EM Resident
Official Publication of the Emergency Medicine Residents' Association
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An End-of-Residency Checklist for You

EMRA's Annual Report

Pericardiocentesis for Tamponade

Dangerous Bias in Cardiac Arrest Research

BOUNDLESS

UCLA'S INAUGURAL EM SPACE MEDICINE FELLOW

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.

JOIN OUR TEAM
EMERGENCY MEDICINE
OPPORTUNITIES AVAILABLE

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:

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Innovating for the Future

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

"The difference between science and the arts is not that they are different sides of the same coin, or even different parts of the same continuum, but rather, they're manifestations of the same thing... They spring from the same source. The arts and sciences are avatars of human creativity. It's our attempt as humans to build an understanding of the universe, the world around us. It's our attempt to influence things, the universe internal to ourselves and external to us."

— Mae Jemison, MD, physician and first woman of color in space

Photo courtesy of NASA

Mae Jemison, MD

Physician, astronaut, professor, and dancer Dr. Mae Jemison shared the quote above in her 2002 TED talk "Teach Arts and Sciences Together." After two decades, the urgency in her message still resonates. Science, like art, stagnates without creativity.

As emergency physicians, we must embrace creativity and boundless curiosity in order to move our field into the future. I've had the opportunity to see that spark of creativity burning brightly across this issue of EM Resident. Creativity shines in Dr. Haig Aintablian's passion for the rapidly evolving field of space medicine, and his boldness in piloting UCLA's fellowship. It shines in the op-ed calling for an end to possessive eponyms and the impact this has on our patients with eponymous diseases. It's in a thought-provoking analysis of cardiac arrest research biases, biases that limit evidence-based treatment of Black patients, rural patients, and women. In so many of these articles, your creativity is not just in the prose of your research or the sculptures in your sim models. It's in your willingness to boldly challenge the status quo.

Emergency medicine is a highly nimble specialty, and advancements have come very quickly over the past 50 years. But this is not a fact we can take for granted indefinitely. Being good stewards of EM's creative momentum takes energy. We must actively question our own dogma, which may mean repeating old studies to evaluate their reproducibility and generalizability. We must promote diversity in our EDs and select physician leaders who foster creativity. We must explore new technologies and strategies for serving patients, from rural medicine to space medicine and beyond.

As Dr. Mae Jemison said, "Never be limited by other people's limited imaginations."

Let's keep in touch!
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References available online.

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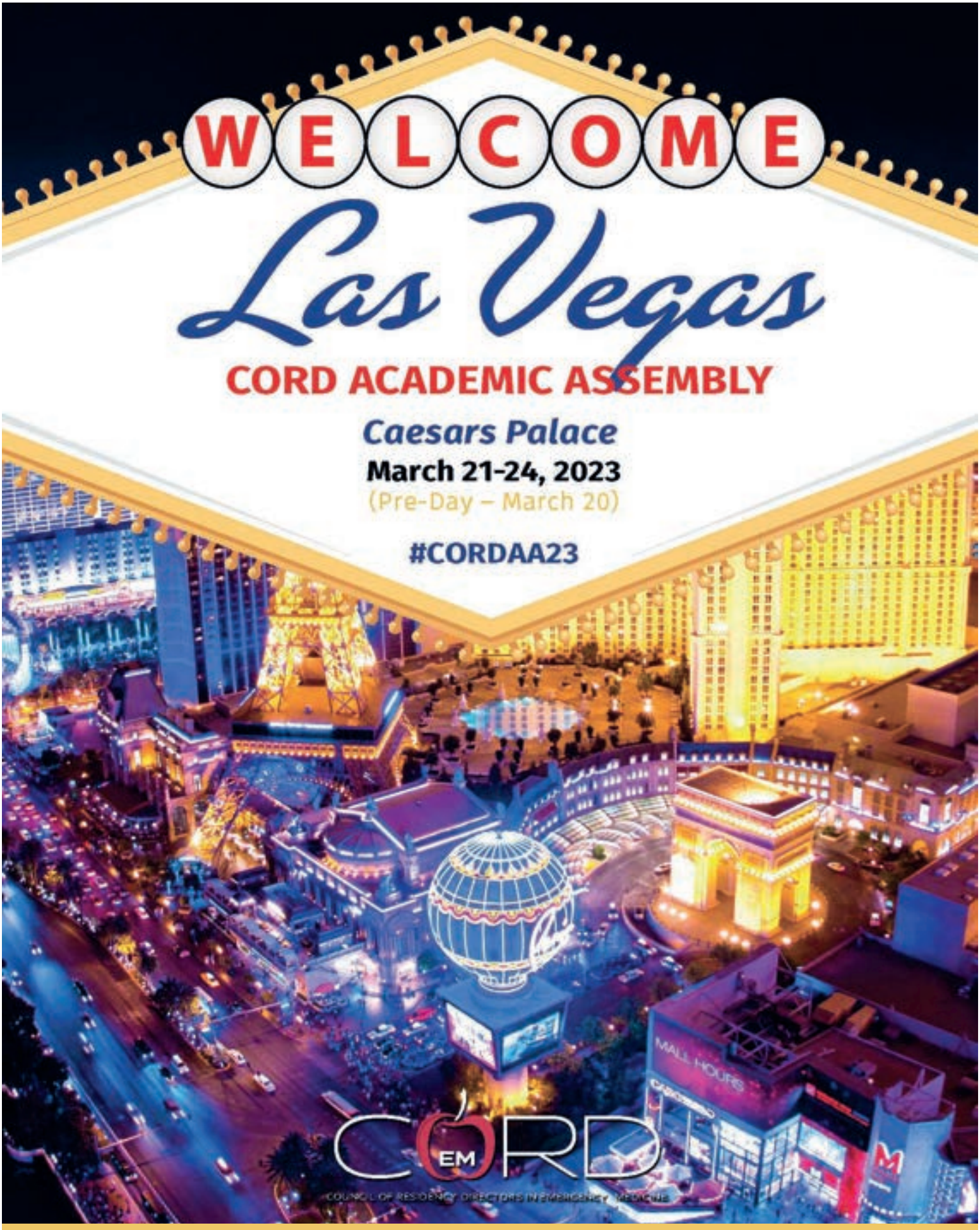
EM Resident (ISSN 2377-438X) is the bi-monthly magazine of the Emergency Medicine Residents' Association (EMRA). The opinions herein are those of the authors and not of EMRA or any institutions, organizations, or federal agencies. EMRA encourages readers to inform themselves fully about all issues presented.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



Approved for AMA PRA Category 1 Credit(s)™

EMRA Report
Fiscal Year 2022

Membership **19,191**

President
Angela Cai, MD, MBA

Emergency Medicine Residents' Association Annual Report 2022

EMRA is honored to present our FY22 Annual Report celebrating our mission to serve as the voice of EM physicians-in-training.

We are thrilled to welcome our new Executive Director **Kris Williams, CAE**, who joined EMRA in March.

We recently concluded our triennial strategic planning process, which refocused our organization in the wake of exponential growth in membership, programming, and engagement over the past several years.

It has been a tremendous privilege to serve as your president for the past year and on your board of directors for the past four years.

EMRA is here for you all the way.

>95%

of EM residents are EMRA members

EMRA helps you become the *BEST DOCTOR* you can be.

EMRA's resources provide meaningful and tangible ways to serve and connect with our 19,000 members. Every member receives a free member kit filled with our most popular printed guides tailored to training level. Our publications also allow us to fund our programs and sustain our financial independence by diversifying our revenue.

I use our bedside guides on every single shift with the MobilEM app (https://www.emra.org/about-emra/publications/mobile-applications/), which gives instant, searchable access to the Antibiotic Guide, PressorDex, and more. EMRA continuously updates these resources in print and in-app to ensure our members stay up to date.

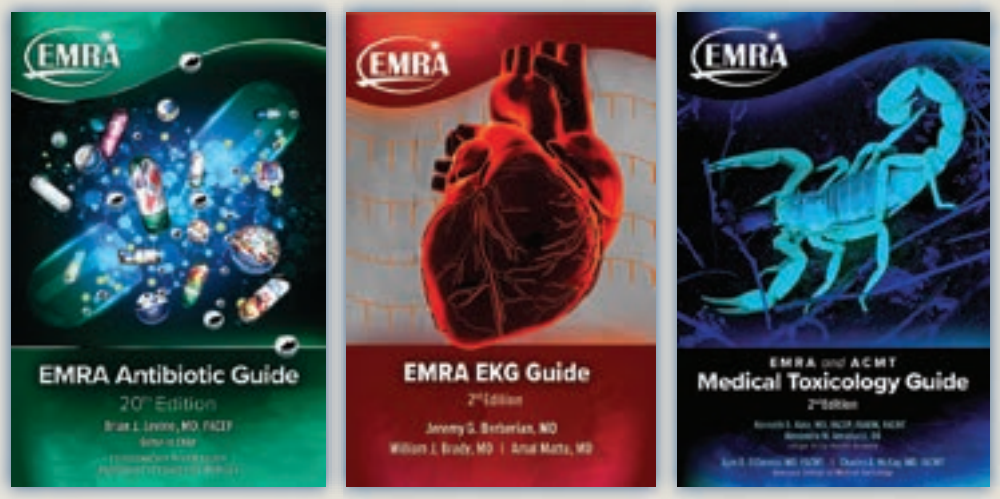
EMRA also has responded to member needs for non-clinical education, which is in high demand yet often identified as a gap in residency training. For the business side of EM, EMRA and ACEP launched Practice Essentials of Emergency Medicine, an online, asynchronous curriculum starting with the first module Billing & Coding. On the EM career-planning front, EMRA and ACEP have launched a newly designed, user-friendly emCareers site (https://www.emcareers.org) with live, searchable job postings. Furthermore, EMRA is updating and augmenting career-planning resources and information on our own website via a new initiative dubbed "EMployED."

PRACTICE essentials OF EMERGENCY MEDICINE

$1.4M 43% revenue

8 new or newly revised EMRA guides, reference cards, videos, & apps

36 on-shift clinical resources free for EMRA members



EMRA CAST

1,000+ downloads per month

24 new episodes per year

EMRA HANGOUTS

8

student-focused national Hangouts sessions

EM Resident

Official Publication of the Emergency Medicine Residents' Association

80,000 average monthly online views

18,000 print distribution

EMRA Match

>500,000 searches of EMRA Match for:

- *Residents*
- *Clerkships*
- *Fellowships*
- *Jobs*

239

programs with 100% membership

EMRA helps you become the *BEST LEADER* you can be.

EMRA aims to cultivate lifelong leaders who will shape the future of EM. We do this by providing opportunities, mentorship, and networking with our specialty's current leaders.

EMRA committees provide leadership opportunities for 120 trainees across 18 committees, including new subcommittees for Rural EM and Climate Change. We have improved our application questions and data collection to prioritize diversity of experiences and alignment with EMRA's mission.

EMRA appointed more than 120 liaisons to ACEP committees, sections, and taskforces. Taskforces include critical topics such as EM group ownership, ED accreditation, and new practice models. The annual EMRA/ACEP Leadership Academy had 69 graduates.

Our policy-focused leadership positions include Health Policy Academy Fellows and a new program for funded Regional Representative to Representative Council. With increasing member interest in issues adjudicated at the state level, we announced an objective to work with all ACEP chapters to build a pipeline of leaders across the country.

20
committees

225+
meetings, webinars, publications

4,986
members of EMRA's 20 committees

- *Medical students*
- *Residents*
- *Fellows*

104
funded national leadership opportunities for members

114
Leadership Academy fellows

69 graduates and **45** currently in the program

33
categories of awards, scholarships, and grants

- *Scholarships*
- *Leaderships*
- *Professional excellence*

>$2.8M annually invested into members

EMRA helps EM become the *BEST SPECIALTY* we can be.

With record Representative Council engagement this past year, EMRA passed resolutions on the following topics:

- Workplace violence and occupational protections for trainees
- Family, medical, and bereavement leave
- Restrictive covenants and non-competes
- Corporate practice of medicine
- State medical licensure questions regarding mental health
- Nonphysician provider supervision and telemedicine
- SLOE transparency
- Criminal justice reform and equitable care of incarcerated patients

EMRA released a workforce statement (https://www.emra.org/be-involved/be-an-advocate/working-for-you/2022-workforce-study-statement-cai/) to advance our members' voice and provide education on the following topics: residency program growth and training standards; nonphysician providers; rural EM; expanding the scope of EM; and the corporate practice of medicine.

We are committed to recruiting the best and the brightest to our specialty by acknowledging uncertainty about the workforce while promoting positive messaging (https://www.emra.org/emresident/article/presidents-message-august-september-2022/) about the specialty. Our dedicated Medical Student Council has reached out to 220+ EMIGs. We engaged more than 300 medical students during our medical student forums and residency fairs. The EMRA Diversity and Inclusion Committee has been mentoring and teaching HBCU medical students since 2019 to strengthen the pipeline to EM.

Additional statements (https://www.emra.org/be-involved/be-an-advocate/working-for-you/) included:

- Abortion as health care
- Reaffirming our commitment to firearm safety
- Consensus recommendations on away rotation
- Joint statement on 2022 residency match

The annual EMRA/ACEP YPS Health Policy Primer in Washington, D.C., delivered timely education on the history of contract management groups and case studies of identifying problems and creating solutions at multiple levels of government — from local to federal, regulatory to legislative.

EMRA represented the trainee voice at:

- ABEM Model of the Clinical Practice of EM Task Force update to residency content specifications for ABEM exams
- Taskforce on Recommendation to the ACGME Review Committee on residency program standards
- ACGME Review Committee Scenario Planning workshop
- CORD preference signaling working group
- Medical Summit on Firearm Injury Prevention

EMRA is proud of its accomplishments working on your behalf. We look forward to designing our best possible futures together as we continue to implement our mission and strengthen our specialty.

Workforce

EMRA continues to take part in task force actions to protect and shape the future of the specialty

11 Resolutions proposed and debated by EMRA members and the Representative Council

- *Workplace safety*
- *Family and medical leave*
- *Mental health questions for state licensure*
- *Corporate practice of medicine*

12

Partnerships to advance our mission (ABEM, ACEP, ACGME, AEROS, AFFIRM, AMA, CORD, ECQMC, EDPMA, EMF, EMPI, NEMPAC)

EMRA Health Policy Academy

A Call for Applications

The EMRA Health Policy Academy is a highly selective yearlong experience that will train you to be a policy advocate in emergency medicine. It's designed to give passionate residents like you the opportunity to impact policies that affect EM and your patients at a local, state, and national level. This gives you the knowledge and tools to set you up for a lifetime of advocacy.

Application Deadline — Dec. 1, 2022

More Information

Apply Here

EMRA Emergency Medicine Residents' Association

EMRA/ACEP Leadership Academy

A Call for Applications

The EMRA/ACEP Leadership Academy is a professional development program and virtual community for emerging leaders in emergency medicine. This one-year progressive experience not only provides instruction in key leadership tenets, but also helps you meet peers and mentors who are building the specialty alongside you.

Supported by Vapotherm

Application Deadline — Dec. 1, 2022

More Information

Apply Here

EMRA Emergency Medicine Residents' Association

2023 WINTER AWARDS

A Call for Nominations

If you know someone who deserves recognition or scholarships, nominate them for our Winter Awards. Submit your entries now!

CATEGORIES

Chair of the Year

Residency Director of the Year

Associate Residency Director of the Year

Sherrill Mullenix Residency Coordinator of the Year

Academic Excellence Award

ACEP Scientific Review Subommittee Appointment

Dr. Alexandra Greene Medical Student of the Year Award

Chief Resident(s) of the Year

Fellow of the Year

Resident of the Year

Rosh Review “One Step Further” Award

Jean Hollister Contribution to Pre-Hospital Care Award

ACEP/CORD Teaching Fellowship Scholarship

Travel Scholarships (ACEP Scientific Assembly, EDPMA, and SAEM)

More Information

Nomination deadline — Dec. 4

To Nominate

EMRA Emergency Medicine Residents’ Association

The Final Frontier for Emergency Medicine

An Inside Look at UCLA's Space Medicine Fellowship Program

Sriram Venkatesan, FAWM
Vice Chair, EMRA Prehospital and Disaster Medicine Committee
Sri Ramachandra Medical College & Research Institute
University of Colorado-Denver

Kyle Essex, NRP, FP-C, CCP-C, C-NPT
Asst. Vice Chair, EMRA Prehospital and Disaster Medicine Committee
Burrell College of Osteopathic Medicine

In 1961, Dr. James Mills Jr. launched the first emergency medicine practice in Alexandria, Va. That same year, the former Soviet Union successfully sent a human into space. In 1969, Neil Armstrong became the first man to set foot on the moon. Just one year later, Dr. Bruce Janiak became the first emergency medicine resident. With such similar time progression, it was only a matter of time before these paths intersected on the space-time continuum.

Fast-forward to the present: It now appears that space has met its physician, and the physician has met his program. The physician is Dr. Haig Aintablian, and the program is UCLA's Space Medicine Fellowship.

Space: The Final Frontier. These are the voyages of the UCLA Space Medicine Fellowship. It's a 2-year mission — to explore strange new wounds, seek out new life, and discover new med calculations. To boldly go where no physician has gone before.

During this time, Dr. Aintablian — inaugural fellow at UCLA's new Space Medicine Fellowship program — will trade his leatherman raptors for surgical scalpels and astronautics books, adding scope and skill that span multiple professions from surgery to engineering. He will endure a training program designed to produce physicians who will not only sit in mission control on the ground, but also in spacecrafts on missions.

EMRA's Prehospital and Disaster Medicine (PH&DM) Committee spoke with Dr. Aintablian about this fellowship opportunity, offered exclusively to emergency medicine residency-trained physicians. He gave us an eagle's eye view of his training experience so far, future direction for the field, and opportunities for medical students and residents who are interested in getting involved.

EMRA PH&DM Committee: How would you define "space medicine" for those not familiar with the term (i.e., emergency medicine residents and medical students)?

Dr. Aintablian: Space medicine is essentially the combination of skills and traits that would be needed in a physician to be able to treat someone in space — not just on the ground, like in mission control, but also to treat them as a member of the crew. So this includes understanding all of the terrestrial pathologies, but on top of that, understanding all of the pathologies that could develop in microgravity or zero gravity.

EMRA PH&DM: What was your path to space medicine? What inspired you to pursue this field?

Dr. Aintablian: I have always been fascinated with space since I was a little kid. My dad and I would look through a telescope and try to find distant stars and planets. I got more involved in the world of space medicine when I was doing my master's degree in molecular genetics and biochemistry, where part of what I was looking at was structural changes in our DNA that could happen in microgravity and what structural limitations these could have. During medical school, I ended up doing an away rotation with NASA during my fourth year, which really piqued my interest. Later on in residency, when an opportunity came up for us to build this space medicine fellowship with the private space industry, I jumped on it.

EMRA PH&DM: What is your day-to-day or week-to-week life like as a space medicine fellow?

Dr. Aintablian: I have a baseline level of shifts that I do each month in the ED, as an attending. This is a requirement of the fellowship for us to be able to keep our clinical skills sharp, as being a good terrestrial doc, in our minds, is the best way to be a good space doc. On top of these shifts, I have a robust engineering curriculum that I work on asynchronously to better understand the fundamental principles of aerospace engineering and bioastronautics — things like aircraft mechanics up to biological exposures to ionizing radiation. It's truly fascinating stuff. And then, I have clinical rotations, where I learn additional clinical and procedural skill sets, like percutaneous procedures, appendectomy, cholecystectomy, and even dental procedures. The goal of these is not to become an expert (as we all know how many years it takes to gain expertise in a field through our residency training), but to have a sort of competency in case specific scenarios were to arise in space. In addition to all this, my favorite part is just how heavily involved I get to be with the private space industry, doing specific projects that will help humanity reach places like the moon and Mars. I can't discuss specifics, but I will say that it's

pretty awesome. In addition to this, we have an austere medical rotation, Mars analogs we get to practice in, and a few other gems. Overall, it's a pretty busy fellowship with so much crammed into 2 years, but I wouldn't want it any other way.

EMRA PH&DM: You're involved in a newly launched fellowship, which means that everything — the curriculum and connections, for example — had to be developed from scratch. Describe your experience.

Dr. Aintablian: My experience has been fantastic. I think we've hit the ground running with this fellowship. It took us about a year-and-a-half to develop the curriculum, to build all the connections to make this happen. Since then, it has been going extremely well. The quality of the fellowship is top notch. It's a very busy fellowship. It's almost as busy as residency, if not equally busy. And honestly, every minute is filled with something that's very intellectually stimulating. And we're doing things with some of the best experts in these fields.

EMRA PH&DM: What makes this field unique in your mind, and what qualities do you think are necessary to be successful as a space medicine specialist?

Dr. Aintablian: I think the main thing that makes this field unique is that we're training a physician to essentially also be part of a crew. That's a pretty cool thing. It's almost like doing a hyperbarics fellowship but also training on what it takes to be a diver. You're learning and understanding not just physiology in the field (space), but also how to be a Swiss army knife, of sorts, in case you go up to "the field." It's multidisciplinary. It's way more than just medicine. It's way more than just emergency medicine. It's surgery, it's ophthalmology. It's dentistry, engineering, psychiatry, physics — there's just a whole host of things that build the field.

What we've discovered is that having an EM background is ideal because it gives you most of the skills needed to treat most pathologies. But there are certain things we're not trained in during an EM residency. Like what if an appendicitis or cholecystitis were

to pop up in space? What would you do if antibiotics didn't work and you're 3 months into your 6-month journey to Mars? During the fellowship, we're trained to do surgical procedures like appendectomies, cholecystectomies, nephrostomy tube placements, all the way down to dental procedures and US-guided minimally invasive procedures. We're also getting trained to do fundoscopic exams at a higher level, using ultrasound more than just to diagnose emergency conditions, but also to understand physiological parameters to get the minutiae for research purposes, understanding radiation dosages and physics forces, etc. It's a baseline skill set of being an EM doc, but on top of that, tossing in a bunch of additional skills to make you as much of a versatile Swiss army knife as possible — you know, the ones that are fatter with more and more tools!

EMRA PH&DM: Tell us more about the curriculum outside of clinical work. Where else do you get to train? Is there a standard set of skills that you're required to know by the end?

Dr. Aintablian: Our curriculum can be found on the UCLA EM website. We've got a bunch of engineering courses that we take with the UCLA School of Engineering, as well as a customized curriculum developed with Caltech JPL. We do a few away rotations with the private space industries and national space agencies. We do clinical shifts in the ED every month to keep up with our clinical skills. And then we have additional exposure to certain specialties (similar to clinical rotations) including ultrasound, ophthalmology, dentistry, toxicology, general surgery, interventional radiology, orthopedic surgery, sports medicine, hyperbarics, and psychiatry. The curriculum is

designed to help us get as wide a skill set as possible. And the engineering courses are designed to help us learn how to communicate effectively with engineers, as well as to be engineering-minded whenever we're helping design medical systems for the moon, Mars, and other future missions.

EMRA PH&DM: How challenging is it for physicians to adapt this engineering mindset that's needed to thrive in space medicine?

Dr. Aintablian: It's definitely not easy because we don't get much engineering training; the only real overlap with engineers is the physics that we take. And we take a life sciences version of the physics course. It has been very interesting for me; it has been an intellectual challenge. I hadn't previously been exposed to, or read, most of this stuff. But it's really interesting stuff. It's also more than just becoming proficient in basic engineering — it's being able to speak with engineers in their language, to understand what they're looking at when they're looking at a medical system, and understanding their specific limitations on what can or can't be done.

EMRA PH&DM: How important is fellowship training to the field of space medicine? Is it possible to be a space medicine physician without the additional training?

Dr. Aintablian: Potentially it's possible. But going through the fellowship pathway is a good way to really build that skill set under a controlled environment — especially with what we're trying to do, with the additional surgical training or dental and other subspecialty training, as well as all the specific engineering courses we take. You never know if you can get that sort of training outside of a fellowship. I can't imagine, for example, emergency physicians asking to scrub into the OR for these types of things when they're attendings. Or for an ER doc to start taking graduate-level engineering courses at Caltech. It's possible, but it wouldn't be easy. With regards to space-related physiology, it's possible to read and gain expertise with the knowledge base that's available, but it would be difficult to practice it in real life without formal training. That being said, I've had many mentors in the space industry who are self-taught physicians, and they are some of the legends who built the structure of this field. I think, though, as we are formalizing this training pathway, fellowship training is going to become a more desirable and necessary attribute.

EMRA PH&DM: As a space medicine physician-in-training working in all these different clinical subspecialities, how have folks in these areas viewed you? Have you received any pushback for specialty encroachment?

Dr. Aintablian: That's a good question. Surprisingly, there hasn't been as much territorialism as you'd expect, perhaps because we've built this fellowship with the understanding that we're not going to be specialists in any of these fields. And this isn't stuff we're going to practice terrestrially. I'm not going to go to the ER and start pulling teeth, or, you know, start doing appendectomies. This is more like, if we were to send one doctor up to space, who would we send? And what skill sets would be needed? When we built

this fellowship, we made it clear: When we spoke to the surgeons, we conveyed that we're not trying to be surgeons, but just want to be familiar enough with the anatomy of a procedure to be able to do this, if we had to, ideally under the guidance of a surgeon in mission control. Our goal is to be a Swiss army knife, not to have the biggest set of scissors or the biggest knife or the sharpest whatever tool, but to have every tool in some capacity, and the ability to do everything the best we can with the resources available. We've been really surprised with just how many departments and specialists reached out willing to help in whatever way they can, which was so awesome to see. There's a saying that it takes a village to make a doctor. That saying can absolutely be applied to the type of training needed here — it takes a village to train a space medicine physician.

EMRA PH&DM: Given current and future EM workforce concerns, what are the job prospects for a space medicine-trained EM physician? Where do you see yourself in the next 5-10 years?

Dr. Aintablian: I'm always going to work in the ER one way or another when I'm not involved with space medicine full time. As space becomes more and more available, we are going to be sending a lot of people up. The trajectory is that it's booming, and we're at the point now where we're at the bottom of that curve, and we're just about to see that steep rise in space tourism and expedition missions. So I think the market is going to be fantastic for space medicine. And, in the coming years, we're going to need many more space medicine-trained physicians for these crewed space expeditions.

EMRA PH&DM: In a basic exam, we often take gravity for granted in positioning for heart/lung sounds (understanding where fluid accumulates) and testing reflexes. I would imagine even EKGs look quite different with reduced stresses and lack of gravity. How differently is medicine practiced?

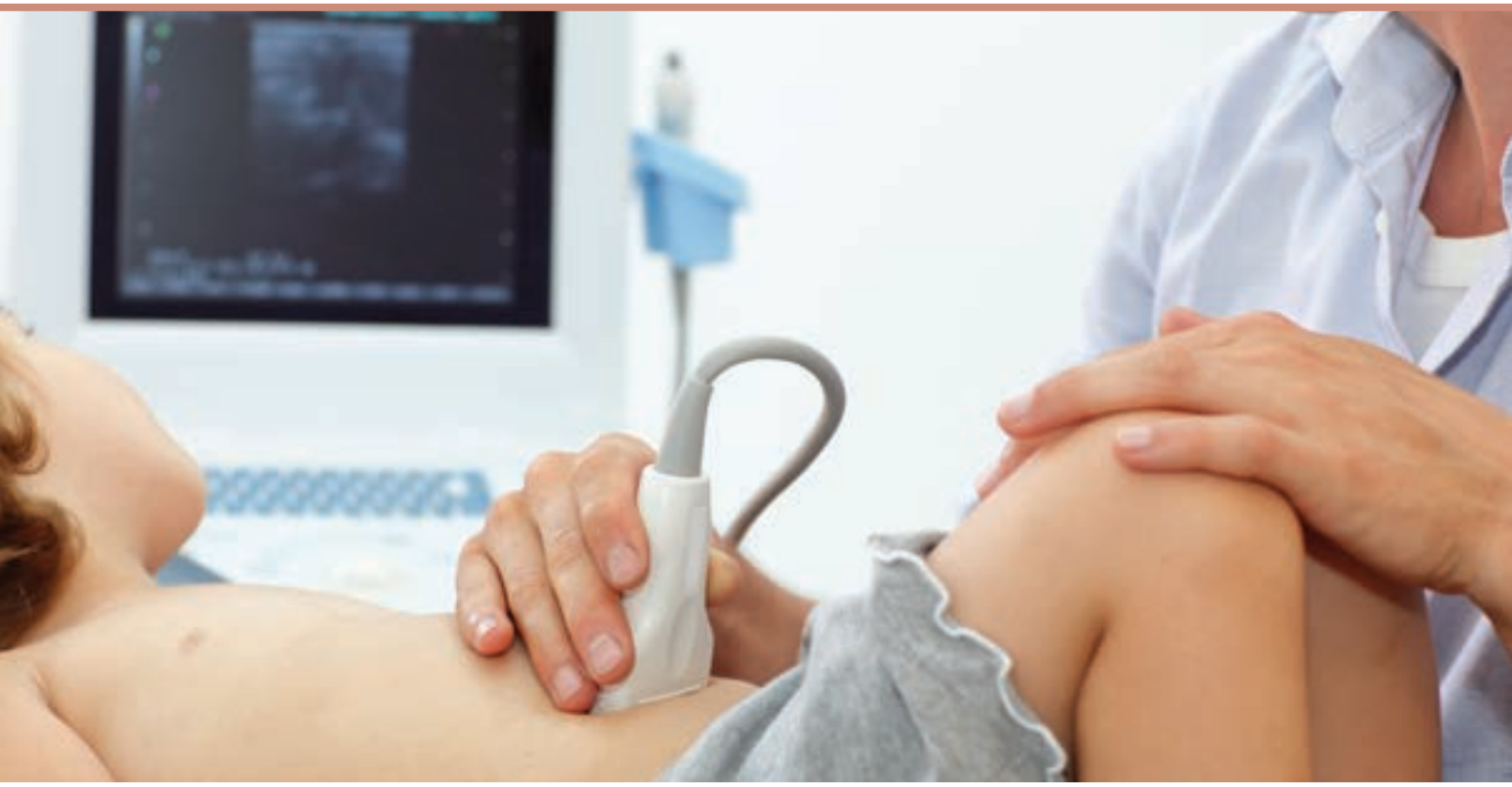
Dr. Aintablian: Yeah, totally! So I think EKG is not as big a deal. But with regards to drawing medications up from a vial or how to perform CPR in space when you're both floating around … There are all these things that need to be dealt with in the space environment. And these are all things that are being addressed for long-duration spaceflight. Then you have structural changes of the heart or eye changes that happen in microgravity. There are lots of changes that we don't have a lot of information about just yet. So it's a whole host of factors to consider, and we're going to have to be very well-versed in all of these space medicine issues to have successful long-duration space missions.

EMRA PH&DM: What's your advice for EM residents and medical students who are interested in the field and would like to learn/explore more? Does your program offer or plan to offer electives or rotations for prospective residents?

Dr. Aintablian: I think the biggest piece of advice is to be a good EM physician first. Learn your clinical skills really well because they're going to pay dividends later, even possibly in space. If you're actually interested in doing space medicine, and you know you're pretty committed to this, feel free to reach out. Email us, follow us on Twitter, follow me on IG. I definitely foresee rotation opportunities in the future. Right now, we're just focused on making this fellowship as great as it can be. But yeah, learn to be as good a clinician as you can be, and don't be scared to reach out if you've got any questions.

The UCLA Space Medicine Program is an EM-based fellowship designed to develop the next generation of flight surgeons who will advance the understanding of human physiology in space and directly support the medical endeavors of human space travel and planetary expeditions. Learn more about the program at https://www.emergencymedicine.ucla.edu/education/fellowships/space_medicine. Email spacemed@mednet.ucla.edu if you have any questions. Follow @UclaSpaceMed on Twitter and @Haig.MD on Instagram!

The interview with Dr. Aintablian was edited for length and clarity. ✶



Yes, We Scan: Change Core US Requirements to Include Pediatrics

Jenn Purcell, DO
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Emergency Medicine
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Attending Physician
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BACKGROUND

Appendicitis is the most common pediatric abdominal surgical emergency, with a lifetime risk of 7 percent. Ultrasound is the primary imaging modality in children to avoid the harmful effects of radiation.[1] The estimated lifetime risk of developing cancer after one abdominal computed tomography (CT) scan in a 1-year-old child is increased by 0.18 percent.[2]

Driven by pediatric emergency medicine physicians, recent trends in CT utilization in children have been reduced over the past 10 years, but use of the technique in children remains common. It was found that non-pediatric hospitals used CT for appendicitis in >70 percent of cases, compared with 25 percent of cases at children's hospitals.[3] However, most pediatric appendicitis cases will present to non-pediatric hospitals.[4] It was estimated that 57 percent of graduating EM residents will go on to practice community medicine, and thus require training to assist their patients outside of pediatric hospitals.[5]

Appendix point-of-care ultrasound (POCUS) has a sensitivity of 92 percent when evaluating for secondary signs of appendicitis (appendicolith, peri-appendiceal free fluid).[6] Imaging and management modalities can shift drastically based on the time of day (radiology ultrasound availability), practice location (freestanding or critical access ED, community vs. academic/pediatric hospital), EM faculty experience with POCUS, as well as surgeon preference. EM ultrasound education is continuously evolving with academic leaders re-examining what constitutes core content for POCUS.[7]

Our purpose is to increase the momentum behind adding appendi ceal ultrasound to core EM training competencies.

EXPLANATION

We surmise that with more focused pediatric abdominal ultrasound training in residency, a foundation of comfort and knowledge would eventually lead to a trickle-down effect to future generations of EM learners.

A second-year EM resident interested in pediatric emergency medicine POCUS spent a one-month advanced ultrasound elective focused on pediatric ultrasound.

References available online.

This elective included dedicated didactics focusing on pediatric sonography, simulation, and hands-on experience. The resident was required to submit >25 percent of the 150 scans required for the rotation on pediatric patients for quality assurance. Additionally, weekly POCUS rounds in the pediatric intensive care unit provided an opportunity for increased comfort and familiarity with scanning the pediatric patient and also supplanted times when ED pediatric volumes provided less opportunity.

DESCRIPTION

In 2012, the Council of Emergency Medicine Residency Directors (CORD) and the American Institute of Ultrasound in Medicine (AIUM) recognized appendix ultrasound as an advanced POCUS application.[8] This resident learner felt increasing confidence and familiarity not only with pediatric appendicitis but with a general approach to scanning children. She endorsed that patients' families were overall pleased to have these educational scans performed on their children. This resident was encouraged by the added value she brought to her patients and training program with this innovation.

As she progressed to a third-year resident, she had many cases of accurately diagnosed appendicitis by POCUS. This is an example of a perforated appendicitis diagnosed by this resident (**Figure 1**). The patient was a previously healthy 9-year-old male presenting to the ED with a chief complaint of sharp non-radiating right lower quadrant abdominal pain for three days. His mother endorsed multiple episodes of emesis. Physical exam showed a febrile male with right lower quadrant abdominal tenderness with accompanying rebound tenderness. Laboratory results revealed a leukocytosis of 24,000. The patient was seen by general surgery and taken directly to the operating room based on the resident's POCUS scan.

By using the aforementioned educational model, resident learners were able to accurately diagnose appendicitis by POCUS prior to obtaining comprehensive ultrasound, thereby expediting patient care.[9–11] Ideally, training would reflect a balance of both pathologic and non-pathologic scans, as well as a required percentage of all educational ultrasounds to be performed on pediatric patients. In times of reduced pediatric volumes or in adult only EDs, rounding in a pediatric unit may present more opportunity for exposure.

If pediatric abdominal ultrasound is routinely and deliberately taught to EM residents, a cultural shift away from obtaining abdominal CTs in children would be the desired outcome. ✶

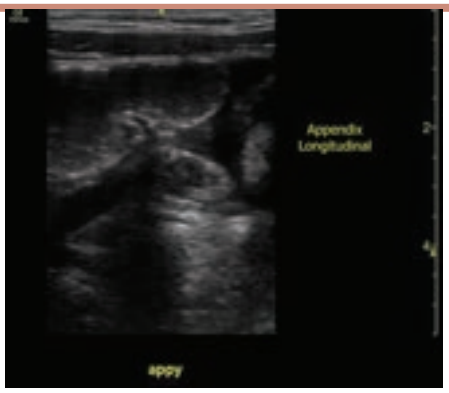

FIGURE 1

Disclaimer: This research was supported (in whole or in part) by HCA Healthcare and/or an HCA Healthcare affiliated entity. The views expressed in this publication represent those of the author(s) and do not necessarily represent the official views of HCA Healthcare or any of its affiliated entities.

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Update on State-of-the-Art Imaging and Stool Tests for IBD in the ED

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The approach to a suspected inflammatory bowel disease (IBD) flare in the ED involves a thorough diagnostic workup and addressing potential serious complications of the underlying disease. Although care may be transferred to an inpatient team, early ED workup utilizing state-of-the-art methods can facilitate earlier definitive intervention, significantly impacting hospital course and outcomes.

CASE

A 32-year-old female presents to the ED with 10 days of nausea, emesis, abdominal cramps, bloody diarrhea 10-12 times daily, and 7-pound unintended weight loss. She has had Crohn's disease (CD) since age 23 and no prior surgeries. She has received infliximab every 8 weeks for the last 4 years. Her last flare was 2 years ago, after which she received intravenous steroids. Temperature is 100.2°F, heart rate 102, blood pressure 90/58, respiratory rate 16, and oxygen saturation 98%. She is lying in the right lateral decubitus position, has dry mucous membranes, and has right lower quadrant and suprapubic tenderness to palpation with guarding but no rebound. The remainder of the physical exam is within normal limits.

INTRODUCTION

IBD is a chronic relapsing-remitting auto-inflammatory condition that includes Crohn's disease (CD) and ulcerative colitis (UC) caused by environmental exposures in genetically susceptible individuals. CD can affect any part of the gastrointestinal tract from the mouth to the anus and is more heterogeneous than UC, which begins at the rectum and can extend proximally into the colon. Extraintestinal manifestations include rheumatic, mucocutaneous, ocular, hepatobiliary, renal, pulmonary, and pancreatic disorders.[1] IBD is increasing in incidence and prevalence and is associated with ED visits,[2] healthcare utilization, and costs.[3]

The differential diagnosis for a suspected IBD flare should be broad[4] and include specific complications such as: fistulae, intra-abdominal abscess, stricture, obstruction, perforation, infection (opportunistic or non-opportunistic), toxic megacolon, NSAID-induced colitis, nephrolithiasis, malignancy, and hepatopancreatobiliary disorders. Abdominal examination of skin, scars, ostomies, masses, fistulae, and direct anal visualization is crucial, as well as appropriate laboratory tests and imaging.

DIAGNOSTIC STUDIES

Workups are extensive for suspected IBD flares and include imaging as well as laboratory tests requiring blood and stool samples. Stool samples can be logistically challenging to collect, and results may be required before antimicrobial or immunosuppressive therapy. Thus, early collection of stool samples in the ED is highly consequential, especially before symptomatic or other treatment, as earlier initiation of IBD therapy during flare is associated with better outcomes.[5]

Stool Studies: A stool sample can be used to evaluate for gut infections and general inflammation, which is an important distinction when considering antimicrobials or immunosuppression for IBD. An enteric pathogen polymerase chain reaction panel and C. *difficile* toxin testing account for most relevant organisms. Calprotectin is a calcium and zinc binding protein found in phagocytic cells and most abundant in neutrophils, effectors of acute inflammation.[6]

Fecal calprotectin is a recently developed stool analyte measured by enzyme-linked immunosorbent assay preferred by gastroenterologists over the nonspecific inflammatory markers C-reactive protein and erythrocyte sedimentation ratio. While fecal calprotectin is not specific to IBD, its elevation reflects inflammation specific to the gut, due to IBD, celiac disease, infectious colitis, intestinal cystic fibrosis, colorectal cancer, NSAID use, alcohol use, and/or another process.

Thus, elevated fecal calprotectin without other indicators can suggest an IBD flare, with levels correlating to clinical, endoscopic, and histologic inflammation and a negligible false negative rate.

Normal fecal calprotectin levels suggest a non-inflammatory process, such as irritable bowel syndrome or an extra-intestinal disorder. Sensitivity and specificity depend on disease severity and cutoff values and are generally considered high by specialists. Therefore, use of fecal calprotectin can obviate the need for urgent colonoscopy. Next-generation adaptations are being developed to improve accuracy, rapidity, and ease of sample acquisition.[7]

Imaging Studies: Imaging modalities utilized in IBD include plain film radiograph, computed tomography, magnetic resonance imaging, magnetic resonance enterography, ultrasound, esophagogastroduodenoscopy, colonoscopy, flexible sigmoidoscopy, and anoscopy.

IBD is a chronic disease often diagnosed at an early age, meaning

References available online.

patients have increased risk of cancer with cumulative radiation exposure due to frequent radiographic scans.[8] It is important to balance the risks of exposure to diagnostic radiation and to bowel preparation required for endoscopy with the sensitivity and specificity of each modality. Magnetic resonance enterography is preferred for IBD patients under 35 years of age[9] and is gaining favor for other patients as well. As technology and training improve, point-of-care ultrasound is increasingly utilized and has been shown to detect important IBD complications, such as bowel strictures and pericolic abscesses, in the ED.[10]

SPECIAL CONSIDERATIONS FOR INITIAL MANAGEMENT

Symptoms can be severe and debilitating even if not visually apparent. IBD patients may have had their symptoms dismissed or attributed to a psychosomatic condition. Assume symptoms are pathophysiologic until proven otherwise. While definitive management of an IBD flare typically occurs under care of specialist teams following admission to the hospital,[9,11,12] initial steps should be taken in the ED to stabilize the patient.

Surgery is avoided when possible in CD due to worse outcomes, but can be curative in UC. Avoid making abrupt changes to outpatient medications without consultation, as these regimens can be complex and extensively titrated. Avoid antibiotics unless there is high suspicion for infection, as certain antibiotics are associated with poor IBD outcomes. The gut-selective steroid budesonide may be used for mild to moderate flares, as it has reduced side effects compared to systemic steroids.

Most patients with severe IBD flares will be admitted to the hospital. Risk scores are being developed utilizing indexes at presentation to help determine disposition. For instance, presence of tachycardia and hypoalbuminemia were associated with complex disease and admission in a recent study.[13] Close follow-up with a gastroenterologist following discharge is critical, without which patients have increased ED visits.[14]

TAKE-HOME POINTS

- Maintain a broad differential diagnosis for IBD flare, including complications such as fistulae, abscesses, stricture, obstruction, and infection.
- Prompt initiation of appropriate workup in the ED facilitates earlier intervention and better outcomes.
- Obtain stool samples as early as feasible for laboratory testing.
- Prioritize magnetic resonance and ultrasound over radiographic imaging modalities to reduce cumulative radiation exposure. ✶

References available online.

Unusual Causes of Hematuria

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CASE

A 77-year-old male with a history of atrial fibrillation (not on anticoagulation), heart failure with reduced ejection fraction, hyperlipidemia, hypertension, gout, and prostate cancer (status post transurethral resection of the prostate in 2014) presents to the emergency department with right-sided flank pain for 2 days. The pain radiates to his back and right lower abdomen and is described as constantly aching, dull, and 8/10 in severity. He has been taking 800mg of naproxen every 6 hours with only mild relief. Earlier in the day, he had one episode of emesis.

In the ED, the patient is afebrile, blood pressure 192/90 mmHg, 100/min and irregular, and respiratory rate 18/min and non-labored. Pulse oximetry is 100% on room air. Labs are notable for a creatinine of 1.53 with eGFR 50. Although he denies hematuria or dysuria, urinalysis shows moderate blood.

The initial differential includes urolithiasis, pyelonephritis, and renal artery or vein thrombosis. Since the patient is not on anticoagulation for his atrial fibrillation, suspicion for infarction is high, and a CT abdomen and pelvis with IV contrast is ordered. The CT shows a subtotal infarct of the right kidney secondary to a proximal right renal artery thrombus (**Image 1**). Vascular surgery does not recommend any surgical intervention, and a heparin drip is started.

DISCUSSION

There are two kinds of hematuria: (1) macroscopic hematuria, which is gross blood in the urine, and (2) microscopic hematuria, which is blood in the urine visualized only under the microscope.[1]

Macroscopic hematuria can be caused by as little as 1mL of blood in 1 liter of urine and can be due to arterial or venous blood, though

References available online.

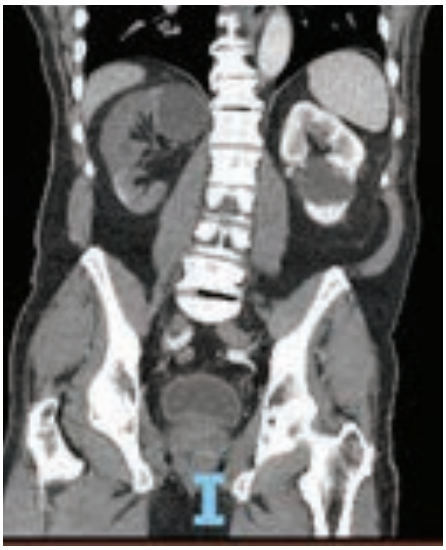

IMAGE 1: CT abdomen pelvis with IV contrast showing subtotal infarction of the right kidney due to a proximal right renal artery thrombus.

myoglobinuria from rhabdomyolysis and hemoglobinuria from hemolysis can also turn urine a red color.

The definition of microscopic hematuria can vary but is generally thought of as 3 or more red blood cells per high-powered field.1-4 A false negative result on urine dipstick for hematuria can occur from low urine pH, high urine specific gravity, or high dietary vitamin C.[1,2] Certain medicines and foods, including rifampin and beets, can cause urine to appear red grossly but will be negative for hematuria on dipstick or urinalysis.[2] Though the true causes of hematuria can be extensive, this review will focus on the most common and emergent causes.

When working up a patient with hematuria, the clinician must consider the larger context in which the patient is presenting. For a patient with colicky flank pain, costovertebral angle tenderness, and hematuria, a non-contrast CT abdomen and pelvis is an excellent first imaging modality choice, but what does the clinician do when that CT comes back negative for renal stones? Additionally, what about the patient presenting with atypical flank pain? Gross hematuria without any flank pain? These patients may warrant broadening the differential and further investigation with additional imaging in the ED.

UROLITHIASIS

A common cause of hematuria is urolithiasis, although 10-20% of patients with urolithiasis will present without hematuria. Microscopic hematuria on urinalysis has a 69-84% sensitivity for urolithiasis. One study by Mefford et al found that more severe obstructive uropathy correlated with a lower incidence of hematuria, although the reasons for this are unclear.[5] Non-contrast CTs are currently the best imaging modality for diagnosing kidney stones, with a sensitivity of 97–100% and specificity of 94–96%. Stones larger than 5mm are more likely to cause severe hydronephrosis and obstruction and are more likely to require urology consultation.[5]

URINARY TRACT INFECTION

Urinary tract infections cause up to 20% of microscopic hematuria cases and less commonly may also present with macroscopic hematuria.[3] On urinalysis, positive nitrites, leukocyte esterase, WBCs, and bacteria may all be indicative of a urinary tract infection.[2-4] Left untreated, a urinary tract infection could turn into pyelonephritis. However, in the setting of a patient with fever and colicky flank pain, an infected obstructive kidney stone must be considered.[5] If the hematuria does not resolve after the infection has been appropriately treated, further investigation into alternative causes of hematuria should be initiated.[3]

MALIGNANCY

Asymptomatic hematuria ranges from 2-31% in the general population and does not necessarily portend pathology.[1,3] However, 20-40% of patients with macroscopic hematuria have an underlying urological malignancy, usually bladder cancer. Up to 90% of patients with urologic malignancy initially will present with hematuria. Even a single episode of macroscopic hematuria — even in patients on anticoagulation — should prompt further testing for malignancy.[2,6]

RENAL ARTERY THROMBOSIS

Renal infarction presents similarly to renal stones; this can lead to delayed diagnosis.[7] The patient will have flank or abdominal pain, nausea, and vomiting, with labs showing leukocytosis and elevated lactate dehydrogenase.[7] Renal infarction is more commonly caused by renal artery thrombosis, either by chronically forming cholesterol-based clots in patients with hyperlipidemia or acute dislodgement of a blood clot from the left atrium in patients with atrial fibrillation. Endocarditis, trauma, and other pathology may also precipitate an embolic event.[7,8,9] As patients present with similar symptoms as those with urolithiasis (i.e., flank pain and hematuria), the clinician must have a high index of suspicion for this often-missed diagnosis.

The imaging modality of choice is a CT angiogram of the abdomen, which has 100% specificity for renal artery thrombosis.[7,8] A CT with or without contrast may still diagnose the infarction, but it is much less specific. Sending these patients home without the correct diagnosis can be life-threatening. These patients need to be admitted, as the decreased perfusion to the kidney causes kidney injury and potentially irreversible kidney failure if not treated with heparin or local thrombolysis from interventional radiology.[8,9]

Patients on anticoagulation therapy may still present with renal infarction, either due to failure of anticoagulation therapy or medicine non-adherence. However, anticoagulation medications can also cause gross hematuria from conditions that, without anticoagulation medications, may not have caused as much or any hematuria. In addition, studies have shown an association of higher complications of hematuria (including hospitalizations and urologic procedures for hemorrhage control) for patients on antithrombotic medication.[10]

RUPTURED ABDOMINAL AORTIC ANEURYSM

As abdominal aortic aneurysms (AAAs) are often asymptomatic, a patient's first presentation for AAA will often be for AAA rupture, a dangerous complication that has up to a 75% mortality.[11,12] Aneurysms considered at risk for rupture and requiring preemptive surgical

References available online.

intervention include those over 5.5 cm in size or those increasing 0.5 cm or more in size over 6 months.[11,12]

The textbook presentation of ruptured AAA is a pulsatile abdominal mass with a bruit and high output cardiac failure. Several studies have shown, however, that while these findings are only present in some patients (around 50%), hematuria will be present in upwards of 87% of patients.[13,14] As this is an unusual cause of hematuria, it may delay the diagnosis, though further data is needed to investigate the impacts of hematuria as the chief complaint on ruptured AAA mortality.[13] The diagnosis can be confirmed by ultrasound or CT angiography, and definitive management is by open or endovascular repair.[11,12]

TUBERCULOSIS

Classically, tuberculosis will present with pulmonary symptoms, night sweats, and weight loss, but when tuberculosis invades extra-pulmonary systems, its presentation can be strange and unexpected. There have been multiple case reports of genitourinary tuberculosis presenting as hematuria with or without dysuria.[15,16] Diagnosis may be elusive, as less than 5% of these patients have active pulmonary tuberculosis.[15,16] It is often only recognized in the late and chronic stage, by which time it may have caused renal parenchymal destruction, ureteral strictures, or end-stage renal disease.15,16 Though biopsy may be necessary, urine culture and PCR testing are key to diagnosis, and treatment is similar to that for pulmonary tuberculosis.[15,16]

NEPHROPATHY

From familial diseases such as Alport syndrome to the acquired diseases of post-streptococcal glomerulonephritis or IgA nephropathy, there are multiple other causes of hematuria to consider.[2,6] These are less common and usually present within the setting of other signs and symptoms to clue the physician in. If the patient is stable and otherwise doing well, outpatient nephrology follow-up may be appropriate after the initial ED workup.

CONCLUSION

Hematuria in the setting of flank pain is a common presentation to the ED. As kidney stones affect up to 15% of the population, it is often standard to order a non-contrast CT abdomen and pelvis in these patients to search for a stone.[5] However, in the setting of a negative non-contrast CT scan and especially in patients with risk factors for renal infarction, the next step should be a CT angiogram to rule out renal vein or artery thrombosis. Other unusual causes of hematuria to keep in mind are ruptured abdominal aortic aneurysm and tuberculosis, as these can be life-threatening if not recognized. ✶

References available online.

Overlooked Sources of Bias in Adult Out-of-Hospital Cardiac Arrest Research

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ABSTRACT
Bias is prevalent throughout the medical literature; however, out-of-hospital cardiac arrest (OHCA) studies have unique sources of bias due to the complex disease state. These sources of bias are often overlooked without efforts to mitigate their effects on study results. We review commonly overlooked sources of bias specific to OHCA studies to facilitate critical appraisal.

INTRODUCTION AND BACKGROUND

Sources of error threaten the validity of medical research. In medical studies, bias is the non-random effect of error on study results.[1] Although there are many types of bias,[1,2] each type can have various causes.[1,3] Bias is prevalent throughout the medical literature,[3,4] and it has variable consequences, from insignificant changes in effect magnitude to false positive and false negative study results.[3] However, studies on OHCA have unique causes of bias.

OHCA research has the potential for impactful improvements in patient care; however, there are multiple barriers to conducting unbiased OHCA studies. OHCA occurs when the heart stops due to an abnormal cardiac rhythm.[5] It is common, occurring in 60-140 adults per 100,000 population; however, only 9% of OHCAs treated by emergency medical services had survival with good neurological outcomes.[6] Despite this disease burden, OHCA is challenging to study with multiple etiologies, an unpredictable onset, an immediate need for intervention, and a complex recovery phase.[7,8] These challenges create avenues for bias affecting enrollment, treatment,

References available online.

analysis, and implementation of OHCA studies.

Recognizing these unique sources of bias in OHCA research is critical to preventing misinterpretation and misapplication of study results. Therefore, the purpose of this review is to summarize commonly overlooked sources of bias that impact OHCA research.

REVIEW: ENROLLMENT

OHCA studies often exclude patients with characteristics that are expected to have poor outcomes, including those with non-shockable cardiac rhythms.[9] While this mitigates the risk of false negative study results, it can impair the generalizability of the study population. This selective enrollment of subjects is known as sampling bias, occurring when enrollment yields a study population that differs systematically from the population of interest.[2] When sampling bias underrepresents one sex over another, a sex bias may occur.[10] For example, excluding OHCAs with non-shockable rhythms can result in a lower proportion of female subjects in the study population since women in OHCA are less likely to present in a shockable rhythm.[11] Therefore, inclusion and exclusion criteria in OHCA studies may inadvertently cause a sex bias and make study results less applicable to female patients. Readers should look for a representative sample of female subjects.

Similarly, black race is associated with predictors of poor OHCA outcomes that may preclude enrollment, resulting in a race bias. For example, in a systematic review and meta-analysis of 15 OHCA studies evaluating outcomes between races, black subjects were less likely to have an initial shockable rhythm, bystander resuscitation, or a witnessed arrest compared to white subjects.[12] Therefore, black subjects may be systematically excluded from enrollment of OHCA studies due to predictors of poor outcomes, which may make study results less applicable to black patients. Readers should look for a representative sample of minority subjects.

REVIEW: TREATMENT

Similar to sampling issues during enrollment, the selection of patients for post-arrest interventions can be a significant source of bias. Like intra-arrest interventions, post-arrest care is critical to OHCA outcomes.[13] However, unlike intra-arrest interventions such as chest compressions and basic airway management that are indicated for all OHCA patients,[14] aspects of post-arrest care may be provided to study treatment groups unequally due to patient-related factors. This selective treatment indicates a selection bias resulting from study groups differing in ways other than the defined independent variables.[2]

For example, post-arrest cardiac catheterization and implantable cardioverter defibrillators are associated with reduced all-cause mortality, but not cardiac-specific mortality as would be

References available online.

expected.[15] Therefore, those who receive these interventions are less likely to die from non-cardiac causes than those who do not.[15] While this difference questions the efficacy of these interventions post-arrest, it also indicates a selection bias in who receives these interventions post-arrest.[15] Therefore, the selection of patients for post-arrest treatments may compromise OHCA study results. Readers should look for measurement of and adjustment for post-arrest interventions.

REVIEW: ANALYSIS

A lack of measurement and adjustment of confounding variables is a common source of error in OHCA study analyses. Confounding variables are factors associated with the exposure and outcome of interest that mask the true association or lack of association.[2] For example, end-of-life decisions may confound mortality outcomes in OHCA studies. However, in a systematic review of 178 randomized control trials of OHCA with in-intensive care unit mortality as an outcome, only 62 (35%) studies addressed end-of-life decisions in their methodology.[16]

Similarly, channeling bias occurs when the severity of illness dictates the exposure.[3] For example, a patient who is comatose after resuscitation from OHCA has lower odds of survival and would receive additional treatments like mechanical ventilation compared to a patient following commands and protecting the airway.[17] However, in a review of 213 OHCA studies with survival outcomes, only 60 (28%) included illness severity or comorbidity indices and scores, and only 39 of those used the indices and scores to adjust for confounding.[18] Therefore, readers should look for measurement and adjustment for end-of-life decisions, illness severity, and comorbidities, which may confound OHCA study results.

Timing of interventions intra- and post-arrest are also major confounders in OHCA study analyses. Resuscitation time bias refers to the concept that the longer a patient is in cardiac arrest, the more likely they will receive interventions but the less likely they will survive.[19] This association may bias cardiac arrest studies by making intra-arrest interventions like positive pressure ventilation, which usually occurs later in the resuscitation, seem less effective.20 Meanwhile, immortality time bias refers to the concept that the longer a critically ill patient (i.e., a post-arrest patient) is alive, the more likely they will receive an intervention and survive.[19] In contrast to resuscitation time bias, immortality time bias may make post-arrest interventions like cardiac catheterization seem more effective.[21] Therefore, readers should look for measurement of and adjustment for the timing of interventions, which may confound OHCA study results.[19]

REVIEW: IMPLEMENTATION

OHCA studies often ignore implementation barriers to the studied interventions. Barriers to implementation threaten the generalizability of study results or their applicability to specific populations.[3] For example, OHCA patients in rural or difficult to access locations (e.g., high-rise buildings) have worse outcomes due to longer response times and practical barriers to accessing advanced care (e.g., endotracheal intubation).[22-25] Therefore, an intervention may be efficacious when there is a brief response time and more experienced clinicians, but it may be less efficacious when there is a prolonged response time and less experienced clinicians.

Conversely, some interventions facilitate the care of OHCA in rural or difficult to access locations but do not improve outcomes overall. For example, mechanical chest compression devices do not improve outcomes compared to high-quality manual chest compressions.[26] However, these devices have practical benefits for clinicians performing prolonged resuscitations or chest compressions in a moving ambulance,[26] mitigating existing implementation barriers. Therefore, readers should consider implementation barriers when interpreting study results.

CONCLUSIONS

Although bias is pervasive in medical research, OHCA studies have unique sources of bias that are infrequently addressed in study methods (**Table**). Recognizing these biases is vital to the critical appraisal and application of the OHCA literature. Therefore, OHCA study results should be interpreted within the limitations of these sources of bias. ✶

TABLE: Summary of Overlooked Sources of Bias in Out-of-Hospital Cardiac Arrest Research

SOURCE OF BIAS	MITIGATION STEPS
Sex Bias in Enrollment	Enrollment of a representative sample of female subjects
Race Bias in Enrollment	Enrollment of a representative sample of minority subjects
Selection Bias in Post-Arrest Care	Measurement of and adjustment for post-arrest interventions
End-of-Life Decisions, Illness Severity, and Comorbidities	Measurement of and adjustment for these confounding variables
Resuscitation Time Bias and Immortality Time Bias	Measurement of and adjustment for timing of interventions
Implementation Barriers	Consideration of local practice environments

References available online.

Verapamil in SVT: NO NEED TO DROP THE BEAT

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INTRODUCTION

In the ED, acute arrhythmias — including paroxysmal supraventricular tachycardias (pSVT) — gather much attention. Upon recognition of the arrhythmia, there is often a storm of actions including EKGs, intravenous access, and defibrillator pad application. A patient who is already experiencing symptoms of palpitations, chest pain, or dyspnea will frequently be disturbed by the sensations and experience a sympathetic adrenergic response.

Pediatric patients in this situation will often and understandably have difficulty controlling their emotions. While this is a common occurrence with 1.26 million cases estimated episodes in 2018 alone,[1] treatment of pSVT itself comes with a sense of urgency, a need to act and reach for reliable medications such as adenosine to "break" them from their abnormal rhythm.

It is worth considering that adenosine administration can compound the problem by causing distinctly uncomfortable symptoms associated with the sino-atrial pause. Calcium channel blockers (CCBs), which were a previously commonly used pharmacologic tool for pSVT, can offer a reliable and smoother transition from a tachyarrhythmia to a normal sinus rhythm in a more comfortable way for the patient.[2]

In this case report, we depict how verapamil was used in an extremely anxious child with autism during a pSVT episode to restore him to a normal sinus rhythm with no complications.

CASE DESCRIPTION

The patient in question, transferred from a local urgent care facility, was a 10-year-old male with autism spectrum disorder and new onset supraventricular tachycardia (SVT). Per the family, the patient awoke at midnight complaining of chest pain and palpitations. His mother, a former nurse, noted that his heart rate was beating fast and took the patient to an urgent care center. At the urgent care center, vagal maneuvers of blowing into a syringe and ice water to the face (so-called "dive reflex") were attempted but unsuccessful. The patient was extremely anxious despite intranasal midazolam and resistant to placement of an intravenous catheter. At 100kg, he was strong enough to make restraint impossible to safely perform by the staff at the urgent care facility. The patient was transferred to the nearest pediatric referral hospital for management of his SVT and for cardiology evaluation.

On arrival, the patient continued to complain of chest pain, was extremely anxious, and threatened violence in response to attempts at IV access. The mother noted the patient had a history of escalation. Verbal redirection, negotiation, and maternal efforts at de-escalation all failed. Intramuscular ketamine was used to transiently sedate the patient to allow us to get IV access. With the patient sedated, we were able to attach the cardiac monitor/defibrillator and get a reliable EKG showing a narrow complex tachycardia consistent with pSVT with a rate of 208 bpm (**Figure 1**). Given the significant anxiety of the patient and his poor coping mechanisms, an effort was made to avoid the unpleasant side effects that accompany

References available online.

adenosine. The decision was made to use a CCB.

After attaching the cardiac monitor, preparing the room, and outlining the plan with the care team and family, we gave the patient 5 mg of intravenous verapamil over a 2-minute slow push. In the next 5 minutes, the patient's heart rate steadily trended downward from greater than 200 to 180 before abruptly converting to 110 beats per minute. Repeat EKG at that time showed a pediatric normal sinus rhythm (**Figure 2**). The patient expressed no discomfort or startle response to the heart rate change. The patient was observed in the ED for 1 hour with no resumption of his SVT. He was then transferred to the pediatric intensive care unit for close cardiac monitoring. No further episodes were reported. A follow-up echocardiogram showed no structural abnormalities. The patient was discharged to cardiac follow-up on no new medications.

DISCUSSION

Paroxysmal supraventricular tachycardia (pSVT) is an umbrella term that can refer to one of several pathologies, such as atrioventricular node re-entrant tachycardia and less commonly atrioventricular reciprocating tachycardia, as well as several other less common atrial tachycardias. Together, these pathologies represent a circling electrical depolarization occurring within the atria, above (or within) the atrioventricular node. The methods of treating pSVT generally modulate the AV node with vagal maneuvers, pharmacokinetics, or cardioversion to terminate the re-entrant depolarization.[3]

The management of a patient with pSVT is primarily determined by their blood pressure; as with most tachyarrhythmias, hypotensive patients receive cardioversion (usually 0.5J/kg). Next, it is important to examine the EKG for QRS width and regularity. A narrow, regular QRS complex without preceding P-waves indicates that AV nodal blocking may be safe. However, irregular, wide complexes should prompt concern for the presence of an accessory pathway, which can result in ventricular fibrillation.

If there are no barriers, the first attempt to break the pSVT is often a vagal maneuver such as bearing down, dive reflex, or leg lifts. These maneuvers work by stimulating the vagus nerve, which directly inhibits the AV node, and are frequently sufficient to terminate the re-entrant cycle. Bearing down with and without leg lifting afterward is successful 43% and 17% of the time, respectively. If vagal maneuvers are not successful, then a pharmacologic intervention is usually required.[4]

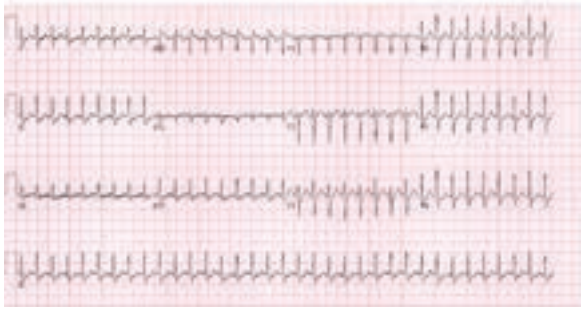

FIGURE 1: Paroxysmal SVT. Note the tachycardia (>200 bpm) with a regular rhythm and the lack of evidence of a delta wave.

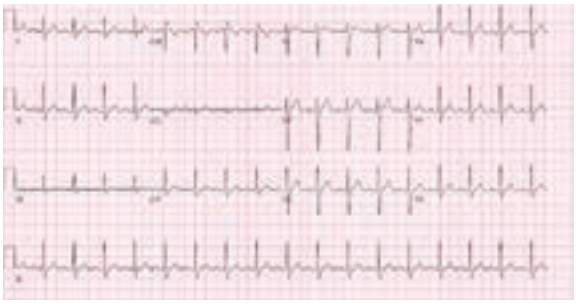

FIGURE 2: Normal pediatric sinus rhythm

ADENOSINE VS. CALCIUM CHANNEL BLOCKERS

Adenosine acts as a strong atrioventricular nodal blocking agent with a short half-life (less than 1 minute). It must be administered aggressively and meticulously to ensure a brief but full block of the AV node; a slow push or inexperienced administration can result in a failure.

Dosage of adenosine is well understood — 0.1mg/kg for the first dose (maximum of 6mg) followed by 0.2mg/kg (maximum of 12mg) for the second and third doses.2 Unfortunately, adenosine has short-lived but very unpleasant adverse reactions of dyspnea, chest pain, and a sense of impending doom.[5]

Non-dihydropyridine CCBs like verapamil and diltiazem are the classic CCBs used for pSVT. CCBs act by inhibiting the calcium movement across the cardiac cell membrane, delaying cascade depolarization. This effect is most prominent in the electrical conduction system (the SA and AV nodes) and results in perturbing the re-entrant cycle, allowing a normal sinus rhythm to resume. Diltiazem is dosed at 0.25mg/kg IV over 2 minutes with a re-bolus at 15 minutes later of 0.35mg/kg as needed.[6] Verapamil is contraindicated in infants as there have been rare reports of asystole and hypotension, but it is usually well-tolerated with an IV dose of 0.1mg/kg (maximum 5mg) and subsequent doses of 0.2 mg/kg (maximum 10mg) as needed.[7,8]

When compared to adenosine, CCBs are inexpensive, require less coordination for administration of the medication, and are not accompanied by the same patient discomfort. CCB side effects worth considering are hypotension and dizziness; much more rarely AV block and pulmonary edema can occur. Prophylactic doses of calcium infusions can reduce the effect of verapamil-induced hypotension and improve efficacy of the verapamil bolus.[9] Either adenosine or verapamil can be used safely in pregnant or breastfeeding mothers.[10]

TAKE-HOME POINT

When a pediatric patient presents to the emergency department with pSVT and vagal maneuvers have failed, choice of AV nodal blocking agent is important. This case shows that CCBs offer several advantages over adenosine, which should prompt a more careful consideration of which agent to use in pediatric patients. Adenosine is the first-line agent, but it is worth pausing to consider the benefits of alternative agents. ✶

References available online.

Possessive Eponyms: Removing the Apostrophe from Medical Diagnoses

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Before groaning about diving into grammatical nuances in medicine, let us remember that how we speak about our patients often mirrors how we treat them. Language is powerful and intentional. Are you familiar with the American Medical Association (AMA) Manual of Style? It is not about jogger-pant scrubs and stitched, collared white coats. It is a guide to the writing, editing, and citation styles that form the language of medical publications. Perhaps you used the Modern Language Association (MLA) or Chicago Manual of Style (CMOS) format in college, but medical journals tend to follow the AMA Manual of Style. Originally published in 1962, the much anticipated 11th edition was released in 2020, both in print and online.[1] Among its hundreds of pages of grammatical rules are guidelines on the use of apostrophes in medical eponyms.

What's the deal with apostrophes and why should we care? What we are actually referring to are possessive eponyms, such as Bell's palsy, Down's syndrome, Alzheimer's syndrome, Grave's disease, Crohn's disease, etc. Both the American Medical Association and the World Health Organization have suggested that we retire this use of possessive eponyms.[2,3] The practice suggests, misleadingly, either a sense of ownership or perhaps a personal affliction of the individual named. Additionally, eponyms complicate and misguide literature searches. The use of possessive eponyms is a matter of habit, and change is long overdue.

Eponyms have traditionally been used to indicate the describer, the patient, or the location where an illness or finding was first described. Historically, the possessive form has been used, but the non-possessive form is

References available online.

becoming more accepted. If you want to geek out even further, naming a disease based on a location (e.g., Lyme disease) is actually a toponym, not an eponym (a highly encouraged icebreaker for your next cocktail party). Possessive eponyms suggest that someone owns a disease. The National Down Syndrome Society made a push to shift terminology from Down's syndrome to Down syndrome, since the syndrome does not belong to anyone.[4] If the name of a disease or syndrome should rightfully belong to anyone, it would be the patient, not the discoverer.

Using possessive eponyms makes searching the literature confusing and inefficient. A study that compared PubMed searches for common eponyms with and without the "'s" found large discrepancies in search results depending on how the term was searched. Even though the non-possessive eponym is recommended, searches that did not include the possessive form missed hundreds of potentially relevant results.[5] Furthermore, when specifically looking at a disease process like Grave's disease, results were confounded with the adjective "grave." (And if you are a patient, it does not inspire hope to be told you have a Grave disease).

The recommendation against possessive eponyms has been in place since 1974,[6] and yet, we still can't say "Bell palsy" without raising an eyebrow (Get it? Raising an eyebrow?). Shouldn't it be Bell's palsy? Or Bells palsy? Can you really bring yourself to say "Bell palsy"?

Say it with me: Bell Palsy. Alzheimer syndrome. Down syndrome. Crohn disease. We have all said and heard stranger things in our careers. We can do this.

We can take this one step further. Instead of Bell palsy, it should be "idiopathic facial palsy." Why use the eponym at all? I'm not sure we are fully ready for this diversion from our medical school textbooks that we worked so hard to memorize. But think on this…why should a patient's pathology be named after anyone?

For CorePendium, we are developing our own style guided by the principles outlined in the AMA Manual of Style, but with adaptations and modifications in some instances to reflect our own editorial perspective (for example, we format our references to include links to the article's abstract in PubMed). Spirited internal debate about possessive eponyms fills our Slack channels. Should we follow the path of grammatical righteousness? Or should we cave in to the commonly typed words in the Wiki search? The time is now to abandon the possessive eponym. ✶

References available online.

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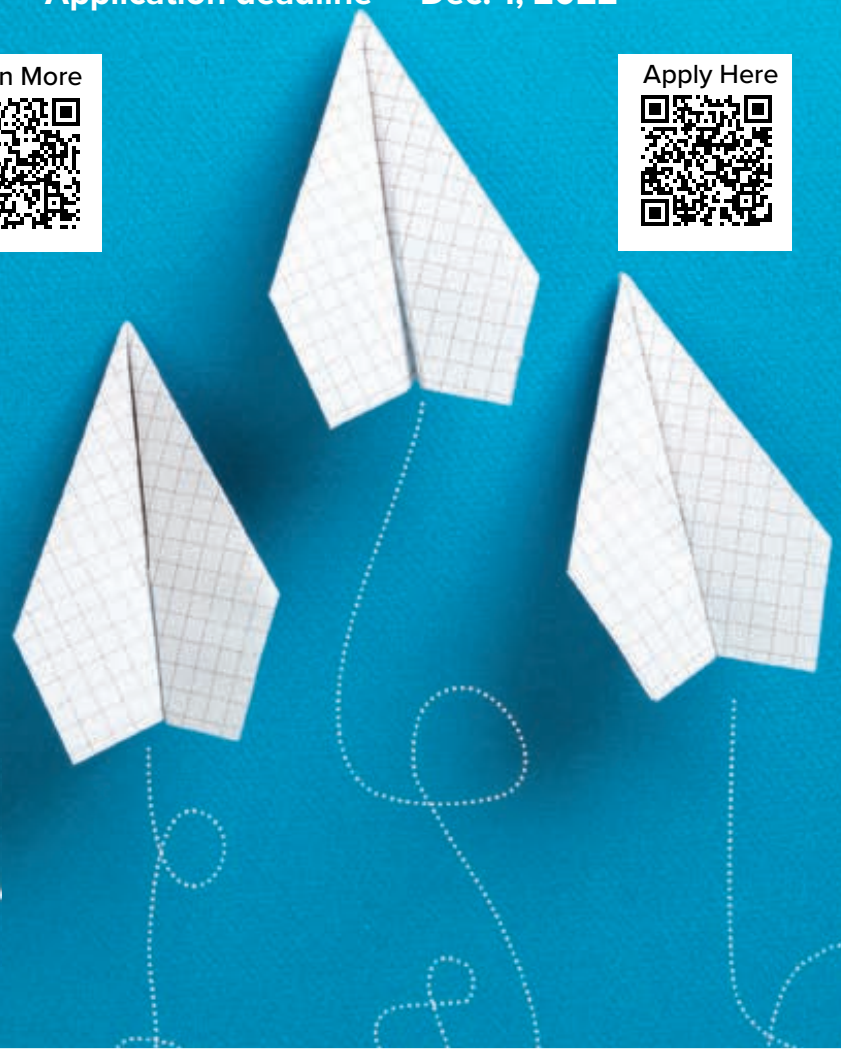

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Complications of Elective Medical Abortions

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CASE VIGNETTE

A 21-year-old female, G1P0A1, presents to the emergency department with a chief complaint of vaginal bleeding. The patient reports she had a medical abortion 5 days ago and has been bleeding since. She was 7 weeks gestation by her last menstrual period at that time. She decided to come in today because last night she soaked through 8 pads in 3 hours, so she is becoming increasingly nervous that something could be wrong. Patient reports she has abdominal cramping that has not been relieved by taking Advil. She believes she has had a fever but does not have a thermometer at home. She denies nausea and vomiting at this time.

INTRODUCTION

There are two methods to terminate a pregnancy: medical and surgical abortion. Surgical abortion, also known as uterine aspiration, is the most common type of abortion. It is usually performed in the first trimester, meaning up to 14 weeks of gestation in an outpatient setting. According to the Guttmacher Institute, two-thirds of all abortions (surgical and medical) occurred at 8 weeks of pregnancy or earlier, and 88% occurred in the first 12 weeks.[1] The procedure consists of premedications, infection prevention, cervical block, paracervical dilation, and finally, manual or electrical vacuum aspiration.[2]

Medical terminations can be used earlier in pregnancy and require no surgical procedure. Two medications are typically used: 200 mg of mifepristone, a progesterone antagonist, followed by 800 mcg of misoprostol, a prostaglandin analog, 24 to 48 hours later. The combination of mifepristone and misoprostol are FDA-approved to terminate a pregnancy up to 70 days (10 weeks) of gestation.[3] Although the majority of abortions are completed surgically, the number of medical abortions increased by 123% from 2010 to 2019. Of early first-trimester abortions completed, where an early abortion is defined as occurring at 9 weeks gestation or less, 53.7% were medical abortions.[4]

Patients may present to the emergency department following an abortion due to adverse events. Post-medical abortion visits are more frequent than post-surgical abortion visits due to both typical and atypical symptoms.[5] Typical symptoms include self-limiting abdominal pain, cramping, vaginal bleeding, fever, headache, dizziness, nausea, vomiting, and diarrhea. It is atypical for any of these symptoms to be severe enough that oral, over-the-counter medications cannot provide relief.

While treating patients who have recently had a medical abortion, it is important to elicit key details such as when the abortion occurred and their last menstrual period to approximate the gestational age. Mifepristone and misoprostol have expected side effects,

References available online.

but also red flag signs. Patients must be counseled on what to monitor for at home that would prompt them to present to an ED. Incomplete abortions, where some products of conception are not expelled, are more likely with medical abortions and can lead to life-threatening complications. Therefore, emergency medicine providers should be prepared to evaluate and treat patients who have had medical abortions with a keen awareness of the anticipated adverse events and the less common, though more dangerous, complications.

EXPECTED ADVERSE EVENTS

Self-limiting signs and symptoms are expected after a medical abortion. A patient should be prepared based on appropriate physician counseling prior to the abortion. Patients' symptoms tend to be more severe with increased age of gestation.

Symptoms may include:

- Cramping and abdominal pain that will gradually improve over 3 to 14 days
- Bleeding, usually heaviest 3 to 8 hours after taking misoprostol
- Self-limiting low-grade fever
- Self-limiting mild headache and dizziness without loss of consciousness
- Self-limiting nausea, vomiting, and diarrhea

SIGNIFICANT ADVERSE EVENTS

Major adverse events in patients who underwent a medical abortion may be life-threatening, requiring urgent and appropriate evaluation and careful monitoring by the physician. Rare, however possible, events include hemorrhage, incomplete abortion, and infection.

Symptoms may include:

- Heavy bleeding, defined as soaking through 2 menstrual pads (thick pads, not thin liners) per hour for at least 2 hours
- Cramping and abdominal pain that cannot be controlled with oral over-the-counter pain medications (such as acetaminophen or ibuprofen)
- A persistent temperature above 38.0° for more than 4 hours despite appropriate dosing of antipyretics.

ED MANAGEMENT

When a patient presents to the ED with a complaint following a recent medical abortion, a thorough history and physical exam are essential. History should include gestational age, time, dosing, and route of administration for each medication, as well as pertinent medical history, such as bleeding, coagulopathy disorders, and conditions that would increase likelihood of infection like diabetes or chronic immunosuppression. Additionally, previous gynecological surgeries and prior obstetric history, including route of delivery of children and prior elective or spontaneous abortions, should be noted.

The physical exam includes vital signs, abdominal exams, and pelvic exams. A pelvic exam will provide further information about the quantity of bleeding and whether there is uterine atony. Presence of blood pooling in the vaginal vault and active or even brisk oozing from the cervical os can help characterize the degree of bleeding. A pelvic ultrasound study should be conducted to determine if there are retained products of conception, accumulation of blood in the uterus, or free fluid in the abdomen. Laboratory values should be studied and include CBC, quantitative beta HCG, coagulation studies, and type and screen.[6] A type and screen test is for the purpose of assessing need for Rhogam for possible Rh isoimmunization and preparing in case a blood transfusion is needed.

Management of the patient will depend on the physical findings and studies completed. For patients who need further treatment due to complications — like a laceration, or needing a dilation and curettage (D&C) for retained production of contraception — obstetrics and gynecology will be consulted. For uterine artery embolization, interventional radiology would be consulted, typically after discussing the case with obstetrics and gynecology. If the patient has a significant hemorrhage, she may require a blood transfusion in the ED and admission to an ICU. In the case of infection, the typical diagnosis is endometritis, or an infection of the endometrial lining, which can occur whether or not retained products of conception remain in place. For endometritis, patients require broad-spectrum antibiotics and fluids. If either a massive hemorrhage occurs or the patient becomes septic and does not improve after 24-48 hours of antibiotic treatment, a hysterectomy may have to be performed.[6]

SUMMARY

It is typical for a patient to have side effects after a medical abortion. Differentiating expected versus significant adverse events is crucial to providing proper treatment. Patient education prior to administration of medications to terminate a pregnancy is vital, as many patients present to the ED afterward with anticipated adverse events. These visits could be limited if patients had a more in-depth understanding of what to expect throughout the process and for the days to weeks after undergoing a medical abortion. Providers must discuss red flag signs with these patients in case their symptoms persist and become severe, requiring additional medical attention after they have been discharged.

VIGNETTE CONCLUSION

The patient was evaluated in the ED. She was hemodynamically stable and currently afebrile, her hemoglobin demonstrated mild anemia and a decrease from baseline, and a transvaginal ultrasound showed signs of retained products of conception. She was taken to the operating room for a suction dilation & curettage for an incomplete abortion. Intraoperative ultrasound demonstrated a thin endometrial stripe, also known as an empty uterus. She was discharged home with strict return precautions, including heavy bleeding exceeding saturating 2 pads in an hour for at least 2 hours, severe cramping or abdominal pain that cannot be controlled with oral over-the-counter pain medications, or a temperature exceeding 38.0° that cannot be controlled with antipyretics. ✶

References available online.

Tearful from Topiramate: A Case of Bilateral Secondary Angle Closure Glaucoma

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We present the case of an adult male with bilateral eye pain and visual changes. He is ultimately found to have bilateral acute angle closure glaucoma secondary to topiramate toxicity. Acute angle closure glaucoma is a common ocular emergency. Rapid diagnosis, immediate intervention, and appropriate referral directly correlate with patient outcome and morbidity. Secondary acute angle closure glaucoma is more commonly a unilateral diagnosis.[10] This is an unusual case of bilateral acute angle closure glaucoma.

CASE

A 72-year-old male with a history of migraines presented to the ED with bilateral vision loss and eye pain for two days. He was driving cross country when he developed a migraine headache and decreased vision. The pain was localized behind his eyes and worse on the right side. He described this as being different from his normal migraine. Initially, he presented to a rural hospital. A CT scan of his head was negative for an acute intracranial abnormality. The patient was treated symptomatically with an oral analgesic medication, which mildly improved his symptoms, and he was discharged.

The following morning, he woke and immediately noticed decreased vision in both eyes, photophobia, large visual field defects as well as redness and tearing in both eyes, with his right eye being more affected than his left. At this time, he presented to the ED at a large academic center. Further history revealed he wore glasses for reading but no contacts and had no previous ophthalmologic conditions or surgeries. He mentioned he was started on topiramate the week prior as a prophylactic medication for his chronic migraines.

His systemic vitals were within normal limits, but the patient's eye exam was concerning for acute ocular pathology. His visual acuity was decreased with 20/150 OD and 20/50 OS. His intraocular pressure (IOP) was increased bilaterally, 39 mmHg on the right and 35 mmHg on the left. He had no pain with extraocular movements and full ocular range of motion bilaterally. However, the patient had multiple visual field defects in both eyes. His right eye was found to have a mid-dilated minimally reactive pupil, and both eyes appeared injected, cloudy, and tearful. The remainder of his physical exam — including cranial nerves, gross systemic sensation, and motor function — remained intact.

Based on his history and physical exam, he was diagnosed with bilateral acute angle closure glaucoma, and treatment was initiated to decrease his IOP.

PATHOPHYSIOLOGY

Angle closure glaucoma occurs when there is narrowing of the anterior chamber angle. A normal angle allows for aqueous humor to drain out of the anterior chamber of the eye. When the angle is narrowed, accumulation of this fluid causes increased IOP and damage to the optic nerve. Normal IOP is approximately 8-21 mmHg. In acute angle closure glaucoma, pressures are often greater than 30 mmHg.[5]

The two main types of acute angle closure glaucoma are primary and secondary. Primary occurs due to genetic susceptibility and the intrinsic characteristics of the eye, whereas secondary occurs as a result of an external event causing the angle to close (for example, a mass or hemorrhage in the posterior aspect of the eye pushing the angle closed, or as in the patient described above, an adverse drug reaction driving the acute angle closure glaucoma).[5]

Patients presenting with acute angle closure glaucoma can often have decreased visual acuity and describe seeing halos around lights that are associated with severe headache, eye pain, nausea, and vomiting. On physical exam, these patients commonly have conjunctival injection, corneal clouding, a shallow anterior chamber, and a mid-dilated pupil that reacts poorly to light. Dilation of the eye will cause worsening symptoms, whether it is physiologic from poorly lit areas or pharmacologic with dilating drops. Typically, acute angle glaucoma affects one eye and rarely occurs bilaterally.[5]

This patient had a recent addition of

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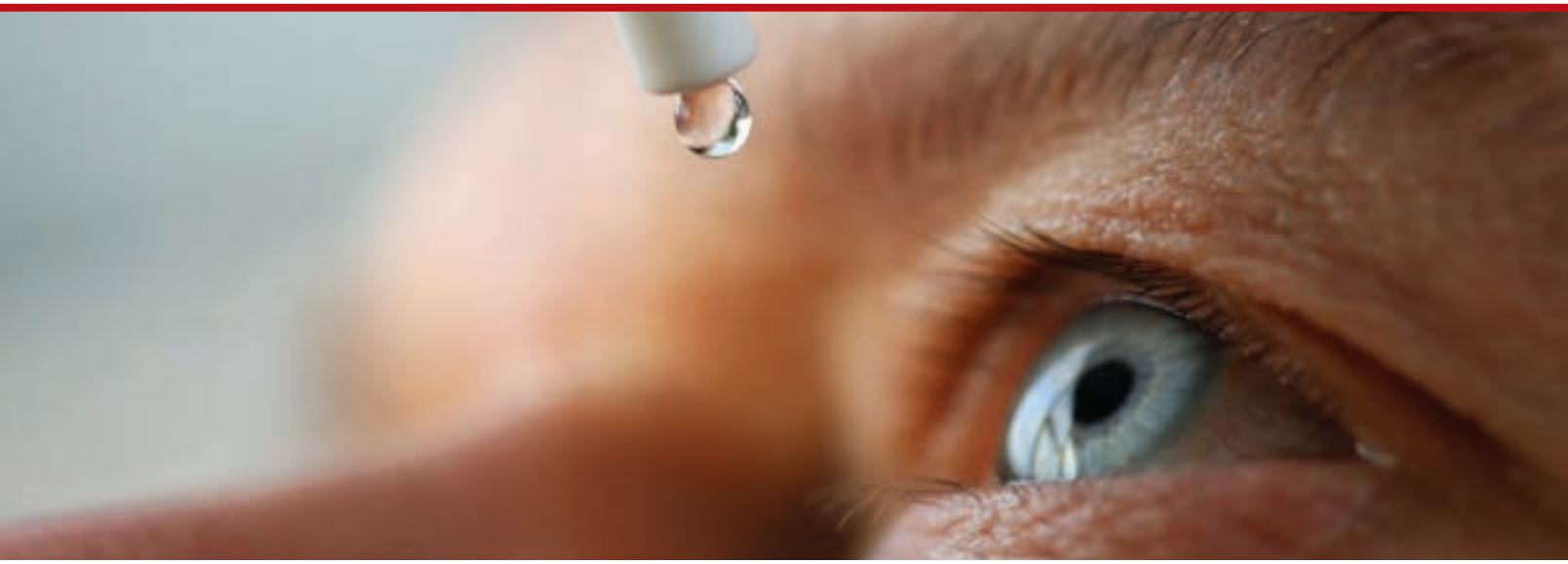

Managing acute angle closure glaucoma in the ED involves decreasing intraocular pressure caused by excess aqueous humor. Once the diagnosis is made, steps can be initiated to rapidly decrease the pressure inside the eye and preserve vision. This includes a combination of patient positioning, topical eye drops, and systemic medications.

topiramate to his medication regimen as a migraine prophylaxis medication. Topiramate, more commonly known as Topamax, is a sulfa derivative used traditionally as an antiepileptic or for migraine prophylaxis. Recently, it has also become a popular medication to treat alcohol and drug dependence, eating disorders, PTSD, depression, and weight loss. Its mechanisms of action are as follows: It blocks voltage dependent sodium channels, antagonizes an NMDA glutamate receptor, enhances the effects of GABA at its nonbenzodiazepine receptors, and weakly inhibits carbonic anhydrase in the central nervous system.[4,8]

Common adverse effects of topiramate include metabolic acidosis, weight loss, cognitive impairment, paresthesia, fatigue, depression, and mood lability. Rare complications include kidney stones, acute myopia, and secondary acute angle closure glaucoma as seen in our patient.[6]

The mechanism by which topiramate causes acute glaucoma is thought to be through forward displacement of the lens-iris diaphragm from ciliochoroidal effusion, which causes anterior chamber shallowing and eventually a backup of aqueous humor.[6] This causes an increase in IOP, which results in the pain and physical exam findings as described above.[2,3]

Other more common precipitating factors of acute angle closure glaucoma include dimly lit areas that cause physiological mydriasis and other drugs such as antihistamines, antidepressants, bronchodilators, cough/cold and flu medications, and other anticonvulsants.[5]

MANAGEMENT AND CASE CONCLUSION

Management for acute angle closure glaucoma in the ED revolves around decreasing the IOP caused by excess aqueous humor.[5] Once the diagnosis is made by history and physical exam, steps can be initiated to rapidly decrease the pressure inside the eye and preserve vision. This includes a combination of patient positioning, topical eye drops, and systemic medications.

The patient should be positioned to lie flat on their back, which helps to displace the lens posteriorly. Administration of intravenous acetazolamide (500 mg) can further reduce the production of aqueous humor. Also, 1 to 2 grams of IV mannitol can be considered to reduce the volume of aqueous humor. Topical eye drops include a beta blocker such as timolol, an alpha 2 agonist such as apraclonidine or brimonidine, prostaglandin analogues such as latanoprost, and pupillary constricting agents like pilocarpine, repeated every 30-60 minutes until the pressure has decreased. In addition to the above measures, it is important to treat pain, nausea, and vomiting, as these symptoms can further increase IOP.[5,7]

The previously mentioned treatment should be initiated by the ED or the ophthalmologist, if at a facility with in-house ophthalmology. Further management depends on the type of glaucoma. For primary acute angle closure glaucoma, definitive management consists of peripheral iridectomy performed by an ophthalmologist. This allows for fluid to flow between

References available online.

the anterior and posterior chambers without increasing the IOP. In contrast, secondary acute angle closure glaucoma does not require this procedure.[9] Secondary acute angle closure glaucoma should be treated symptomatically with the medications above as well as cessation of the inciting medication or removal of the secondary cause of glaucoma.[1]

Prognosis for this condition is dependent on how long the IOP remains elevated. The longer it goes without treatment, the more likely the patient will have irreversible damage to the optic nerve, resulting in permanent vision loss.[7]

Our patient began treatment and returned for follow-up in three days with ophthalmology. At that time, his IOP had normalized to OD 14 mmHg and OS 9 mmHg. He reported improvement in his vision as well as resolution of his pain.

TAKE-HOME POINTS

- Screen for ocular pathology when assessing a patient with headache complaints.
- With ocular complaints, the five vital signs of the eye can rapidly assist in identifying the correct diagnosis: (1) visual acuity; (2) ocular pressure; (3) visual fields; (4) pupil examination; and (5) extraocular motility.
- Acute angle closure glaucoma presents as a painful red eye often associated with headache, nausea, and vomiting. The eye will appear injected, with a cloudy cornea and mid-dilated mildly reactive pupil. The anterior chamber will appear shallow, and there will be an increased intraocular pressure on tonometry.
- Glaucoma treatment should be initiated in the ED by decreasing intraocular pressure with systemic IV acetazolamide and topical eye drops including a beta blocker, alpha agonist, prostaglandin analog, and topical pilocarpine.
- Topiramate is a sulfa-derived medication. One of its rarer adverse reactions is bilateral secondary acute angle closure glaucoma.
- Don't forget to ask about recent medication changes, especially in patients with unique clinical presentations. ✶

References available online.

How To Make Your Survey Count

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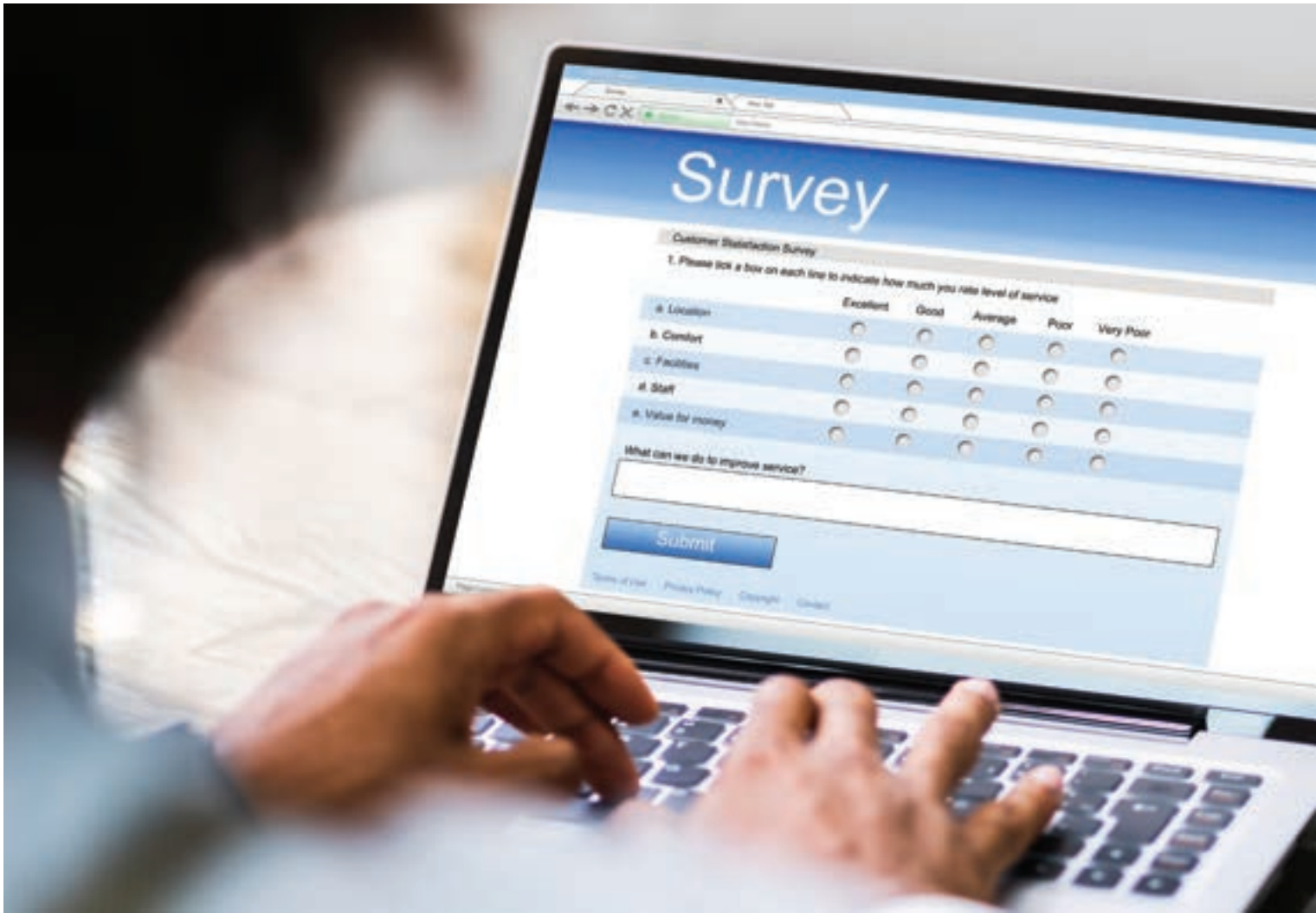
RESEARCH DURING RESIDENCY OFTEN SEEMS LIKE A DAUNTING TASK, given the limited amount of time we have to accomplish it. For this reason, many of us choose to create surveys. The method used to conduct data collection, as well as the quality of questions asked, will determine the accuracy and reliability of the data.[1,2] This article aims to give you practical guidance on ways to improve the quality of the data and make your research more fruitful.

WHEN TO USE A SURVEY

While a survey seems like an obvious choice for research during residency, it is not applicable to every situation. Surveys are best utilized when researching human phenomena such as emotions or opinions.[1] Surveys can also be used when the data you want to analyze is not directly observable or available in documents.[1] Additionally, surveys may be used when directly obtaining the data is not practical.[1]



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TYPE OF SURVEY TO USE

The three primary types of data-gathering methods described here are phone surveys, face-to-face interviews, and written surveys.

Phone surveys have a higher completion rate and some inherent quality control because the interviewer can clarify survey questions that the responder does not understand.[3]

Face-to-face interviews are best for complex topics.4 They have the highest response rate but are also the most expensive.[3] With both phone surveys and face-to-face interviews, data might be less reliable because participants may be biased to choose a more socially desirable response.[3]

Written surveys have lower response rates but are the easiest to standardize because they avoid differences between interviewing techniques.[4] Written surveys are also less expensive and more convenient because they do not require an interviewer.

WRITING QUESTIONS

Once you have decided that a survey is the best method to obtain the data you're looking for, and you have determined which type of survey to use, the next step is to write the questions.

When you start writing questions, it is important to keep the answering process in mind. Typically, responders first interpret the question and try to determine what is being asked. Responders then search their memory for relevant information and try to fit it into a single response. Lastly, if response choices are offered, they try to find their response among these options.[5] The less effort required by the respondent, the more accurate the answers will be.[6] Furthermore, accuracy of responses decreases with increased task difficulty, the respondent's ability to answer a specific question, and lower motivation level.

Response choice order has important implications for the way respondents answer. For instance, in written surveys, respondents are more likely to choose the first choices, whereas in verbal surveys, they are more likely to choose the last option.[6] One way to try to combat the bias of either choosing the first or the last response based on the survey technique is to change the order of the responses when giving individuals the same survey.[2,5]

Consider using "filter" questions, so that if a question does not pertain to the respondent, they can skip to the next question. This way, respondents won't answer questions that don't pertain to them; if they did, it could affect the accuracy of your data.[5]

Surveys that include "I don't know" as a possible response may limit the reliability of the data. While this option encourages people who "do not know" to avoid choosing a random answer and skewing the data, it also is an easier choice for less motivated respondents. Furthermore, when an "I don't know"

References available online.

answer choice is added, data quality does not improve.[5] Therefore, it may be best to avoid this option altogether.

Phone surveys should be limited to 15 minutes or less to avoid participant dropout. Question-and-answer choices must be simple and clear because the responder does not have the opportunity to read the questions.[3]

QUESTION ORDER

The order of questions can help increase participant engagement and accuracy of responses.

Initial questions should be strongly tied to the survey's overall topic and purpose, thereby piquing participant interest.[5,6] Start with easy questions to help build rapport, and then transition to more sensitive or difficult questions.[2,4,5] To decrease respondents' cognitive burden, organize your survey so that questions belonging to the same category are grouped together. Also group together "how often" and "how much" types of questions, especially within categories.[5,7]

Consider respondents' motivation for the survey. Participants are more likely to be motivated if they benefit from survey results, have a desire for self-expression, perceive the survey as an intellectual challenge, and/or wish to help the survey sponsor. Respondents may be less motivated if the survey is required. Other participants may be motivated at the start but become tired or disinterested and therefore less motivated as they continue the survey.[5] If respondents are less motivated, their most accurate answers will be at the beginning of a survey, so it might be best to place less pertinent demographic questions at the end.[5,6] On the other hand, highly motivated participants may actually increase the accuracy of their responses with more questions.[6]

TESTING YOUR SURVEY

Prior to administering a survey for research data collection, it is important to review the survey for comprehensibility, participant burden, and validity.[3] This can be accomplished by having an expert in the field you are researching review your survey to identify confusing questions or answers and determine which, if any, terms or phrases require definitions or explanations.[7] Software programs are also available to evaluate questionnaires and surveys for common errors.[5]

The next step would be to test your survey on a small sample of the relevant population under conditions that are close to or identical to those of the actual survey.5 For verbal surveys, consider cognitive interviewing where the respondent is asked to think out loud about how they would answer the question, and verify that the respondent's comprehension of the question matches your intention.[5,7] After the survey has been completed, debrief with respondents to evaluate the time it took them to complete the survey, identify questions that arose during completion, and note other ways to improve the survey.[7]

RECRUITING SUBJECTS

Recruiting subjects for surveys can be difficult. It is important to tell potential subjects why you are doing the survey, why they were chosen to be part of the study, what is expected of them, and what they will gain from the experience.[4]

INCREASING RESPONSE RATES

To increase the response rate, make sure that instructions are clear and that the survey is as brief as possible to obtain the appropriate meaningful data.[3] Choosing a topic that is highly relevant to your sample population will increase motivation.[3] If ethically and financially feasible, consider giving incentives and rewards for participation.[3,7] Lastly, consider sending out repeated reminders for non-responders. [3,4]

DATA INTERPRETATION

Because the average response of online surveys is 20-30%, it is important to consider how non-responders will affect the results (validity, generalizability, etc).[3,4,9] To reduce or eliminate this non-responder bias, consider trying to sample a small group of non-responders using vigorous efforts (for example, messaging or calling them), to see if their responses are different from the responder results.[4] Take into account the demographics of the non-responder population compared to the responder population to determine if that could potentially affect your data from the survey versus the true population data.[4]

Finally, when reporting and presenting the data obtained in a survey, be certain to include details related to the persons asked to participate in the study, including how they were contacted, response rate, and any strategies used to increase response rate. A description of the survey and a copy of the actual survey should be included, if applicable and if space allows.[3,9]

Surveys can be great research tools to obtain data when carefully designed and correctly administered. When writing a survey, consider the best method to administer it, take into account participant motivation, and develop questions that maximize participation and generate valid and meaningful responses. ✶

Tips for writing survey questions:

- Ask simple, concise questions[2,3]
- Use wording that will be interpreted the same way among respondents[7]
- Avoid double-barreled questions - asking two questions at once[2,3,7,8]
- Avoid double negatives[6]
- Avoid negatives in the question or answer choice(s)[2,7]
- Avoid technical jargon and uncommon words[3]
- Avoid vague words and ambiguous meanings[3,5]
- Avoid leading questions that insinuate a "right" or socially desirable answer[2,5,7]
- Avoid true/false, agree/disagree, yes/no answers [because people are more likely to choose true, agree, yes][5]
- Use caution with "check all that apply" questions since they often yield less reliable answers[8]

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Real-Time Ultrasonography in Malignant Pericardial Effusion with Tamponade and Concomitant Pulmonary Embolism

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Ultrasound is a dynamic and innovative tool used to perform rapid evaluation and diagnosis in the emergency department. It is instrumental in the guidance of clinical decision-making, enabling real-time assessment and feedback during the resuscitation of a critically ill patient.

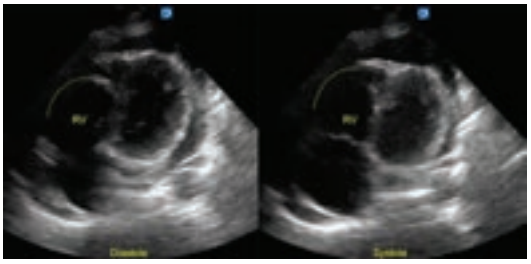
This case report highlights the critical role of ultrasound in the early detection of right heart strain from a pulmonary embolism and concomitant pericardial effusion with tamponade. Additionally, this case highlights how diagnostic ultrasound in real time facilitates optimal medical management in highly complex cases with a fragile hemodynamic state.

Pulmonary embolism and cardiac tamponade are already serious conditions that can precipitate acute circulatory failure in even otherwise healthy patients. A hemodynamically significant pulmonary embolism can cause a substantial increase in pulmonary vascular pressure, resulting in pressure overload and dilation of the relatively thin right ventricle. Additionally, it results in an increase of physiologic dead space and a downstream reduction of left ventricular preload, which leads to the characteristic signs of tachycardia, hypotension, and hypoxia seen in our patient.

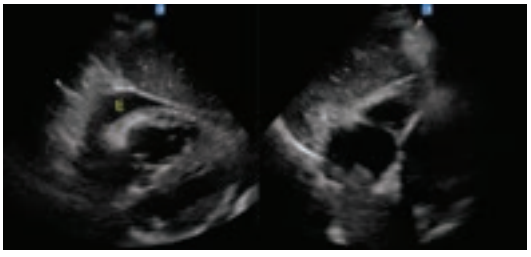
A sizable pericardial effusion of any etiology can precipitate acute cardiac tamponade once the pericardial pressures exceed that of the right ventricle, leading to the end-diastolic collapse that disrupts venous return of blood to the heart.

Simultaneous occurrence of both conditions further complicate medical management, as the elevated right ventricular pressures from the pulmonary embolism can protect it from end-diastolic collapse due to tamponade.

Although formal contrast CT imaging is the diagnostic for both conditions, it is not readily feasible to perform, especially in a patient who is too unstable for transport. Real-time sonography yielded a diagnosis within minutes of this patient's arrival, allowing guidance of appropriate resuscitation efforts, which rendered this otherwise critical patient stable enough for definitive evaluation.



Effusion



Pericardiocentesis

In our case report, we were able to quickly determine right heart strain with McConnell's sign and pericardial effusion with tamponade physiology using bedside ultrasound. With diagnostic imaging, it revealed the aforementioned diagnoses along with a right atrial thrombus, bilateral pneumonia, and a right-sided pleural effusion.

Additionally, this case highlighted the procedural utility of bedside ultrasound, permitting the use of ultrasound-guided pericardiocentesis compared to the traditional landmark-guided technique, which is susceptible to considerable error due to variations in patient anatomy.

CASE REPORT

A 65-year-old Vietnamese male presented to the emergency department with shortness of breath, leg edema, productive cough, and hemoptysis. His past medical history was significant for paroxysmal atrial fibrillation on diltiazem and digoxin, Stage IV lung adenocarcinoma of the left upper lobe on chemo and radiation therapy, and chronic hypoxemic respiratory failure on 2L home oxygen. He was not on any anticoagulant medications. When he arrived in the ED, the patient was afebrile, tachycardic at 106 bpm, normotensive at 114/66 mmHg, tachypneic at 20 per minute, and requiring 4L NC, tripoding in the EMS gurney.

The patient was immediately placed into a room and an initial bedside cardiac ultrasound revealed right heart strain, McConnell's sign, and moderate pericardial effusion with tamponade physiology.

Upon verifying the patient's code status and desire for full treatment, a Pulmonary Embolism Response Team (PERT) alert was activated. Critical care, interventional radiology, and cardiothoracic surgery services were notified as the patient underwent diagnostic labs, chest X-ray, EKG, and

References available online.

CT angiography of the chest.

The CT angiogram confirmed the presence of a large pulmonary embolism with RV strain, in addition to a pericardial effusion, a right-sided pleural effusion, a large thrombus in the right atrium, and bilateral pneumonia.

The patient was given slow hydration of fluids and then placed on bilevel positive airway pressure (BIPAP). Meanwhile, an extensive discussion took place among ICU, IR, CT surgery, and the emergency medicine attending regarding the patient's management. Due to his terminal cancer, he was a poor candidate for surgical intervention. The agreed-upon plan of care included an IV heparin drip for the pulmonary embolism, and thoracentesis and pericardiocentesis for the aforementioned effusions.

The thoracentesis was performed first to optimize the patient's ventilation and oxygenation status. After 1.90L of fluid was drained from the right pleural space, the patient underwent conscious sedation with IV midazolam, etomidate, and succinylcholine.

Ultrasound-guided pericardiocentesis was performed via in-line technique, using a phased-array probe in the subxiphoid view to visualize the pericardiocentesis needle in real-time as it advanced into the pericardial space. Then, 300cc of serosanguinous fluid were drained from the pericardium, yielding significant relief of the patient's symptoms.

The patient was subsequently placed on a heparin drip for thrombolysis for the pulmonary embolism and was admitted to the ICU.

During the course of his hospital stay, the patient conferred with his family and made the decision to transition to hospice care. He was discharged three days later to be home with his family.

DISCUSSION

Concomitant pericardial effusion with tamponade, pleural effusion, and pulmonary embolism are common in malignancy, which usually yields a poor prognosis. In the setting of malignant pericardial effusion with tamponade physiology and concomitant pulmonary embolism, management can be difficult. These two pathologies also seemingly work hand-in-hand due to the right ventricular pressure delaying cardiac tamponade.[1]

Although somewhat stable, the patient did not meet indications for pericardiotomy; however, a pericardiocentesis was indicated in order to alleviate stress around the heart and for heparin infusion. One study showed that there was no change in the overall survival of patients with a malignant pericardial effusion undergoing pericardiocentesis or pericardiotomy.[2]

Most emergent pericardiocenteses are performed through landmarks, but this case provided a unique learning opportunity in using ultrasound guidance and in-line technique. Procedures involving ultrasound guidance help reduce errors when compared to landmark-based techniques.[3] Using in-line technique enabled us to visualize the needle and prevent any complications, such as a perforation of the ventricular wall. After 300cc of serosanguinous fluid were removed, the patient did symptomatically improve, and this enabled further medical treatment for his other complications.

CONCLUSION

In the setting of a malignant pericardial effusion with tamponade and pulmonary embolism, medical management can be difficult due the need for heparinization as well as pericardiocentesis. In a peri-stable patient, the use of ultrasound-guided pericardiocentesis enabled us to prevent adverse outcomes and complications while providing symptomatic and therapeutic relief to the patient. It also allowed further management of the pulmonary embolism through heparinization. This case report highlights a fine balance of medical management as well as the use of ultrasound guidance in ED procedures that were previously performed blind. ✶

References available online.

ST Elevation Myocardial Infarction in a Biventricular Paced Rhythm

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Ventricular pacemakers typically cause a delay in repolarization of the ventricles; this in turn can obscure the usual ST changes seen in an acute myocardial infarction. Left bundle branch blocks (LBBBs) are commonly seen in ventricular paced rhythms and require further evaluation of acute ischemic events using the widely recognized Sgarbossa criteria.

We present a patient with a biventricular pacemaker and chest pains. His EKG findings show ST segment elevations in anterior and lateral leads, concerning for an anterolateral STEMI.

CASE

Our patient, a 93-year-old male with a complex cardiac history including complete heart block with biventricular pacemaker and atrial fibrillation on clopidogrel, presented to a community emergency department with symptoms of constant left-sided non radiating chest pressure that awakened the patient from sleep. He found himself in cold sweats at that time, prompting emergency department evaluation.

On physical exam, his vital signs were significant for hypertension of 148/100, otherwise unremarkable. There were no murmurs, asymmetric pulses, chest wall tenderness, or crepitus. The initial EKG was concerning for a ventricularly paced rhythm with a rate of 70 bpm and ST elevations in lateral and anterior leads (**Image 1**).

These were new changes when compared to the last documented EKG. The cardiology team was contacted prior to activating the cath lab, as the patient had a biventricular pacemaker. Upon discussing the patient's case with the interventional cardiologist, the decision was made to activate the cath lab. The patient was given aspirin and ticagrelor and transferred to Akron City for heart catheterization.

The patient was found to have total occlusion of the mid left anterior descending artery, for which he received balloon angioplasty and PCI (**Images 2 and 3**).

DISCUSSION

Biventricular pacemakers are devices that stimulate both the right and left ventricle simultaneously, which can result in a range of abnormal baseline EKG morphology related to variations in impulse timing and lead placement. As such, it can be challenging to diagnose coronary-related ischemia in these patients.

There is limited literature on the sensitive and specific EKG changes for myocardial infarction in these patients.[3] However, depending on the location, some pacemakers can create left bundle branch morphology, allowing for the well-known and reliable Sgarbossa criteria and the modified Sgarbossa criteria to play a role in diagnostic evaluation. Several studies have validated both the Sgarbossa and modified Sgarbossa criteria in patients with RV pacemakers, and this may be the closest criteria that can be useful in evaluating patients with ventricularly paced rhythms.[4]

To review, the Sgarbossa criteria refers to:

1) ST-segment elevation >1 mm in the same direction as the QRS complex (concordant) in any lead
2) ST-segment depression >1 mm in the same direction as the QRS complex (concordant) in at least one chest lead (i.e., V1 to V6).
3) ST-segment elevation >5 mm in the opposite direction as the QRS complex (discordant)[1]

The modified Sgarbossa criteria has the same first and second rule, but the third is replaced by:

3) ST-elevation (mm) / S-wave amplitude (mm) ratio >0.25 (a measure of discordance) from the lead that generates the greatest ST-elevation/S-wave ratio[2]

Of these criteria, literature suggests only the third criteria of the original Sgarbossa (ST-segment elevation >5 mm discordant with the QRS complex) was highly specific and statistically significant in ventricular paced rhythms.[6,7,8]

Patients with ventricularly paced rhythms are at higher risk for poor outcomes due to the difficulty in diagnosing ischemia in this population. In addition, patients can have atypical presentations such as the absence of chest pain. Atypical presentations in conjunction with no current highly sensitive or specific EKG criteria for ischemia can lead to delayed intervention and overall greater morbidity and mortality in ventricularly paced patients,[5] which highlights the relevance of judicious evaluation by ED providers. The Sgarbossa criteria can help more timely management in ventricularly paced patients and should not be limited to those with LBBB only.

The patient discussed in the case above had concordant ST-segment elevation of >1 mm in 1, aVL, V3 - V6.

References available online.

Despite placement of a biventricular pacemaker, the patient had classic ST-elevation in contiguous leads warning of ischemia, a highly unusual finding in the setting of a paced rhythm.

Overall, more studies need to be done to create sensitive and specific criteria for coronary-related ischemia in patients with biventricular pacemakers, but high clinical suspicion, changes in EKG, and early recognition can help with timely intervention for appropriate care.

TAKE-HOME POINTS

- There is neither very good data nor scores to diagnose a STEMI from a paced ECG considering the sensitivity and specificity of Sgarbossa.
- Classic ST elevation pattern can be seen in ventricularly paced rhythms.
- Clinical suspicion may be more telling than the EKG in patients with biventricular paced rhythms. ✶

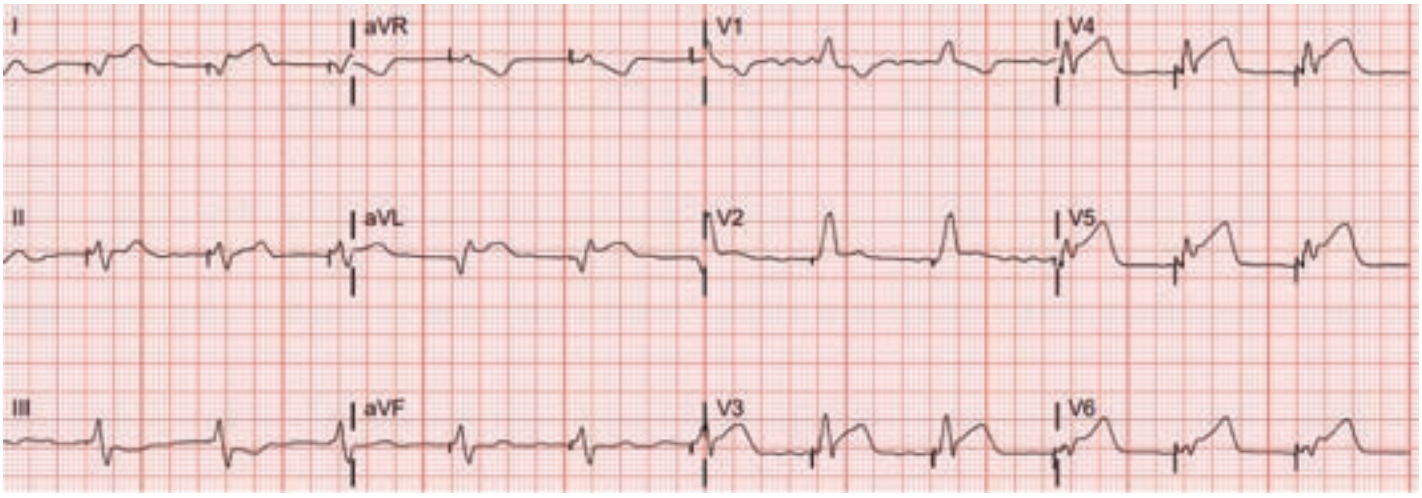


IMAGE 1: EKG

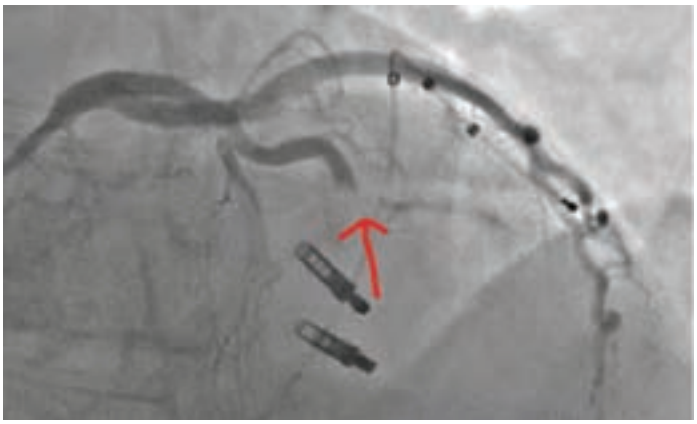


IMAGE 2: Pre-balloon angioplasty and PCI

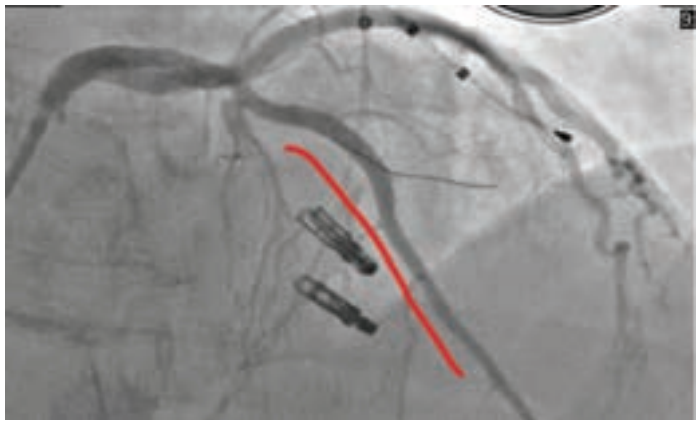
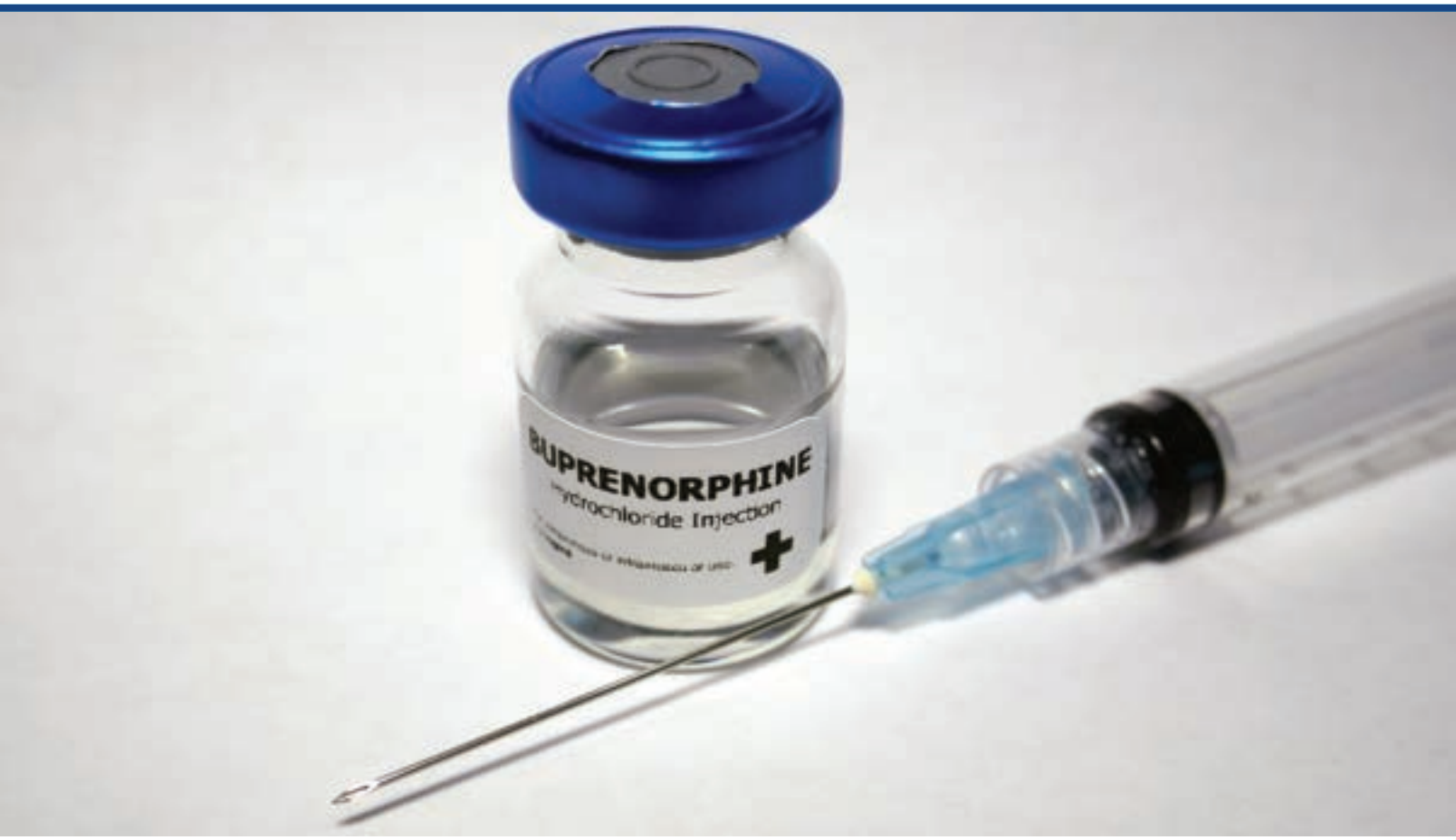


IMAGE 3: Post-balloon angioplasty and PCI

References available online.



BUPRENORPHINE INDUCTION: Where We Are and Where We're Headed

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Buprenorphine, a partial μ opioid receptor agonist, has been approved by the Food and Drug Administration for outpatient opioid dependence pharmacotherapy since 2002.[1] Multiple settings have proven to be safe for buprenorphine, with evolving literature on efficacy in these variable practices. Two well-researched settings for induction include the unobserved induction and induction in the emergency department.

Since the early 2000s, a safe alternative to physician-observed induction of buprenorphine has been off-site, "home," or unobserved induction. In a home induction, a patient is evaluated prior to beginning treatment. Eligible patients are often still using opioids and not experiencing active withdrawal. If eligible, the patient receives a buprenorphine prescription and self-administers the first dose of buprenorphine unobserved. Recent descriptions of home induction approaches prescribed in two Boston practices (as well as many other studies on unobserved induction) revealed the practice to be safe and found no differences between groups in longitudinal outcomes.[2,3]

A 2007 Massachusetts survey indicated that 42% of physicians had adopted home induction as regular practice.[4] A 2008 study of more than 100 patients undergoing home induction found that a home buprenorphine induction protocol resulted in no observed serious adverse events, few reported induction complications, and treatment retention rates consistent with previous studies.[5]

Suboxone induction is also undergone frequently with less forward-planning than unobserved induction. Many of these inductions happen in the emergency department. Since the publication of a landmark trial demonstrating the efficacy of buprenorphine induction for ED patients with opioid use disorders (OUDs),

References available online.

emergency departments across the United States have developed protocols for facilitating buprenorphine induction.[6] Studies have reported that induction of buprenorphine in the ED, when compared to control, resulted in higher engagement in addiction treatment. Despite this, significant gaps in access to treatment remain. One analysis suggested that only one in three patients who screen positive for OUD actually receive treatment for OUD.[7]

A third setting, undergoing active research, is emerging as a potential solution to a subset of patients who would not otherwise access buprenorphine. Immediate initiation of buprenorphine after opioid overdose reversal with naloxone in the field by trained emergency medical technicians (EMT) was first reported in New Jersey. In the first U.S. study of its kind, investigators did not report any complications with subsequent induction of buprenorphine once the patients reached the ED. This novel induction strategy suggests that immediate administration of buprenorphine after naloxone is safe and valuable for emergency medical service clinicians.[8]

The decision on which patients are appropriate for buprenorphine induction is well-researched. A 2020 review of 14 peer-reviewed manuscripts and 11 scientific abstracts related to OUD treatment in the emergency department reported variable inclusion and exclusion criteria.

This review found that inclusion criteria have most often been a diagnosis of OUD age over 18, a positive urine test for opioids, recent or multiple visits related to overdose, intoxication, or admission to an ED observation unit.

Exclusion criteria were pregnancy, current enrollment in an OUD treatment program, abuse of benzodiazepines or alcohol, liver disease, critically ill patients, active suicidality, police custody, or an inability to communicate due to dementia or psychosis.

While ED studies have often excluded pregnant patients, research outside of emergency settings has shown that buprenorphine is safe and effective for pregnant patients.[9]

In deciding dosing for initial induction, most studies report an initial dose ranging from 2 mg to 8 mg with consistent maximum doses reported around 16 mg and as high as 32 mg.[9]

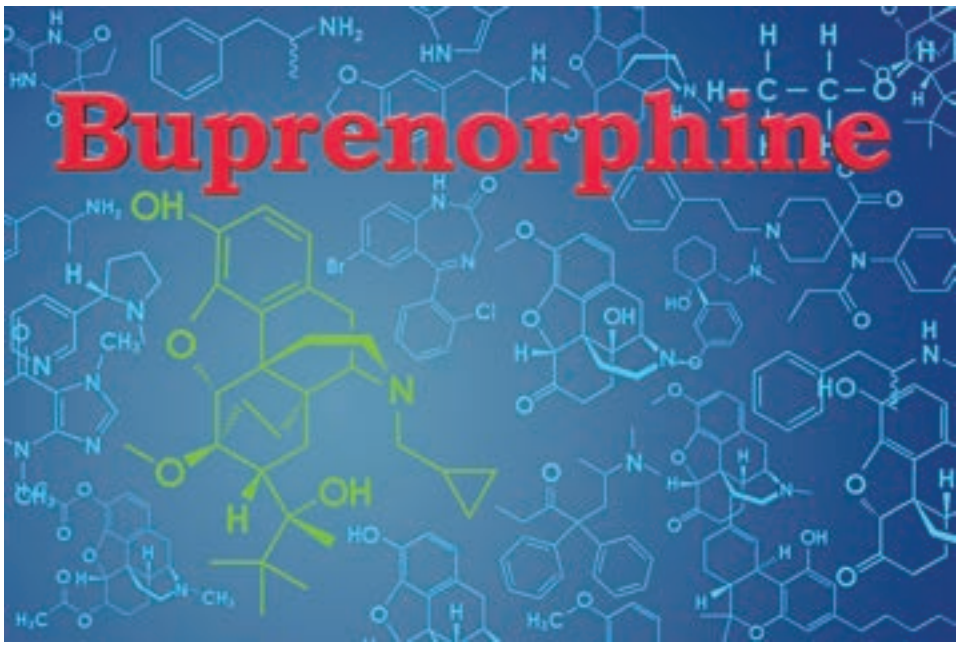
There is also extensive research offering general guidance for dosing and timing of induction. Most protocols use the Clinical Opioid Withdrawal Scale (COWS) to assess withdrawal severity. The COWS is a clinician-administered tool for assessing a patient's level of opioid withdrawal by rating 11 commonly seen opioid withdrawal symptoms.[12]

Clinical assessment of withdrawal is imperative for decreasing the risk of precipitated withdrawal. Precipitated withdrawal is a particularly worrisome adverse effect. This occurs when a patient undergoes induction too soon after use of another opioid agonist. The patient experiences an immediate withdrawal syndrome due to displacement of that agonist by buprenorphine, a partial agonist with high affinity.

Precipitated withdrawal can largely be avoided by only commencing induction in a patient with objective evidence of opioid withdrawal. There are no strict cutoff criteria based on COWS score, but studies in the aforementioned 2020 review used COWS score cutoffs as low as 6 and as high as 13.[9]

In conclusion, buprenorphine has been shown to be a safe and effective medication in multiple settings with extensive research on both unobserved induction and physician-observed induction. Emerging evidence suggests that EMT-observed induction of buprenorphine may represent a third setting that could allow another subset of patients to access treatment. In planning how this might come to fruition, numerous studies exist to help guide decisions on patients to include/exclude, timing of induction, and dosing. ✶

Buprenorphine has been shown to be a safe and effective medication in multiple settings with extensive research on both unobserved induction and physician-observed induction. Emerging evidence suggests that EMT-observed induction of buprenorphine may represent a third setting that could allow another subset of patients to access treatment.



References available online.

BRAO and CRAO: Quick Diagnosis, Management Lead to Better Outcomes in Retinal Artery Occlusion

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A 55-year-old female with a history of paroxysmal atrial fibrillation with multiple failed ablations, hypertension, chronic back pain, and a 100-pack-year smoking history presented to the ED via Mobile Stroke Unit (MSU) with a chief complaint of "blurry vision."

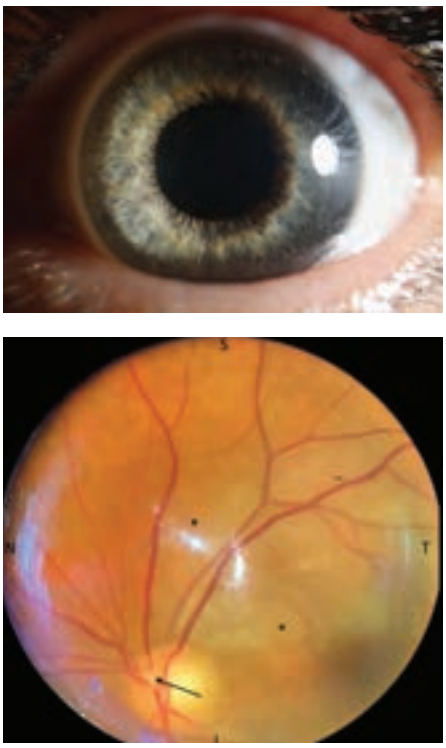


FIGURE 1. The patient's left eye as seen on the fundoscopic exam, with nasal (N), temporal (T), superior (S), and inferior (I) orientation. It exhibits a cup-to-disc ratio of 0.4, normal optic disc nerve appearance without disc edema, plaque at proximal superotemporal arterial arcade (arrow) with corresponding area of wedge-shaped retinal edema (*), normal vessels, and normal periphery.

The patient reported discontinuing her anticoagulation regimen (aspirin 81 mg daily) because she was receiving epidural injections for musculoskeletal neck pain. The patient reported experiencing left-sided chest pain which radiated to her left arm during an argument with her daughter 36 hours previously. The morning after, she awoke to circumferential left-arm numbness from her shoulder to her fingers accompanied by painless "blurry" vision that improved when she closed her left eye. These symptoms persisted for 24 hours, at which point she presented to the ED.

Physical examination upon her arrival was notable for a normal right eye, and a left eye with 20/70 visual acuity and an inferior nasal quadrantanopia. She also reported paresthesias in the V1, V2, and V3 branches of the left trigeminal nerve. Intraocular pressures were normal bilaterally.

An ECG showed a normal sinus rhythm. Computed tomography (CT) and CT angiography of the brain and intracranial vessels were negative for acute bleeding, aneurysm, or large vessel occlusion. A bedside ultrasound of the left eye was performed and was also normal. An MRI of the brain was subsequently performed and was also negative for acute pathology.

A dilated eye exam revealed findings concerning for branch retinal artery occlusion (BRAO). It is suspected that this finding was due to cryptogenic stroke in the setting of paroxysmal atrial fibrillation while the patient was not on anticoagulation.

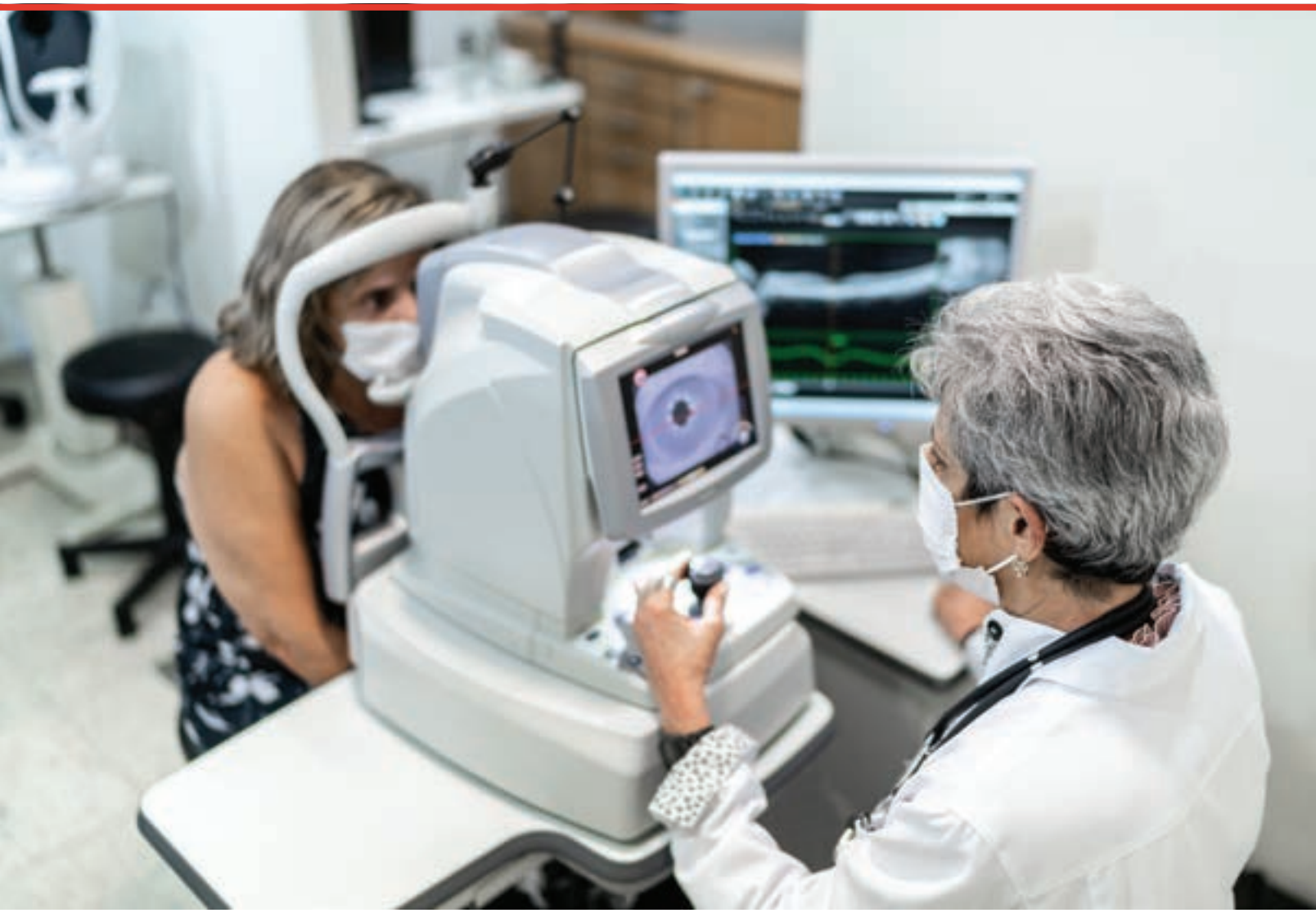
The differential diagnosis of acute, painless vision loss necessarily mandates consideration of both intracranial and intraocular etiologies. BRAO often causes superior or inferior quadrantanopia, which can also be associated with damage to Meyer's loop within the temporal horns of the brain, whereas central retinal artery occlusion (CRAO) is associated with damage to Baum's loop within the parietal lobe.

CRAO may also present with amaurosis fugax, a common presentation of multiple sclerosis. Retinal hemorrhage and detachment may also present similarly, and giant cell arteritis must also be considered in patients older than 50.

Loss of partial or whole perfusion of the retina from either BRAO or CRAO mandates quick recognition in order to prevent permanent vision loss. CRAO can compromise the entire arterial supply of the retina, whereas BRAO is more localized. Causes include thrombotic and embolic events as well as impaired venous drainage and external ocular trauma. Severity of visual loss depends upon the area affected, and permanent vision loss can occur in as few as 90 minutes.

Emboli associated with CRAO and BRAO are generally caused by embolized cholesterol fragments (Hollenhorst plaques), which appear as a yellow, fuzzy spot within the artery. Emboli can also originate from cholesterol thrombi found on native or mechanical heart valves, which appear white on fundoscopic exam. Obstruction of the central artery

References available online.



in CRAO often results in the appearance of diffuse retinal pallor and edema associated with a "cherry red spot" at the macula, whereas a wedge-shaped area of retinal pallor is pathognomonic for BRAO. As shown in **Figure 1**, a portion of the patient's superotemporal arcade was obstructed, causing an inferonasal visual field defect.

CRAO and BRAO are ocular emergencies mandating rapid diagnosis and management to prevent permanent loss of vision. Treatment options include ocular massage to attempt to relocate a lesion to a more peripheral area, and acetazolamide to reduce intraocular pressure via decreased aqueous humor production. Hyperbaric oxygen therapy has also shown promising results. While catheter-directed thrombolysis may be effective for CRAO, the smaller size and peripheral location of most BRAOs makes them less amenable to this therapy.

CRAO and BRAO are ocular emergencies mandating rapid diagnosis and management to prevent permanent loss of vision. Treatment options include ocular massage to attempt to relocate a lesion to a more peripheral area, and acetazolamide to reduce intraocular pressure via decreased aqueous humor production. Hyperbaric oxygen therapy has also shown promising results.

Our patient, unfortunately, presented on the third day of her visual symptoms, which limited treatment options. The patient was admitted to the cardiology service, with neurology making recommendations. During her admission, she noted symptomatic improvement in her vision. Serial troponins were negative; she remained in sinus rhythm. She was discharged on apixaban for paroxysmal atrial fibrillation. She declined placement of an internal loop recorder but agreed to have an MCOT cardiac monitor. At her four-week follow-up appointment, she continued to be in normal sinus rhythm while taking aspirin, statin, and apixaban. Unfortunately, she was unable to afford carvedilol, as she was spending $500 per month on prescriptions while waiting for insurance. ✶

References available online.

Addiction Medicine Certification Strengthens EM Career

Special Contribution from the American Society of Addiction Medicine (ASAM)

Soon after he became board certified in addiction medicine in 2020, Dr. Andrew King, an emergency physician, started working at an opioid treatment program on Wednesday mornings.


Dr. Andrew King

While he originally thought of this time as a professional break from the quick pace of the emergency department, he found an unexpected sense of satisfaction working at the methadone clinic. That satisfaction still endures today.

"It's my favorite day of the week," said Dr. King, who is also board certified in emergency medicine and medical toxicology. "It helps me gain a new and deeper perspective on patients, seeing them outside the hospital, getting more insight as to what their home life is like, their background, and the way they view the medical establishment overall. After multiple visits, you really get to know the patient as a person, which is not something that you get to do, per se, as an emergency physician."

Dr. King is an attending physician at Sinai Grace Hospital, Detroit Receiving Hospital, and the Michigan Poison and Drug Information Center, all in Detroit. At the latter, he also serves as a medical toxicologist.

His time at the clinic has helped him better treat patients with addiction in the emergency setting — an often high-stress environment where physicians see patients on their worst days.

"It really does help with having more empathy for patients and gives me the motivation to keep going and helping," he said. "When I see them, I feel energized, and any burnout from my shift evaporates."

Dr. King added that he wouldn't have gained this new, valuable perspective without becoming board certified in addiction medicine.

"It has 100 percent increased my satisfaction with emergency medicine," he said.

BENEFITS OF CERTIFICATION

Emergency medicine residents seeking to become certified in addiction medicine can do so through the American Board of Preventive Medicine (ABPM) or the American Osteopathic Association (AOA).

To become certified through the ABPM, applicants must have graduated with a medical degree, hold a current medical license, and have a current primary American Board of Medical Specialties (ABMS) board certification. Those who meet these initial qualifications can apply through two eligibility pathways: (1) time in practice, or (2) the successful completion of a 12-month Accreditation Council for Graduate Medical Education (ACGME) addiction medicine fellowship program.

To become certified through the AOA, applicants with an active license and a current primary AOA or ABMS member board certification can apply through two eligibility pathways: (1) clinical practice, or (2) completion of an AOA- or ACGME-accredited addiction medicine fellowship program. To qualify for the clinical practice pathway, one must have time in practice, hold an active American Board of Addiction Medicine (ABAM) certificate, or have completed an American College of Academic Addiction Medicine (ACAAM) fellowship within the five years prior to the application start date for that year.

Altogether, the certification process can take about a year. The ABPM and the AOA offer four- to five-month application windows during the year, with exams in the fall. Applicants are notified of their exam results in the first quarter of the following year.

There are many benefits to becoming certified in addiction medicine. Emergency physicians who do so add value to their employer and become thought leaders on addiction medicine.

The American Society of Addiction Medicine's (ASAM's) fellowship membership category (FASAM/DFASAM) gives recognition to and raises awareness of ASAM physician members who are board-certified addiction medicine specialists.

Each of these benefits helps advance the physician's career. Additionally, specialists tend to have greater earning potential. It's estimated that physicians with board certification earn 67 percent more than those without certification.

Some emergency physicians with addiction medicine certification also may gain access to federal loan repayment funds. Congress has appropriated funding for student loan repayment of up to $250,000 for physicians who become addiction medicine specialists and practice for six years in designated geographic areas.

Emergency physicians specializing in addiction medicine can earn continuing education credits through ASAM that meet maintenance-of-certification requirements for their licenses, their primary ABMS or AOA certifications, and their addiction medicine specialist requirements.

References available online.

MAKE A DIFFERENCE

Dr. King, now fellowship director of the Detroit Medical Center/Wayne State Medical Toxicology fellowship, received his addiction medicine certification through ABPM. He said it was a "challenging test" that was "fun to study for," and that the application process was more rigorous than he expected.

"After two attempts, I was eligible to sit for the board exam," he shared. "If you're going to apply, don't wait until the last minute and assume everything's going to be OK."

He added that today's emergency medicine residents are needed in the fight against addiction, especially when it comes to the ongoing opioid epidemic. Those who become certified in addiction medicine will be more equipped to change patients' lives.

"You can diagnose and pick up on maladaptive patterns, undisclosed motivations, and a person's logic for taking or not taking certain actions much more readily when you have a background in addiction and talk to patients with substance use disorder frequently," Dr. King said. "You can establish a connection with them that they know that you know. That is therapeutic almost immediately. You can see their sigh of relief. It's like, 'OK. I can talk about this with somebody.'"

Treating patients with addiction often poses multiple challenges in the emergency medicine setting, but the depth of knowledge that the certification process provides can help emergency physicians experience breakthroughs with these patients, making a lasting difference in their lives — even during their relatively quick visits to the emergency department.

"We see people with substance use disorders every day," Dr. King said of these challenges in treating patients. "And when you do it right, treat them with respect and dignity, you'll get unexpected hugs from patients that just make it all worthwhile." ✶

RESOURCES

To learn more about the need for physicians to become certified in addiction medicine, join National Addiction Treatment Week from Oct. 17-23 at treataddictionsavelives.org and on social media at #treatmentweek and #treatmentweek2022.

For information on certification, visit:

American Society of Addiction Medicine (ASAM)
https://www.asam.org

ASAM Guide to Addiction Medicine Certification
https://www.asam.org/education/addiction-medicine-certification

American Board of Preventive Medicine (ABPM)
https://www.theabpm.org/become-certified/subspecialties/addiction-medicine/

American Osteopathic Association (AOA)
https://certification.osteopathic.org/addiction-medicine/

American Board of Medical Specialties (ABMS)
https://www.abms.org

Accreditation Council for Graduate Medical Education (ACGME)
https://www.acgme.org

American Board of Addiction Medicine (ABAM)
https://www.abam.net

American College of Academic Addiction Medicine (ACAAM)
https://acaam.memberclicks.net

American Society of Addiction Medicine (ASAM) fellow designation (FASAM/DFASAM)
https://www.asam.org/membership/about-membership/designations/asam-fellow-designation

Detroit Medical Center/Wayne State Medical Toxicology fellowship
https://em.med.wayne.edu/toxicology-fellowship?c=mw28e

MI Cares
https://micaresed.org/?redir=1&utm_source=site&utm_medium=redir&utm_campaign=msu

References available online.

As You Near the End of Residency, Have a Roadmap for the Future

Priscilla Chao, MD | Rushabh Shah, MD, MBA | John Riggins, MD | Angela Cai, MD, MBA | And the EM class of 2021
SUNY Downstate/Kings County Hospital

You're in your final year of residency, and the light at the end of the residency tunnel is inching closer and closer. You've traveled long and far on a road with twists and turns and bumps and dips and countless exhilarating highs. Greener pastures await — YAY!

But wait — what's next?! Before your "YAY" turns into "YIKES," read on. We've got you covered with a list of essentials.

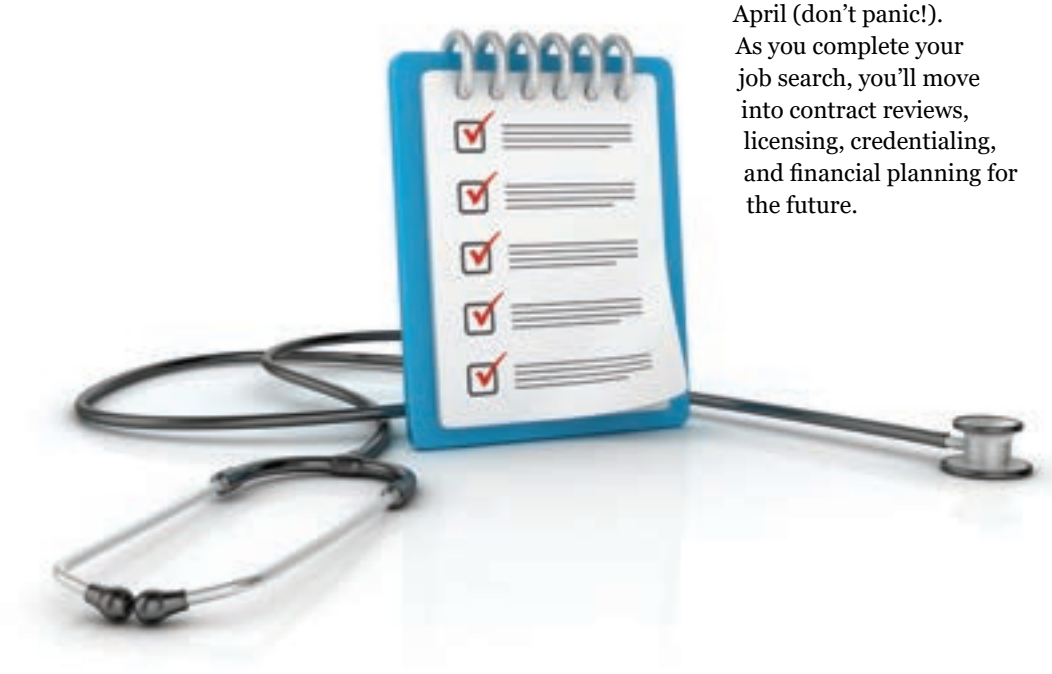
FINAL YEAR OF RESIDENCY: A CHECKLIST

It's fall — and we're getting full-swing into job search season. By now you should have polished your CV and cover letter, with each tailored to your target practice setting or fellowship. (Check out EMRA's career-planning resources, www.emra.org/career-planning, for more detailed CV and cover letter tips.)

Once you've prepared your materials, in September and October it's time to begin your job search by leveraging your network, starting with your faculty and alumni. The importance of your network cannot be stressed enough. Candidates are often identified through networks rather than official postings. It's always preferable to use mutual connections for introductions when you can, but don't be afraid to reach out directly to chairs and directors. If you're having trouble making a connection, don't forget — EMRA is here to help.

You can also find jobs through company or department recruitment events, job boards (such as www.emcareers.org), and job fairs. Additionally, ACEP Scientific Assembly and EMRA's Job and Fellowship Fair are great times to connect with employers. You can often undergo interviews on site during these events.

Job or fellowship offers may come in December through February. A few of you may end up on a later timeline, landing a job closer to April (don't panic!). As you complete your job search, you'll move into contract reviews, licensing, credentialing, and financial planning for the future.



LICENSING AND CREDENTIALING

Licensing and credentialing is an extremely long and complicated process. DO NOT DELAY! It can take months for all of these things to happen, and delays can accumulate and ultimately postpone your start date (and, consequently, your paycheck).

Here are some things to keep in mind:

- Apply for your ABEM certification, and make a plan to study for the written boards. EMRA membership will get you special rates for PEERprep and Rosh Review. Read more about the certification process here: www.emra.org/emresident/article/guide-to-abem-certification/
- Apply for state licensure AS SOON AS POSSIBLE. This can take months, and many other steps depend on having a license number. States vary in their use of online vs. paper applications (or a combination), so check into your state's requirements and submit your application. The Federation of State Medical Boards (FSMB) offers two online applications: the Federation Credentials Verification Service (FCVS), which verifies your medical credentials, and the Uniform Application for Licensure (UA) for basic demographics and training information. These online applications are accepted or required by most state medical boards. An advantage of using these online applications is their portability across state lines. The application for a state medical license requires several components, including your medical school transcripts/diploma, USMLE/COMLEX Official Transcripts (included in the FCVS), and fingerprinting.
- Obtain a U.S. Drug Enforcement Administration (DEA) number. You

References available online.

cannot get a DEA number without a state license, so go through the state licensure process first.

- Job onboarding. In addition to the paperwork required for obtaining your state license and DEA number, you'll face additional paperwork from your soon-to-be hospital employer. Be attentive to email communications; there will be many. Your employer will likely administer drug tests, inquire about malpractice coverage and clinical activity from residency, and require ACLS/ATLS/PALS/BLS recertification (not all employers require ATLS recertification after EM board certification).
- Inquire about independent contractor (1099) status. Does your employer require professional incorporation? If yes, you'll need to hire a lawyer to do this for you (which costs money, by the way).
- National Provider Identifier (NPI) number. Update your NPI taxonomy code from student to Emergency Medicine 207P00000X. This will require a medical license number. Get more information here: https://nppes.cms.hhs.gov/#/.
- Know your malpractice insurance coverage. Details should be spelled out in your employment contract. If you'll be employed full time by a large hospital or in an academic setting, you likely will not need additional coverage. However, if you'll be working at a small community hospital, or if you'll have part-time/contracted (i.e., 1099) employment status, then most likely you'll need additional coverage.

Remember to make time to finish up your program requirements, including Individualized Interactive Instruction (III), conference hours, scholarly activity, and procedure logging.

FINANCES AND HEALTH INSURANCE

That brings us to a final note about finances and health insurance coverage. As a post-residency emergency physician, you're embarking on a rewarding, but not cheap, journey. You will need several thousand dollars during this transition. In addition to the fees associated with licensing, you may need funds for moving expenses (e.g., shipping your car across the country or moving into an apartment in a state with a higher cost of living).

Ensure you're covered with adequate health insurance without gaps between residency and the start of your job. Consider extending your previous plan via COBRA. If your employer does not provide insurance, enroll in either your partner's health insurance plan or a Health Insurance Marketplace plan.

During this transition, it's also important to look into disability, life, and malpractice insurance options.

Here's a great EMRA*Cast episode about finances to get you started: www.emra.org/about-emra/publications/emra-cast/resident-finances/

We cannot stress enough the importance of creating, and sticking to, a budget. Consider refinancing your student loan. Prudent financial planning in the present will save you from headache and heartache in the future.

Congratulations on making it this far in your EM residency journey. Best of luck with your future in the specialty! We're glad you're joining the ranks. ✶

References available online.

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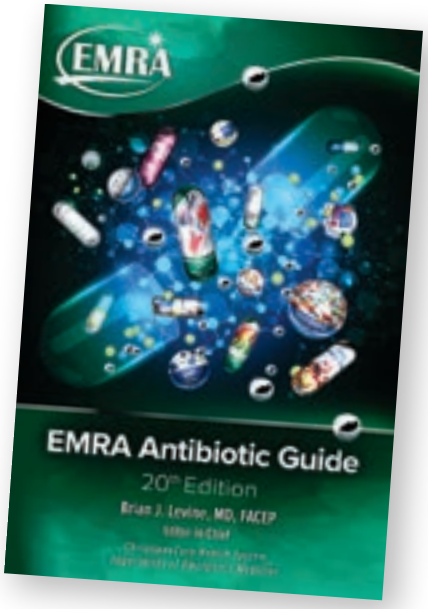

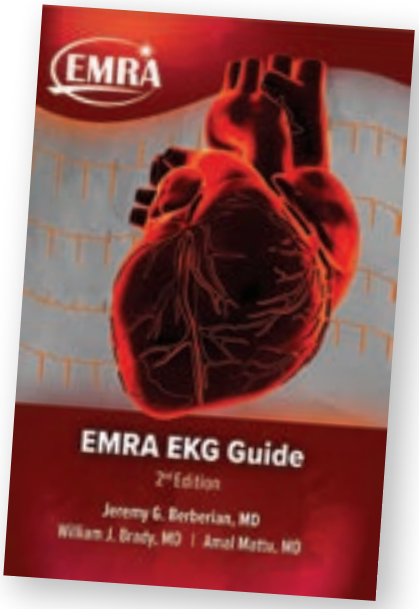

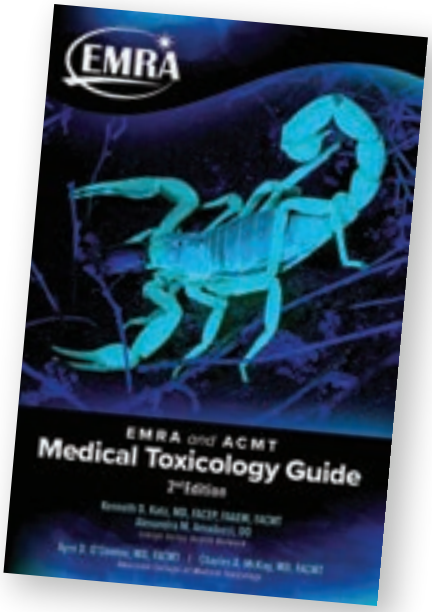

ECG Challenge

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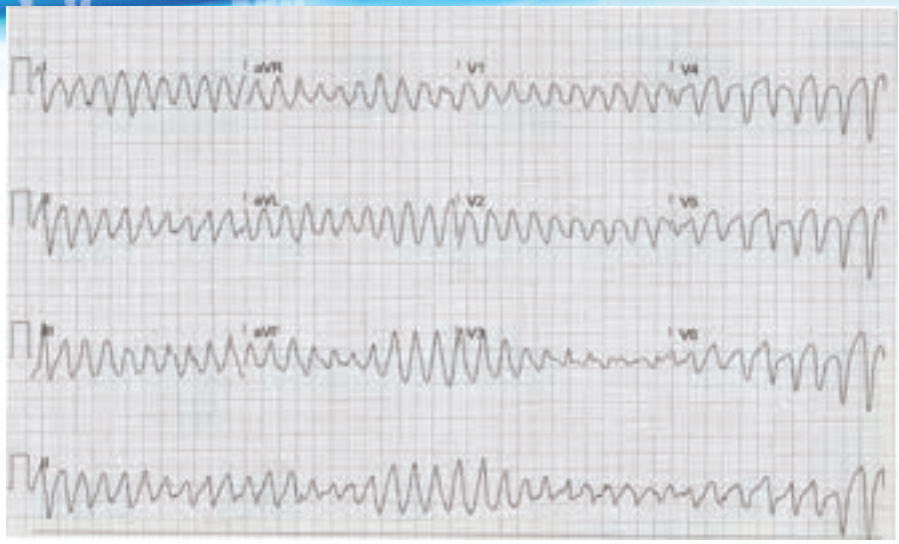

CASE

A 68-year-old female with a past medical history of hypertension presents to the emergency department via private vehicle for recurrent intermittent episodes of unresponsiveness that started just prior to arrival.

What is your interpretation of her ECG?

See the ANSWER on page 52.

ECG Challenge

ANSWER

This ECG shows an irregular wide complex tachycardia at 230 bpm, no discernible P-waves, and phasic variation (i.e., happens over a number of beats) of the QRS complex axis and amplitude.

The differential diagnosis for an irregular WCT includes:

- Ventricular fibrillation
- Polymorphic ventricular tachycardia (including torsades de pointes)
- MAT with aberrant conduction*
- Atrial flutter with variable block and aberrant conduction*
- Atrial fibrillation with aberrant conduction*

*Causes of aberrant conduction include fixed or rate-related BBB, metabolic abnormalities, sodium channel blocker toxicity, ventricular-paced rhythm, and ventricular pre-excitation (e.g., WPW).

The findings in this ECG are consistent with torsades de pointes (TdP), or "twisting of points," which describes the characteristic twisting of the peaks of the QRS complexes around the isoelectric baseline. TdP is used to describe polymorphic VT associated with a prolonged QTc interval on the baseline ECG. It is usually paroxysmal and self-terminating, but it can degenerate into ventricular fibrillation. Conversely, non-torsades polymorphic ventricular tachycardia is defined by the absence of a prolonged QTc interval on the baseline ECG. It is often due to ischemia and is constant, frequently leading to hemodynamic compromise.

A prolonged QTc interval is defined ≥ 460 msec for women and ≥ 450 msec for men, with an increased risk for TdP when the QTc interval exceeds 500 msec. The QT interval should be measured in the lead with the longest QT interval, usually lead V2 or V3. Unfortunately, there is no consensus recommendation for which formula should be used to calculate the QTc interval.

The treatment for unstable polymorphic ventricular tachycardia is defibrillation regardless of classification. Intermittent TdP can be treated with magnesium, electrical overdrive pacing, and potassium repletion (if indicated), regardless of whether it is due to an acquired or congenital prolonged QTc interval. TdP due to an acquired prolonged QTc interval can also be treated with chemical overdrive pacing with isoproterenol. This should be avoided in patients with congenital long QT syndrome, as adrenergic stimuli is a common trigger of dysrhythmias. Even though the QTc interval increases with slower ventricular rates, maintenance therapy for these patients commonly includes beta-blockers to minimize adrenergic stimulation. Patients with non-torsades polymorphic ventricular tachycardia should have an ACS workup.

Case Conclusion

While the initial ECG was being obtained, the patient became unresponsive with palpable pulses. While defibrillator pads were being placed, she spontaneously converted to sinus rhythm with a concurrent improvement in her mental status. A repeat ECG showed sinus rhythm with a QTc interval of 515 msec, and she was immediately treated with 4 g IV magnesium sulfate given over 5 minutes. Labs were notable for severe hypokalemia of 1.6 mEq/L, so aggressive replacement was initiated with both oral and IV potassium. The patient was admitted to the cardiology service and had no further episodes of TdP after her potassium level normalized. An echocardiogram showed no abnormalities, and electrophysiology recommended against placement of an implantable cardioverter defibrillator since the dysrhythmia was triggered by a reversible cause.

Polymorphic Ventricular Tachycardia Learning Points

- Irregular WCT with phasic variation (i.e., happens over a number of beats) of the QRS complex axis and amplitude
- Rate is typically 180-250 bpm
- Classified as either torsades or non-torsades
- Torsades de pointes
- Polymorphic VT with a prolonged QTc interval on baseline ECG
- Usually paroxysmal and self-terminating but can degenerate into ventricular fibrillation
- Non-torsades polymorphic VT
- Absence of QTc prolongation on baseline ECG, typically due to ischemia
- Usually sustained and causes hemodynamic instability
- Treatment includes:
- Defibrillation for unstable polymorphic ventricular tachycardia regardless of etiology
- ACS workup for all non-torsades polymorphic ventricular tachycardia
- Magnesium, electrical overdrive pacing, and potassium repletion (if indicated) for intermittent torsades
- Identify and correct underlying cause(s) for torsades (eg, QT prolonging medications, hypokalemia, hypomagnesemia, hypocalcemia, elevated ICP, etc.)

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

1. A newborn baby is presented for evaluation of yellow skin. The baby was born at home 17 hours ago by spontaneous vaginal delivery at 37 weeks' gestation; the mother received no prenatal care. The baby's vital signs include P 178, R 66, and T 37°C (98.6°F); SpO2 is 97% on room air. What is the most concerning potential cause of this patient's symptoms?

A. ABO incompatibility
B. Breast milk jaundice
C. Familial nonhemolytic jaundice
D. Physiologic jaundice

2. A 22-year-old woman presents after drinking a bottle of toilet bowl cleaner. She complains of nausea with one episode of non-bloody vomiting and epigastric abdominal pain. Her vital signs are BP 125/85, P 99, R 23, and T 37.3°C (99.1°F); SpO2 is 98% on room air. Assessment of her oropharynx is unremarkable, and her voice is normal. Abdominal examination reveals tenderness to palpation of the epigastrium without rebound or guarding. She says she wishes "it would just end." She has no history of suicide attempts. What is the most appropriate next step?

A. Admission for endoscopy and close monitoring
B. Emergent intubation
C. Gastric decontamination with activated charcoal
D. Gastric lavage with placement of an Ewald tube

3. Which characteristic do both staphylococcal and streptococcal toxic shock syndromes have?

A. Blood culture likely to be positive
B. High mortality rate
C. Infection associated with a retained foreign body
D. Toxin-producing gram-positive causative organism

4. A 31-year-old woman presents with seizures. She is 3 weeks postpartum. Her vital signs include BP 160/110, P 95, and T 37.1°C (98.8°F); SpO2 is 94% on room air. What medication should be given first?

A. Hydralazine
B. Lorazepam
C. Magnesium sulfate
D. Phenytoin

5. A 35-year-old man presents via ambulance with hypotension and tachycardia after a motor vehicle crash. He does not know his blood type. Which blood type is the best choice for transfusion in this scenario?

A. AB–
B. AB+
C. O–
D. O+

PEERprep For Physicians

ANSWERS
1) A. 2) A. 3) D. 4) C. 5) D.

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News & Notes in Emergency Medicine

ABEM SELECTS NEW MEMBERS OF RESIDENT PANEL

The American Board of Emergency Medicine (ABEM) is pleased to announce the newest members of its Resident Ambassador Panel: **Joan W. Chou**, MD; **Charles Sanky**, MD, MPH; and **Barbara Van De Water**, MBA, MS, DO. Panel members provide a resident's perspective to certain ABEM activities, such as applying for certification, communication with residents, the Residency Visitation Program, and the ABEM website.

Dr. Chou is an EM resident at SUNY Upstate Medical University in Syracuse, N.Y. During medical school and residency, she has served as a representative to curriculum and academic affairs committees. She is currently the program representative and vice chair to the NYACEP resident committee and EMRA representative for her class. She plans to undertake fellowship training in undersea and hyperbaric medicine.

Dr. Sanky is an EM resident at Icahn School of Medicine at Mount Sinai (New York, N.Y.)–The Mount Sinai Hospital, Elmhurst Hospital Center (Queens, N.Y.), and Mount Sinai Beth Israel Hospital. He is currently vice chair of research for EMRA. Dr. Sanky also holds an MPH degree from the Icahn School of Medicine at Mount Sinai. Prior to attending medical school, he contributed to public health policy at the local, state, and federal levels. He plans to undertake fellowship training in the National Clinical Scholars Program, focusing on health system design, disaster medicine, and health equity.

Dr. Van De Water is an EM resident at the HCA Healthcare/USF Morsani program in Brandon, Fla. Prior to attending medical school, she earned an MBA degree from Northcentral University and an MS degree in Adult Education from Troy University. She has served in the U.S. Air Force and plans to practice in a community setting following residency training.

The Board looks forward to working alongside these residents and to their contributions to ABEM activities.

NAM NAMES ABEM NAM FELLOW

The National Academy of Medicine (NAM) has selected **Caitlin Rublee**, MD, MPH, as the 2022-24 ABEM NAM Fellow.

Dr. Rublee is an assistant professor in the Department of Emergency Medicine and Department of Environmental and Occupational Health; assistant fellowship director, Climate and Health Science Policy fellowship; and director of the Graduate Medical Education, Climate and Health Program, at University of Colorado School of Medicine and Colorado School of Public Health in Aurora, Colo.

The purpose of the ABEM NAM Fellowship is to provide talented early-career health science scholars in emergency medicine the opportunity to participate in evidence-based health care studies that improve patient care outcomes in domestic and global health care systems.

Caitlin Rublee, MD, MPH

During her two-year fellowship, Dr. Rublee will collaborate with eminent researchers, policy experts, and clinicians from across the country, and she will help facilitate initiatives convened by the National Academies to provide nonpartisan, evidence-based guidance to national, state, and local policymakers; academic leaders; health care administrators; and the public. ✶

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