCT. 4

Monday, Sept. 26

6 pm EMRA Resolution
Central Review (virtual)

All times listed are Pacific time unless otherwise noted.

Friday, Sept. 30

4-6:30 pm EMRA/ACEP
Leadership
Academy
(invitation only)

7-9 pm EMRA Leader
Meet Up
(invitation only)

Saturday, Oct. 1

8 am - 4:30 pm Committee
Programming

8 am - 5 pm Case-Con
Competition

1-5 pm Medical Student
Council Meeting
(invitation only)

5-7 pm EMRA Job &
Fellowship Fair
(Moscone Center
Hall D)

While EMRA events do not require paid ACEP Scientific Assembly registration, all attendees must adhere to ACEP COVID protocol. ACEP strives to provide meaningful member experiences in a responsible manner. As such, ACEP is requiring that individuals who attend in-person ACEP events be fully vaccinated. An individual is considered "fully vaccinated" when it has been at least 2 weeks since receiving the final dose, as recommended by the manufacturer, of a vaccine that has been authorized by the FDA for use in the United States, including vaccinations that have been approved pursuant to an Emergency Use Authorization.

EMRA event headquarters:
San Francisco Marriott Marquis



Check out all of EMRA's offerings in San Francisco.
Events take place at the Marriott Marquis unless otherwise noted.

Come for the camaraderie - stay for the enlightenment!

Sunday, Oct. 2

- 7 am - 1 pm** EMRA RepCo & Town Hall Meeting
- 9 am - 3 pm** EMRA Resident SimWars
- 2-5:15 pm** Committee Programming
- 5-6:30 pm** EMRA Awards Ceremony
- 10 pm - 2 am** EMRA Party (location pending)

Monday, Oct. 3

- 8 am - 1 pm** Committee Programming
- 1-3 pm** 20 in 6 Resident Lecture Competition
- 5-7 pm** EMRA Airway Stories

Tuesday, Oct. 4

- 7 am - 5 pm** EMRA MedWAR (Competing teams & volunteers only)

See you in San Francisco!

Monitor for updates at
www.emra.org/acep



Join EMRA for the **FALL 2022 ELECTIONS**

Sunday, Oct. 2 • During RepCo (*meeting begins at 8 am*)

ANNUAL BOARD POSITIONS

- President-Elect (*Succeeds to President / Immediate Past President*)
- Vice Speaker (*Succeeds to Speaker of the Council*)

EVEN-NUMBERED YEAR ELECTIONS

- Resident Representative to the ACEP Board of Directors
- Director of Leadership Development
- Director of Health Policy

SPECIAL ELECTION

- Member-at-Large

EMRA Events @



#EMRALeaders emra.org/acep

Please check www.emra.org/acep for room assignment & updates.



REPRESENTATIVE COUNCIL & FALL BUSINESS MEETING

Sunday, Oct. 2 • 8 am - 1 pm
Voter Credentialing @ 7 am

Actively engage in the important issues affecting your EM residency training. New resolutions will be presented to address policies affecting your current and future professional development, practice, and specialty.

EMRA Events @



#EMRALeaders emra.org/acep

Please check www.emra.org/acep for room assignment & updates.



EMRA Job & Fellowship Fair

Don't miss out on the largest recruiting event in emergency medicine!

Saturday, Oct. 1 • 5-7 pm Live @ ACEP22
Moscone Convention Center - Hall D

The EMRA Job & Fellowship Fair returns in-person at ACEP22. It's the premier event to help you find your next career steps. Connect with employers and fellowship programs from across the nation.

Supported by



*Are you a fellowship director or employer? Join us! Please contact:
Heather Deja • 469.499.0167 • hdeja@emra.org*

Please check www.emra.org/exhibitors/job-fellowship-fair for more information.



Emergency
Medicine
Residents'
Association

Case-con

Medical Student and Resident Case Study Contest

Saturday, Oct. 1 • 8 am - 5 pm

Students and residents will present interesting and notable **emergency medicine cases** in this modern take on poster sessions. Three winners will be named in each category.

EMRA Events @  22  #EMRACaseCon emra.org/acep

Please check www.emra.org/acep for room assignment & updates.



Emergency
Medicine
Residents'
Association

EMRA Resident SIMWARS

Sunday, Oct. 2 • 9 am - 3 pm

Attend this highly engaging, head-to-head skills competition, featuring teams from:

- AMITA Health Resurrection
- Good Samaritan Hospital
- New York Presbyterian-Queens
- Staten Island University Hospital
- University of Kansas
- University of Puerto Rico
- UT Health San Antonio
- Yale New Haven Hospital

EMRA Events @  22  #EMRASIMWars emra.org/acep

Please check www.emra.org/acep for room assignment & updates.

EMRA would like to extend a **BIG**, heartfelt **CONGRATULATIONS** to our **SUMMER AWARD WINNERS!**

Joseph F. Waeckerle, MD, FACEP,
Alumni of the Year Award

Eric Steinberg, DO

St. Joseph's Regional Medical Center

Faculty Mentor of the Year Award

Elizabeth Leenellett, MD

University of Cincinnati Dept. of Emergency Medicine

Faculty Teaching Excellence Award

Ran Ran, MD

Oregon Health and Science University

Steve Tantama, MD, Military
Excellence Award

Lt. Col. Jaysun Frisch, DO

The Ohio State University

Clinical Excellence Award

Renato Rapada, DO

Brooke Army Medical Center/SAUSHEC

Augustine D'Orta Humanism Award

Vivian Tam, MD

The Ottawa Hospital

FOAM(er) of the Year

Kristen Panthagani, MD, PhD

Yale New Haven Medical Center

CORD Academic Assembly
Travel Scholarship

Brittany Ladson, DO

CMU EM

LAC Travel Scholarship

Jose Reyes, MD

Cook County Health & Hospitals System

EMRA/ACEP Resident-Fellow Health Policy
Elective in D.C.

Ashley Brittain, MD

St. Barnabas Hospital

EMRA/ACEP Medical Student
Elective in Health Policy

Adriana Cordova, MS

University of IL at Chicago

Leyla Farshidpour

University of CA, Davis School of Medicine

EMRA/ACEP EDDA Travel Scholarship

Natassia Buckridge, MD

State University of New York

Danielle Haussner, MD

NY Presbyterian Hospital Columbia and Cornell

Kourosch Yazdani, MD

MetHarlem

Samuel Sondheim, MD, MBA

Mount Sinai

Mark Brombacher, DO

Spectrum Health/Michigan State University

EMRA/ACEP EMBRS Scholarship

Kristopher Hendershot, MD

Jackson Memorial Hospital/University of Miami

EMRA Simulation Research Grant

David Fernandez, MD

Northshore-Long Island Hospital, Northwell

Sophia Görgens, MD

Zucker-Northwell NS/LIJ



EMRA 2016 RESIDENT LECTURE COMPETITION

Supported by
HIPPO
EDUCATION

Monday, Oct. 3 • 1 pm

High-yield, quick presentations by up-and-coming med-ed stars!

EMRA Events @  22  #EMRA20in6 emra.org/acep

Please check www.emra.org/acep for room assignment & updates.



EMRA Airway Stories

Monday, Oct. 3 • 5-7 pm

Share the joys, the challenges, and the pain of life on the front lines of health care in this private, safe space with your peers.
You're not in this alone.

EMRA Events @  22  emra.org/acep

Please check www.emra.org/acep for specific location & updates.



Books donated by



ELSEVIER



Tuesday, Oct. 4 • 7 am - 3 pm

Supported by



BTG

EMRA's MedWAR (Medical Wilderness Adventure Race) is a unique event designed to teach and test wilderness survival and medical skills.

EMRA Events @ 22

[#EMRAMedWAR](#) emra.org/acep



EMRA Party!

Sunday, Oct. 2 • 10 pm - 2 am

#EMRAFamily, it has been too long! We can't wait to celebrate with you. Please join us for a reimagined, reinvigorated EMRA Party.

EMRA Events @ 22

emra.org/acep

Please check www.emra.org/acep for specific location & updates.

Thank You



for supporting EMRA @ ACEP22



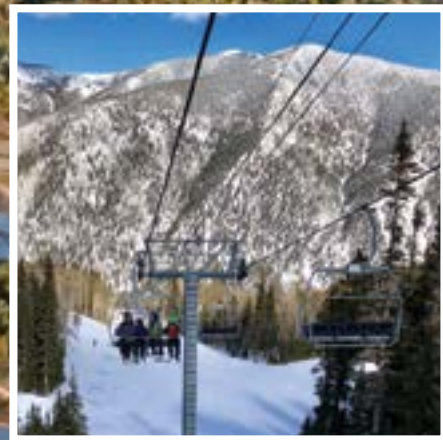
ADVANCING EMERGENCY CARE 



(Current as of 7/15/22)

SETTLE INTO THE LAND OF ENCHANTMENT WITH YOUR OWN BUSINESS

Step into One, Two or Three
Urgent Care Clinics in Northern New Mexico



- Turn-key operations
- Excellent opportunity to enter the fastest growing healthcare sector as your own boss
- Room for growth by expanding services or adding specialties

Financials and specifics shared with interested parties.

Contact William Kotsch at 575.770.1873



Scientific Assembly
SAN FRANCISCO **22**

OCTOBER 1-4

Connect

Jump start your career at the world's largest emergency medicine conference.

**Get
Connected
at ACEP22**

- Attend the Job Fair
- Network with EM's Movers and Shakers
- Position Yourself for Future Success
- Learn the Business of EM
- Discover What They Don't Teach You in Residency

Register Today!

acep.org/acep22

Approved for AMA PRA Category 1 Credit™



ECG Challenge

Masood R. Mohammed, DO
 Baystate Medical Center

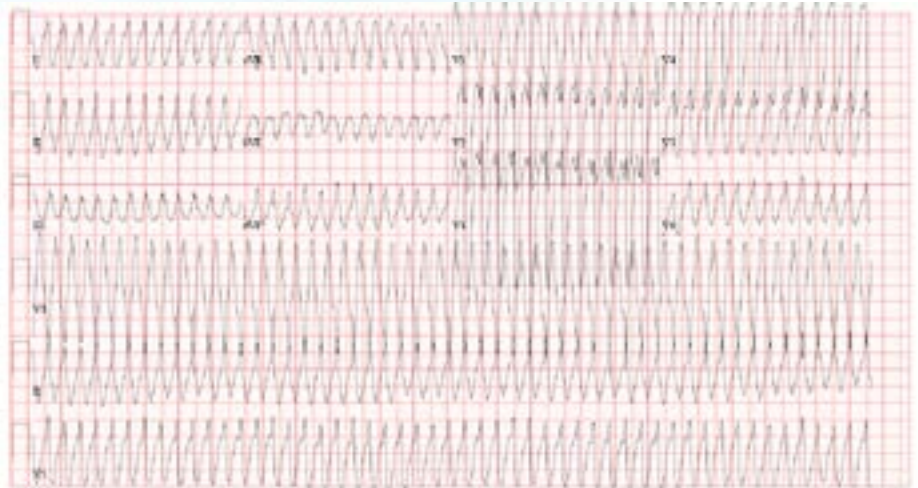
Jeremy Berberian, MD
 Associate Director of Emergency Medicine Resident Education
 Dept. of Emergency Medicine, Christiana Care Health System
 @jgberberian

CASE

A 27-year-old male with a PMH of seizure disorder and developmental delay presents via EMS after a witnessed seizure. The patient was able to intermittently go into sinus rhythm with vagal maneuvers.

What is your interpretation of his ECG?

See the ANSWER on page 52.



CORD

PURCHASE TODAY

The premier conference for EM educators is now available VIRTUALLY!

CORD's Virtual Academic Assembly 2022 is available on-demand for three (3) years and provides 62.5 *AMA PRA Category 1 Credit(s)*[™].

Visit www.cordem.org/virtual2022/ to learn more.

The American College of Emergency Physicians is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

The American College of Emergency Physicians designates this Enduring material for a maximum of 62.5 *AMA PRA Category 1 Credits*[™]. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Approved by the American College of Emergency Physicians for a maximum of 62.5 hours of ACEP Category I credit.



ECG Challenge

ANSWER

This ECG shows a regular wide complex tachycardia with a ventricular rate of 315 bpm, no discernable P-waves, northwest/extreme axis deviation, and a prolonged QRS complex duration with an intraventricular conduction delay.

The differential diagnosis for a regular wide complex tachycardia includes:

- Monomorphic ventricular tachycardia
- Antidromic AVRT
- Any regular SVT (eg, sinus tach, AVNRT, atrial flutter, etc.) with aberrant conduction (eg, fixed or rate-related BBB, metabolic abnormalities, sodium channel blocker toxicity, etc.)

The patient's lack of risk factors (ie, young age, no cardiac history) and ability to break the rhythm with vagal maneuvers suggest a non-VT diagnosis (eg, AVNRT with aberrant conduction), but notable ECG findings that suggest VT include negative QRS concordance in leads V1-V6 (ie, all of the QRS complexes point down) and Brugada's sign (ie, time from the onset of the QRS complex to the nadir of the S-wave is > 100 msec) best seen in leads V5-V6. These seemingly contradictory findings are often seen with idiopathic ventricular tachycardias.

Idiopathic ventricular tachycardias represent ~10% of all VTs and are generally seen in young, healthy patients in the absence of structural heart disease. Episodes of VT typically occur spontaneously but can be precipitated by infection, physical activity, or adrenergic stimulation. The majority of idiopathic VTs originate in the right ventricle, but 10-15% originate in the left ventricle.

Idiopathic fascicular ventricular tachycardia (IFVT), also known as verapamil-sensitive VT or Belhassen VT, is a re-entry tachycardia from an ectopic focus in the left ventricle. The ECG will show a RBBB-like pattern with a relatively mild prolongation of the QRS complex duration (120-140 msec). IFVT can easily be confused with SVTs with aberrant conduction. ECG findings that can help distinguish IFVT from SVT include capture beats, fusion beats, or AV dissociation. As the name verapamil-sensitive VT suggests, this rhythm is very responsive to verapamil, typically dosed 2.5-5 mg IV every 15-30 minutes. Common treatments for SVT (eg, adenosine and vagal maneuvers) are sometimes effective, which can lead to anchoring on a diagnosis of SVT.

The key learning point in this case is that responsiveness of a WCT to vagal maneuvers or nodal blocking medications is not diagnostic of SVT. From a therapeutic perspective, treating a WCT with adenosine is unlikely to cause the patient serious

harm even if the underlying rhythm is VT. From a diagnostic perspective, it would be harmful to incorrectly diagnose the patient with SVT based on the response to adenosine when it was really VT.

Case Conclusion

This patient was effectively treated with IV procainamide and admitted to the cardiology service. His inpatient workup was notable for positive Lyme titers. He was treated for suspected Lyme carditis and discharged home on sotalol.

Monomorphic VT Learning Points

- ≥ 3 consecutive, regular, wide complex beats with rate > 120-130 bpm
 - Non-sustained: < 30 sec duration with no hemodynamic instability
 - Sustained: ≥ 30 sec duration OR causes hemodynamic instability
- Rates < 120-130 bpm can be seen in patients on chronic antidysrhythmic medications (eg, amiodarone, flecainide, sotalol) or with severe cardiomyopathies
- If rate < 120 bpm, consider mimics:
 - Hyperkalemia
 - Sodium channel blocker toxicity
 - Accelerated idioventricular rhythm (AIVR)

ECG Features in a Regular WCT that Suggest VT

- QRS complex duration > 200 msec is almost always VT, hyperkalemia, or sodium channel blocker toxicity
- AV dissociation (ventricular rate > atrial rate)
- Positive or negative QRS complex concordance in leads V1-V6
- Extreme axis deviation ("northwest axis")
- The absence of typical RBBB or LBBB pattern (ie, normal RBBB or LBBB pattern makes SVT with aberrant conduction more likely)
- Fusion beat: hybrid QRS complex formed by both supraventricular and ventricular focus
- Capture beat: sinus QRS complex formed by transient normal conduction amid AV dissociation
- Brugada's sign: time from the onset of the QRS complex to the nadir of the S-wave is > 100 msec
- Josephson's sign: notching on the downslope of the S-wave near its nadir ★

Board Review Questions

PEERprep for Physicians is ACEP's gold standard in self-assessment and educational review.

For complete answers and explanations, visit the Board Review Questions page at emresident.org, under "Test Your Knowledge"

Order PEERprep at acep.org/peerprep

PEERprep
on sale
NOW!

- 1. A 9-year-old boy presents with progressive facial swelling. His features are distorted from swollen eyelids and generalized edema. Examination of his abdomen is consistent with mild ascites. He has not had any recent infections. Urinalysis reveals 4+ protein and no hematuria. What is the physiologic cause of this patient's edema?**
 - A. Decreased excretion of free water due to decreased glomerular filtration in the kidney
 - B. Decreased production of albumin and other proteins in the liver
 - C. Release of nitric oxide, histamine, and other inflammatory mediators from mast cells
 - D. Urinary excretion of protein leading to decreased oncotic pressure in the plasma
- 2. A 52-year-old man presents with severe chest pain radiating to his neck. The pain started acutely 2 hours ago, has been unrelenting, and is worse with deep breathing and swallowing. According to his wife, he "smoked a joint before that and then threw up so hard he vomited blood." The patient confirms this, and his voice is notably hoarse. On examination, he appears distressed due to pain, and his skin is sweaty. His vital signs are BP 124/89, P 112, R 24, and T 37.4°C (99.3°F); SpO2 is 98% on room air. What is the most likely cause of this patient's symptoms?**
 - A. Acute myocardial infarction
 - B. Boerhaave syndrome
 - C. Peptic ulcer disease
 - D. Spontaneous pneumothorax
- 3. What is the treatment of choice for Rocky Mountain spotted fever in pediatric patients?**
 - A. Chloramphenicol
 - B. Doxycycline
 - C. Supportive care
 - D. Trimethoprim-sulfamethoxazole
- 4. An 80-year-old woman presents with pressure in her pelvis that is worsened by standing up or bearing down. She has not had any surgeries and has four grown children. She is otherwise asymptomatic; she denies dysuria, urinary dribbling, vaginal bleeding, and abdominal pain. A descending soft circular mass is discovered within the vagina on pelvic examination. What is the most likely diagnosis?**
 - A. Cystocele
 - B. Prolapsed fibroid
 - C. Rectocele
 - D. Uterine prolapse
- 5. Which method is most reliable for confirming the correct tube placement after endotracheal intubation?**
 - A. 5-point auscultation
 - B. Chest X-ray
 - C. End-tidal capnography
 - D. Endotracheal tube condensation *

Emergency

Fulfilling the promise of medicine



The Permanente Medical Group, Inc. (TPMG) is one of the largest medical groups in the nation with over 9,000 physicians, 22 medical centers, numerous clinics throughout Northern and Central California, and an over 75-year tradition of providing quality medical care. We currently have the following opportunities:

EMERGENCY MEDICINE PHYSICIANS

Openings in Northern & Central California

EMERGENCY MEDICINE RESIDENCY PROGRAM FACULTY

Modesto/Manteca, California

In 2021, Kaiser Permanente Modesto launched our first ever EM residency program in Northern California. We are currently seeking Academic Emergency Physicians who are (ideally) fellowship trained (ultrasound, sports medicine, etc.).

Stop by and see us at Booth #1401 at the ACEP Scientific Assembly or T135 at the EMRA Job and Fellowship Fair.

For more information about these opportunities and others throughout the region, please visit our website at tpmg.permanente.org.

For further details, please contact: Roy Hernandez at (510) 625-2731 or Roy.B.Hernandez@kp.org.

PERMANENTE MEDICINE®

The Permanente Medical Group

A FEW REASONS TO CONSIDER A PRACTICE WITH TPMG:

- **Work-life balance** focused practice, including flexible schedules and unmatched practice support
- We can focus on providing excellent patient care **without managing overhead and billing**.
- We demonstrate our commitment to a culture of **equity, inclusion, and diversity** by hiring physicians that reflect and celebrate the diversity of people and cultures.
- Multi-specialty **collaboration** with a mission-driven integrated health care delivery model.

EXTRAORDINARY BENEFITS:

- Competitive compensation and benefits package, including comprehensive medical and dental
- Moving allowance and home loan assistance - up to \$200,000 (approval required)
- Malpractice and tail insurance
- Paid holidays, sick leave, education leave
- Shareholder track
- Three retirement plans, including pension

Academic Emergency Medicine positions on the beautiful Gulf Coast



The University of South Alabama (Mobile, AL) has an EM residency and expanding EM academic programs at our two hospitals (Level 1 USA Health University Hospital, Children's & Women's Hospital), and a new Freestanding ED.

- Must be EM or PEDS ED trained and board eligible/certified
- Fellowship or equivalent experience in US, PEM, EMS, simulation, education/admin or research is a plus
- Opportunities to lead, initiate or contribute to new programs and services

Applicants are invited to submit CV and letter of interest to:
Edward A. Panacek, MD, MPH,
Chair of Emergency Medicine, USA College of Medicine,
at eapanacek@health.southalabama.edu



Further information:
<https://careers-usahealth.icims.com/jobs/4365/emergency-medicine-freestanding-ed--other-sites---faculty/job>



The GW Medical
Faculty Associates

Department of
Emergency Medicine

WASHINGTON DC – The Department of Emergency Medicine at the George Washington University is offering **Fellowship** positions beginning July 2023:

Health Policy & Advocacy	Telemedicine & Digital Health
International Emergency Medicine & Global Public Health	Wilderness & Telemedicine Combined Fellowship
Medical Leadership and ED Operations	Wilderness Medicine
Ultrasound for Emergency Medicine	Clinical Research
Ultrasound for Family Medicine	Medical Education
Health Equity and Social Emergency Medicine	Medical Simulation
Disaster & Operational Medicine	Medical Toxicology

Fellows receive an academic appointment at The George Washington University School of Medicine & Health Sciences and work clinically at a site staffed by the Department. The Department offers Fellows an integrated, interdisciplinary curriculum, focusing on research methodologies and grant writing. Tuition support for an MPH or equivalent degree may be provided, as per the fellowship's curriculum.

Complete descriptions of all programs, application instructions, and Fellowship Director contacts can be found at:

<https://smhs.gwu.edu/emed/education-training/fellowships>



Talking about the
issues that matter.

Listen today!
emracast.org

Supported by





Conveniently located in beautiful Central Illinois!



Emergency Physician Staffing Solutions

EPSS (Emergency Physician Staffing Solutions) is a privately physician owned, Emergency Medicine staffing company committed to providing quality health care in the communities we serve in Central and Northern Illinois. Our mission and priority is to provide a stable, professional and engaging work environment for our physicians.

We are currently seeking physicians who would like to join our team for full or part-time commitment. Opportunities are available within all our sites for committed physicians, in rural and urban health care settings, with ED volume site range from 40,000 to combined EM/hospitalist services in critical access hospitals with annual volume of 2500-7500 patients per year.

Choose a rewarding career with EPSS in Emergency Medicine!

Physician Benefits

- Independent contractor status
- Competitive compensation; malpractice coverage provided
- Team work approach; multiple physician coverage with APP and scribe at some sites
- Excellent RVU bonus compensation for committed providers
- Flexible provider scheduling; block scheduling available for travel

Looking for a change? GREAT central Illinois locations; family friendly communities located directly on the Illinois River with wonderful tourism while easily accessed from Chicago and St. Louis

EMERGENCY MEDICINE POSITION SPOTLIGHT:

UnityPoint Proctor-Peoria; UnityPoint - Pekin, Illinois

EPSS is currently seeking EM physicians for FT/PT coverage for combined site staffing.

- Independent Contractor Status: Excellent compensation direct to physician
- UnityPoint Proctor 19K volume, UnityPoint Pekin 23K ED volume
- Physician single coverage; 3 shifts per day with APP and scribe coverage
- Excellent support service and staff; EMR: EPIC
- ABEM board certification/eligible or IM/FP board certified with ABPS certification EM experience

OSF St. Luke Hospital, Kewanee, OSF St. Clare Medical Center, Princeton, Illinois

EPSS is currently seeking EM physician coverage for 11K and 7.2K volume critical access ED

- Independent Contractor Status: Excellent compensation direct to physician
- Physician single coverage; flexible 12hr or 24hr shift coverage; EMR: EPIC
- Location easily accessed from Rockford, Chicago and Springfield for block travel
- Excellent support services, hospitalist and surgery, 24hr radiology coverage
- ABEM board certification/eligible or IM/FP board certified with ABPS certification EM experience

Visit www.epsserdoc.com for contact details and to apply

EMERGENCY MEDICINE RESIDENTS - PLAN YOUR TRANSITION NOW!



EMERGENCY MEDICINE RESIDENTS' ASSOCIATION DISABILITY INSURANCE PROGRAM

An individual, long-term disability insurance program designed for EMRA students, residents and alumni.

Important highlights of the program:

- Up to \$7,500 monthly tax-free benefit with no financial disclosure
- Specialty Specific definition of disability – 6 options in most states
- Personalized comparison of plans, taking advantage of regional, GME or association discounts as available to you
- Easy online application process

Obtain your disability protection today!

Visit the EMRA webpage: www.iwcglobal.net/p/emra

Connect with us: @IWCAAdvisor Integrated Wealthcare

We'll be at ACEP22!

OCT 1-4
San Francisco





EMERGENCY MEDICINE SPECIALISTS, INC.

Independent, Democratic
Emergency Medicine
Physician Group

Providing patients
quality emergency
care for over 40 years

Serving a variety of
practice settings in the
Dayton, Ohio region



UNCOVER YOUR FUTURE

- Competitive salary & sign-on bonus
- Maximum employer funded 401k & profit-sharing contributions
- Leadership & ownership opportunities
- Best in class mentorship & professional development programs

DISCOVER DAYTON, OHIO

Find your home with Emergency Medicine Specialists. Join an accessible group of educators and mentors who foster a culture of patient focus and employee wellness.

CONNECT WITH US

Ohio ACEP - Booth 7
August 24, 2022
Columbus, OH

ACEP22 - Booth 1920
October 1-4, 2022
San Francisco, CA





Emergency Medicine Residents' Association
 4950 W. Royal Lane
 Irving, TX 75063
 972.550.0920
emra.org

PRSRT STD
 U.S. POSTAGE PAID
 Madison, WI
 Permit No. 1457

MobilEM

A powerful resource in an easy-to-use app.

Get it @ emra.org/apps



Featuring new:

- Antibiotic Guide
- EKG Guide
- Toxicology Guide
- Patient Convo Kit

- Free content + subscriptions
- All-Access Pass
- Universal search
- Bookmarking & Notes
- Updates in real time
- Available in iTunes & Google Play

Order our newest print editions at www.emra.org/amazon

